Colorado Tobacco Research Program

2001 Compendium
of
Funded Projects

Administered by The University of Colorado
Office of the President
Office of the Vice President for Academic Affairs and Research

COLORADO TOBACCO RESEARCH PROGRAM
2001 COMPENDIUM OF FUNDED PROJECTS

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COLORADO TOBACCO RESEARCH PROGRAM 2001 COMPENDIUM OF FUNDED PROJECTS

The Colorado Tobacco Research Program (CTRP) is pleased to announce the initiation of 14 new research projects, and a Baseline Study Award, that will lead to more knowledge about the etiology, pathogenesis, diagnosis and treatment of tobacco- and addiction-related diseases and the development, implementation, evaluation, and dissemination of existing or novel approaches to tobacco control and substance abuse education. Individual investigators from six institutions from across the state, including Colorado affiliated colleges, universities (e.g., Colorado State University and the University of Colorado campuses), public, private & non-profit organizations (e.g., AMC Cancer Research Center), and non-profit hospitals (e.g., National Jewish Research & Medical Center) are embarking on research that will reduce the morbidity and mortality associated with tobacco use. By providing more than $6.1 million to fund these new awards, the State of Colorado has pledged its commitment to reducing the physical and mental health impact, and the corresponding economic burden, of tobacco-related diseases within the state.

ABOUT CTRP

By creating the tobacco research fund, the Colorado State Legislature made optimal use of newly available monies to benefit Colorado citizens. Senate Bill 00-071 determined how the State of Colorado's share of the national tobacco settlement funds would be spent. The bill allocated up to 8% of the monies received annually for the establishment of a comprehensive clinical, basic science, mental health, and evaluative research grant program that would serve Colorado's tobacco- and substance-abuse-related health care needs. SB 00-071 ensures that the research grant program supports the people of Colorado by directly addressing the mental health, educational, cessation, prevention and illness-related needs caused by tobacco and substance abuse within the state.

To implement the research program, the State Legislature assigned the Office of the President of the University of Colorado (CU) the duty of administering the Colorado Tobacco Research Program. Within the CU Office of the President, the Vice President for Academic Affairs and Research thus created CTRP, with the charge to award research grants based on scientific merit and relevance to the Program's mission in an open, competitive manner. SB 00-071 also directed the Governor to establish a Scientific Advisory Committee to assist the University in administering CTRP. Comprised of members of voluntary health organizations dedicated to the reduction of tobacco use, experts in the fields of biomedical or social/behavioral research, representatives from research universities and institutions focused on tobacco-related issues affecting children and youth, and members of medical or health organizations, the Scientific Advisory Committee primarily develops the strategic objectives and priorities of CTRP, and facilitates coordinated efforts between the Program and other stakeholder entities focused on reducing tobacco use and tobacco-related disease in Colorado.

RESEARCH PRIORITIES

CTRP supports both basic and applied research in social and behavioral sciences, biomedical sciences, and public health and policy. CTRP invites investigations into the etiology, pathogenesis, diagnosis and treatment of tobacco- and addiction-related diseases and the development, implementation, evaluation, and dissemination of existing or novel approaches to tobacco control and substance abuse education. The following is a list of the 2001-2002 CTRP Research Priorities:

Biobehavioral and Nicotine Addiction Treatment Research

CTRP seeks to fund basic biobehavioral investigations of the biological, psychological, sociocultural, and genetic factors that influence initiation of tobacco use, progression to nicotine addiction, smoking cessation, and relapse; the pharmacological basis of nicotine addiction, including, but not limited to, the role of nicotine receptors in addiction; the appropriate role of nicotine replacement therapies (NRT) in nicotine addiction; research that identifies, tests, and disseminates interventions to treat addicted tobacco users; studies that shed light on how nicotine addiction and disease develop; and explorations of applying the "harm-reduction" paradigm to tobacco use.

Biological Research

CTRP requests studies that strive to reduce the morbidity and mortality from tobacco-related diseases. Appropriate areas include basic disciplines — such as physiology, biochemistry, pathology — as well as translational and clinical investigations that focus on problems associated with tobacco use. CTRP encourages studies that identify and validate biomarkers of tobacco exposure and tobacco-induced cellular events that relate to the different stages of disease progression; define the mechanisms by which tobacco use contributes to disease progression and management; examine the effects of prenatal and postnatal exposure to parental tobacco use; contribute to the understanding of the effects of smoking on our physical and mental health, and discern how these effects may differ by age, ethnicity, race or gender.

Effects of Exposure to Secondhand Smoke

CTRP will fund research that focuses on the biological impact of exposure to secondhand smoke. In addition to research on chronic ailments directly associated with tobacco smoke exposure (e.g., atherosclerosis), studies into the mechanisms, diagnosis or treatment of pulmonary diseases associated with childhood exposure to secondhand smoke (e.g., chronic bronchitis) or exacerbated by secondhand smoke (e.g., asthma) are encouraged. Important in this regard are quantifying and understanding the chronic effects of exposure to secondhand smoke; and how the impact of exposure to secondhand smoke differs by age and by other demographic factors, emphasizing the need for appropriately designed studies to characterize potentially disproportionate exposures and sensitivities.

Epidemiological and Surveillance Research

CTRP is interested in funding studies that identify differences in host (inherited and acquired), environmental, and behavioral factors that may help elucidate unique contributors to tobacco use and tobacco-related disease. An important and emerging area of research in tobacco use and addiction control is genetic epidemiology. CTRP encourages investigations into the shifting patterns of tobacco use in youth and young adults, smokeless tobacco and cigar use among Colorado teens, and the relationships of illicit drugs to tobacco use. Surveillance and research is needed to monitor and evaluate trends in tobacco use and related disease risk factors, health services, and policy and environmental interventions to determine the influence of these factors on trends in tobacco-related disease incidence, morbidity, mortality, and survival. CTRP also encourages studies that use Colorado's data collections for secondary data analysis.

