CHESS, A COMMUNITY HEALTH AND ENVIRONMENTAL SURVEILLANCE SYSTEM

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ENVIRONMENTAL PROTECTION AGENCY

1. Introduction

The Community Health and Environmental Surveillance System (CHESS) relates community health to changing environmental quality. CHESS consists of a series of epidemiologic studies in sets of communities representing consistent exposure gradients to common environmental pollutants. The keystone of the CHESS program is the *coupling* of sensitive health indicators to comprehensive environmental monitoring in sets of communities representing a consistent pollutant exposure gradient, thus allowing temporal and spatial replications of dose response studies.

EPA health research needs are practical and problem oriented. CHESS research is thus pragmatic and our goals are threefold: (1) to evaluate existing environmental standards; (2) to quantitate pollutant burdens in exposed populations; and (3) to quantitate health benefits of pollutant control.

2. Chess historic development and present overview

Obligations to prepare air quality criteria documents and set air quality standards were legislated in the Clean Air Act of 1967. CHESS evolution began in the fiscal year of 1968 (FY 68) with the health appraisal of air quality standards (Figure 1). The CHESS concept developed simultaneously with the growth of a multidisciplinary "critical mass" in FY 1969. Growth for this single medium approach (air) was by initial demonstration of both health indicators and monitoring within established CHESS areas and their subsequent expansion into new areas (FY 1970–71). The recent creation of the Environmental Protection Agency (EPA) signalled a more comprehensive and, now, multimedia approach to environmental hazards. CHESS will be fully operational for air pollution effects by FY 1973 and for multimedia toxic substances by FY 1975. Present CHESS operations consist of three basic, integrated functions, namely, Data Collection, Bioenvironmental Measurements, and Information Synthesis, supported by a fourth function, research and development, and coordinated by a

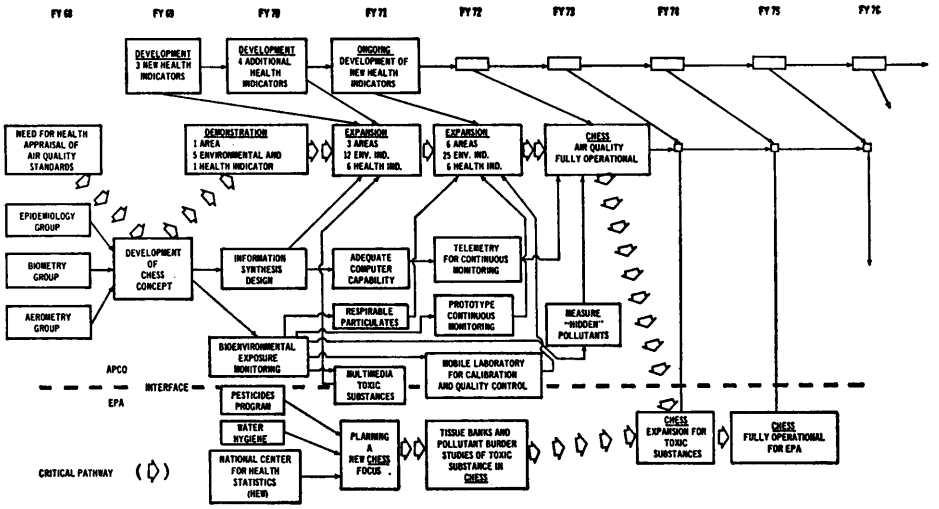

FIGURE 1

Historic development.

fifth function, Program Management (Figure 2). Simultaneous environmental monitoring and measurement of sensitive health indicators in community area sets are the fundamental CHESS components.

3. CHESS area sets

CHESS area sets consist of groups of three or four communities representing an exposure gradient for a pollutant, but similar with respect to climate and socioeconomic traits. Each community within an area set is a defined middle class residential segment of a city containing three or four elementary schools (500 to 1000 children per school) and often a secondary school. CHESS pollutant gradients are as follows:

(1) particulate gradient with low SO_2 (3 Southeast cities),
(2) SO_2 gradient with low particulates (Utah communities),
(3) combined SO_2 and particulate gradient (New York City active, Chicago planned),
(4) photochemical oxidant gradient (Los Angeles Basin),
(5) NO_x gradient (Chattanooga),
(6) trace element and SO_2 gradient (western metal smelter communities).

4. CHESS exposure monitoring

Neighborhood monitoring stations are sited to provide a representative estimate of pollutant exposure for the study population. Supplemental home monitoring of tap water, household dust and soil samples permit even more intimate

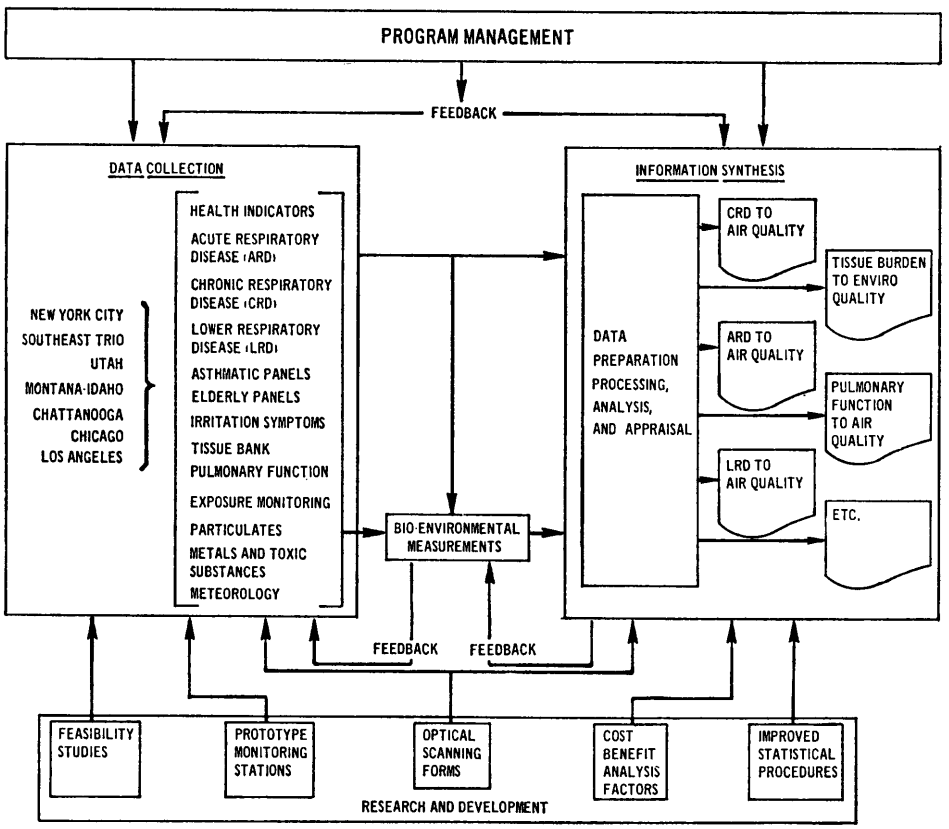

FIGURE 2

CHESS overview.

estimates of environmental trace substance exposure. Study subjects usually live within a 1 to 1.5 mile radius of CHESS stations. Topography, emission sources and local land use are all considered when placing stations. The inlet of the monitoring instruments is usually placed at head level and sheltered from uncommon proximate pollution sources. The CHESS system monitors for the following environmental exposures:

(A) Present CHESS system, all stations
  (1) Total suspended particulate (daily)
    (a) Sulfates (daily)
    (b) Nitrates (daily)
    (c) Organic (monthly)
    (d) Benzapyrene (monthly)
    (e) Trace metals (monthly)
  (2) Respirable particulate (daily)

(3) Dustfall (monthly)
  (a) trace metals (monthly)
(4) Sulfation (Pb O_2 monthly)
(5) Twenty-four hour SO_2 (daily)

(B) Present system, some stations
(1) Two hour soiling index
(2) Twenty-four hour NO_2
(3) Continuous NO_2
(4) Continuous SO_2
(5) Continuous oxidants

(C) CHESS-CHAMP: Community Health Ambient Monitoring Program, prototype field testing
(1) Continuous NO-NO_2
(2) Continuous SO_2
(3) Continuous oxidants
(4) Hydrocarbons
(5) Mobile unit replication
(6) Wind speed and direction.

CHESS-CHAMP (The Community Health and Ambient Monitoring Program) is currently collecting daily 24 hour samples and monthly samples for gases and particulates at 30 environmental monitoring stations. Real time pollutant measurements can accurately relate short term environmental variations to acute response health indicators, distinguishing "peak" exposure effects from 24 hour average effects, if any.

Continuous monitors operate in some CHESS-CHAMP stations and a prototype automatic data acquisition continuous monitoring station with magnetic tape storage and "on call" telemetric output is presently being field tested. "On-call" telemetry permits routine instrument performance checks, daily data processing and thus immediate access to data during air pollution episodes. Duplicate sampling of the environment and frequent calibration of all instruments are systematically obtained to ensure accurate and consistent instrument performance in the CHESS-CHAMP system.

5. CHESS health indicators

Relationships between human diseases and pollution exposures are neither simple nor fully understood. However, one may conveniently think of a five stage biologic response spectrum of increasing severity (Figure 1 in Finklea and co-workers): (1) a tissue pollutant burden unassociated with other biological changes; (2) physiologic changes of uncertain significance; (3) physiologic disease sentinels; (4) morbidity; and (5) mortality. CHESS utilizes health indicators which reflect this entire spectrum. The following acute and chronic response indicators are measured in pre-enrolled subject panels as well as community surveys:

(A) *Acute exposure* (less than 24 hours)
  (1) Reversible pulmonary function changes
  (2) Acute irritation symptoms
  (3) Frequency and severity of asthma attacks
  (4) Aggravation of chronic respiratory disease (CRD) symptoms
  (5) Aggravation of cardiac symptoms
  (6) Daily mortality rates

(B) *Chronic exposure* (greater than 24 hours)
  (1) Pollutant burdens (man as an environmental dose integrator)
  (2) Impairment of lung function
  (3) Absenteeism (no longer used)
  (4) Prevalence of chronic respiratory disease (CRD)
  (5) Frequency of lower respiratory disease (LRD)
  (6) Incidence of acute respiratory disease (ARD)
  (7) Mortality studies

Comparison of similar groups is insured by obtaining covariate information such as age, sex, race and smoking status. These study design covariates all relate to morbidity and failure to measure and adjust for them could cause serious confounding effects. They are summarized as follows: (A) demographic—age, sex, ethnic group, socioeconomic, reporting bias; (B) exposure—diet, water, smoking, occupation, migration, indoor-outdoor differences, daily movement; (C) special risk—temporary such as age, pregnancy, illness; permanent such as alpha-1-antitrypsin deficiency or serum IgE levels.

6. CHESS study strategies

Selection of CHESS area sets and pollutant exposure gradients were dictated by the existence of air quality criteria documents for particulate matter, sulfur oxides, nitrogen oxides, photochemical oxidants, hydrocarbons and carbon monoxide published by the National Air Pollution Control Administration. Area sets for individual pollutants are selected from existing exposure monitoring data. A carbon monoxide (CO) CHESS set was not established because short term CO effects are more precisely studied in controlled exposure chambers and long term CO effects are likely to be confounded with the effects of other vehicle emissions products. Nor has an area set with a consistent CO gradient been found.

Middle class neighborhoods are chosen because they are common, have a more homogeneous family and social class distribution, and are migrationally stable, thus providing a higher likelihood of long term participation. Family participants in the surveys for acute upper, acute lower and chronic respiratory diseases and panels for episodes are recruited from elementary school enrollment listings in CHESS neighborhoods. Subjects for the asthma, cardiac and chronic respiratory disease panels are obtained from prevalence survey results and from patient listings of private physicians. As indicated, our broad data acquisition tech-

niques vary in the frequency and in the type of response they measure. The following methods are currently used: (1) exposure monitoring, (2) single time questionnaire, (3) weekly diaries, (4) biweekly telephone contact, (5) spirometry in schools, (6) telephone contact during alerts, (7) tissue collection, (8) vital statistics.

CHESS programs will operate from three to five years in selected areas. Measurement of sensitive health indicators over a period of increased air pollution control is an optimal way to quantitate the health benefits of this control.

CHESS data collection for FY 71 alone will yield a total of 40×10^6 health indicator and 3×10^5 air determination characters for data processing (Table I).

TABLE I

FY 1971 CHESS PROGRAM: DATA COLLECTION SUMMARY

Indicator	Frequency	Population
CRD	biyearly	30,000
LRD	biyearly	30,000
ARD	biweekly	15,000
Pulmonary Function	triyearly	5,000
Asthma	weekly	300
Elderly	weekly	450
Irritation Symptoms	triyearly	12,000
Pollutant Burdens	biyearly	6,000

Rapid reporting is the rule because high priorities are placed on our study results. Recent CHESS findings span the entire biologic response spectrum and are outlined and referenced in Appendices I and II.

Research goals are essential for optimal CHESS functioning and play a critical role in our development. CHESS research and development goals are threefold: (1) to refine exposure monitoring, (2) to improve statistical procedures and (3) to develope and test more sensitive health indicators. Current and future CHESS health-indicator research is outlined in Appendix III.

Estimating environmental exposure doses has always been a problem. In health studies of multimedia toxic substances, this problem increases. Pollutant burden studies of biological accumulators such as pets, plants, and wildlife in addition to humans should be utilized for appropriate metals, pesticides, synthetic organic materials and selected gaseous pollutants. Sample sets of tap water, house dust and soil collected from CHESS panel families provide intimate information about trace metal exposure when coupled to neighborhood environmental monitoring and dietary metal surveys. Personal monitors for all pollutants would permit the best pollutant dose estimates for individual study subjects.

We have addressed our remarks to the central questions of this conference, namely what pollutants to measure, what health indices to measure, available methods of obtaining both types of data, and available study strategies. CHESS permits a systematic, yet flexible, approach to these problems and has already produced answers to some of them.

APPENDIX

A.1. CHESS: recent findings

(A) Pollutant burdens
  (1) Roadside gradients of Cd, Pb and Zn [6]
  (2) As, Cd, Cu, Pb, Ag and Zn in household dust [36]
  (3) Environmental exposure to As, Cd and Pb reflected in hair and consistent over time and within individuals [19], [20]
  (4) Hg in placentas, Cd in cord blood [35]
  (5) PCB highest in urban whites [15]
  (6) Interlaboratory and interwash variation negligible for hair Cd, Pb and Zn determinations [21]

(B) Physiological changes of uncertain significance
  (1) Hair and blood Pb correlate (.40) over an exposure gradient [19]
  (2) Urinary Cd does not increase with age [18]
  (3) Eye irritation highly correlated with oxidant exposure [22]

(C) Physiologic sentinels of disease
  (1) Pulmonary function in children decreased after SO_x, particulate and NO_x exposure [30], [32], [33]
  (2) Pulmonary function in adults decreased after NO_x exposure [35]
  (3) Systolic BP in adults 40 may be increased after Cd exposure, but not diastolic BP or cholesterol [11]

(D) Morbidity
  (1) ARD and LRD in children after exposure in NO_x [25], [32]
  (2) CRD symptoms in young adults but not adolescents more frequent after exposure to SO_x and particulates but not O_x [35]
  (3) Respiratory and eye irritation symptoms induced by acute urban air pollution exposure [4]
  (4) Asthma attacks more frequent after nitrate, SO_x, and particulate exposure [3]
  (5) Cd not increased in toxemia of pregnancy [7]
  (6) No observed effect of chronic oxidant exposure on epidemic influenza in school children [26]
  (7) Epidemiologic evidence linking Cd to hypertension is weak when critically reviewed [18]

(E) Mortality
  (1) No long term effect of As, Pb on survival of "Neal Cohort" [24]
  (2) No long term effect of acute NO_x exposure on survival [17]
  (3) No effect of water hardness and no consistent effect of Cd on cardiovascular disease mortality [27]
  (4) Possible relationship between chronic urban air pollution exposure and carefully adjusted mortality rates in Chicago and Philadelphia [27]
  (5) Large temperature, influenza and socioeconomic effects on daily mortality [2]

(F) Associations with cigarette smoking
  (1) Increases in ARD, LRD frequency [14]
  (2) Higher influenza attack rates [16]
  (3) Impaired persistence of HI antibody [13]
  (4) Decreased ventilatory function
  (5) CRD symptoms in early adolescence
  (6) Refractiveness to acute air pollution episodes [4]
  (7) No change in ARD, influenza, or antibody persistence among children if parents smoke [10]

(G) Some recent reviews
  (1) General overview of CHESS research [29]
  (2) Overview of human pollutant burden research [12]
  (3) Air pollution episodes—guide for health departments and physicians [5]
  (4) Review of arsenic health effects [1]
  (5) Review of beryllium health effects [28]
  (6) Review of cadmium health effects [18]
  (7) Reviews of environmental lead and human health [8], [31]
  (8) Plasticizers in the environment [23]
  (9) Environmental hazards of optical brighteners [9]

A.2. CHESS: research and development goals

(A) Refined exposure monitoring
  (1) Biological amplifiers (pets, plants, wildlife)
  (2) Personal monitors
  (3) Tap water, housedust, soil

(B) Improved statistical procedures
  (1) Hockey-stick and other dose response functions
  (2) Ridit transformation and linear models for categorical data
  (3) Daily mortality models
  (4) Analysis of truncated and censored data
  (5) Estimating personal exposure

(6) Multivariate techniques for repeated measurements
(7) Health information synthesis system

(C) More sensitive health indicators
(1) Pollutant burdens
(a) Maternal-fetal tissue sets
(b) Patients-biopsy, surgery, autopsy
(c) Special occupations
(2) Altered physiology of uncertain significance
(a) Carboxyhemoglobin
(b) RBC fragility and survival
(3) Physiologic heralds of disease
(a) Other PF tests
(b) Blood lipid patterns
(c) Blood pressure
(d) Immune response
(e) Exfolliative cytology
(4) Morbidity
(a) Aggravation of hypertension
(b) Aggravation of RDS of newborn
(5) Mortality
(a) CO and coronary disease
(b) Area studies linked to SS records

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Discussion

Question: John R. Goldsmith: Environmental Epidemiology, California Department of Public Health

Does the use of the word "after" in statement of "physiological sentinels of disease" of Cadmium on B. P. and in morbidity of asthma and nitrate, SO_x, and particulate exposure, imply a pre-exposure and post-exposure set of observations?

Reply: W. Riggan

No. It generally implies a set of simultaneous observations over an exposure gradient.

Question: B. G. Greenberg, School of Public Health, University of North Carolina, Chapel Hill

I should like to ask the speaker if the surveillance system is looking at early precursors of disease in the social and psychological areas. All of the named

measures are physiological in nature, whereas certain other characteristics might be even earlier in occurrence without any demonstrable physical change. Specifically, such measures are irritability, ability to function at optimal levels in remembering digit sequences, adding numbers, and other mentation tasks might be used as response indicators. Have you given thought to including such measures among the response variables?

Reply: W. Riggan

Yes. Both field and laboratory feasibility studies are in progress.

Question: R. J. Hickey: Institute for Environmental Studies, University of Pennsylvania, Philadelphia

I would like to inquire about the "pollutant" referred to as "oxidant." This, as I understand it, is a sort of conglomerate of chemicals which reacts with a KI reagent, and includes ozone, other peroxides, and nitrogen dioxide. There have been reports suggesting that ozone is, or may be, mutagenic. Further, populations are genetically heterogeneous, of course, and in human populations a metabolic defect occurs in some individuals pertaining to the enzyme, catalase. Both acatalasia and hypocatalasia are, I believe, known. Thus there can be, or are, individuals who are particularly sensitive to hydrogen peroxide because of deficiency of catalase. But, so far as I know, NO_2 is extraneous to this system. My question, therefore, has two parts: (a) for a molecular biological approach to statistical studies, can a measure of peroxide be reported free of NO_2, and (b) is there any way that past "oxidant" data may be modified by removal of the NO_2 effect so that only some peroxide measure may be obtained?

Reply: W. Riggan

Your question regarding a biological response to peroxide in oxidant atmospheres is well taken. The measurement of oxidants in the past has been by the use of the KI reagent where a number of compounds may react to both a positive or a negative response. Advanced methodology detects specific ozone concentration, free of the other oxidant compounds. If specific methods were available for monitoring these other compounds routinely during the course of a population health study, it may be possible to deduce the concentration of peroxides. However, these methods are not available at present for routine air monitoring.

It would be very difficult to adjust past oxidant data for NO_2 proportions with any degree of accuracy since NO_2 concentrations vary from both a spatial and temporal standpoint. Any data adjustment would be academic and would not be consistent to the degree of precision and accuracy required for the scientific determination of health effects.

Question: Burton E. Vaughan, Ecosystems Department, Battelle Memorial Institute, Richland, Wn.

In your discussion of a surveillance system, I am troubled by the lack of explicit mention of any data base to be obtained on those organisms needed to assess environmental degradation. Surveillance concerns not just disease. Certain

environmental changes have real and expensive consequence to human well being *indirectly* (for example, effects on crops, effects on forests, and so on).

Reply: W. Riggan

Our primary responsibility is health effects research, but we coordinate our efforts with the Division of Ecological Research as well as other agencies whenever feasible.

Question: Colin White, Department of Public Health, Yale School of Medicine

Have you had any problem of over reporting when you used a telephone survey during a pollution alert?

Reply: W. Riggan

We found no over reporting from using a telephone survey during an air pollution alert in New York conurbation. Three areas surveyed were alike in ethnic and socioeconomic characteristics but differed markedly in exposure to air pollution. The first call followed a three day rise in air pollution levels accompanied by the formal issuance of an air pollution forecast. The second call followed a three day period of elevated pollution unaccompanied by publicity. The final call followed three days of low pollution. A series of bias control questions were asked in each call. The response rate of acute symptoms was not affected by publicity, but the response rate was very susceptive to air pollution levels. The response rates to the control health questions were similar among all areas for all three telephone surveys.