Prevention of Tobacco Use

CTRP seeks basic and applied social/behavioral research in the prevention of tobacco and substance use. Topics may include, but are not restricted to, tobacco use in schools and communities; experimentation and the casual use of nicotine products; exposure to secondhand smoke; and tobacco usage by mental health populations. Interventions in historically understudied communities or specific racial and ethnic groups to elucidate unique factors and forces shaping their tobacco consumption are invited. CTRP particularly encourages studies that illuminate the resiliency among subpopulations of youth, and that document trends and develop interventions to curb the rise in smoking among young women.

Policy Research

CTRP is interested in funding evaluative research that examines the impact of public policies and programs on smoking rates and practices. Included are studies of regulatory policies that limit or discourage access to tobacco products; studies which look at how safety claims for new projects developed by the tobacco industry will be evaluated; research into health care policies and the medical sector's actual and potential role in reducing tobacco in Colorado; and evaluation of efforts to eliminate the tobacco industry's promotions of tobacco products. CTRP also encourages research that documents the role of anti- and pro- tobacco forces in shaping Colorado tobacco policies (e.g. smoke-free bar issues); assessing the impact of the Master Settlement Agreement (MSA) on state and local anti-smoking policies.

◆ TYPES OF PROJECTS FUNDED (AWARD MECHANISMS)

Independent Investigator Awards

Research Project Awards

Investigator-initiated research projects. The proposals should be fully developed, scientifically rigorous, and include sound background information, hypotheses, and promising preliminary studies or supporting data.

Term: Maximum of 3 years.

Award: Average annual direct costs should not exceed $175,000. Indirect costs are paid at the federally determined rate for eligible institutions.

Innovative Developmental and Exploratory Awards (IDEAs)

IDEA grants are for exploratory research that is not yet sufficiently mature to compete successfully for a Research Project grant. Although the proposed research might lack adequate pilot data or proven methods, it should be creative, intellectually exciting, and show clear promise to yield findings that could serve as the basis for a well-defined future Research Project application.

Term: Maximum of 18 months.

Award: Total costs cannot exceed $75,000 in direct costs, including a maximum of $5,000 for equipment. Indirect costs are paid at the federally-determined rate for eligible institutions.

Career Development Awards

Postdoctoral Fellowship Awards

These are awards for individuals to obtain postdoctoral research training under a designated mentor (not intended for junior faculty members). Applications, which must be prepared and submitted by the fellows, must outline a separate research project and include letters of support. The fellow must commit to a minimum of 80 percent time to the research project.

Term: Maximum of 2 years.

Award: Maximum of $35,000 direct costs per year plus up to $10,000 for fringe benefits and health insurance, averaged over the duration of the award. The indirect cost rate is 8% for eligible institutions.

Dissertation Research Awards

These awards are provided to support the dissertation research of doctoral candidates who wish to pursue tobacco-related research. The awards are designed specifically for students who will have advanced to candidacy by the award start date and who are initiating their dissertation research. Applicants and their mentors must be affiliated with an academic institution in Colorado that grants doctoral degrees and the doctoral candidates must be directly supervised by a mentor who is eligible to be a principal investigator at the applicant's institution. The awardee must commit a minimum of 80percent time to the research project.

Term: Maximum of 2 years.

Award: Maximum of $20,000 direct costs per year for stipend and supplies plus up to $10,000 for tuition remission, fringe benefits, and health insurance. No indirect costs will be paid.

CTRP AWARD PROCESS

Research funds are available to investigators at all universities, colleges, research institutes, and other nonprofit institutions in Colorado via a grant application process. Following the receipt of grant applications, and prior to the peer review process, all applications are screened for their direct relevance to tobacco or substance use or tobacco-related disease. Briefly, most of the proposals reviewed in the prevention, cessation, policy and epidemiological disciplines focus directly on human tobacco use and/or tobacco control issues, making their relevance to CTRP's mission apparent. Those applications that directly focus on the etiology, pathology, diagnosis or treatment of a specific tobacco-related disease, for which there is unequivocal epidemiological evidence, are also considered highly relevant to CTRP's mission. In contrast, those research proposals focused on basic biological phenomena must demonstrate how the research will yield insights into tobacco-specific health effects. Only those applications considered relevant to the goals of CTRP are forwarded for scientific peer review by an appropriate review panel or "study section".

In 2001, CTRP contracted with the University of California Tobacco-Related Disease Research Program (TRDRP) to review CTRP grant applications. Established in 1989, TRDRP operates a grant evaluation program modeled after that of the National Institutes of Health (NIH), utilizing expert reviewers from all states (including Colorado) appropriate for the scientific discipline and subject matter. Rosters of the TRDRP 2001 review panels or "study sections" are provided beginning on page 23. For further information on TRDRP please refer to the following website: www.ucop.edu/srphome/trdrp.

CTRP applications were reviewed in a two-tiered manner similar to the NIH peer review process. Once the peer reviews had been completed by the appropriate TRDRP study section, CTRP applications were then ranked by scientific merit score. Applications scoring in roughly the upper half of the merit range were subsequently forwarded to the CTRP Scientific Advisory Committee (SAC), who made the final recommendations to the President of the University on which applications should be funded, based on the established Program priorities, the scientific merit of the proposals as determined by peer review, and the amount of funds available. SAC members and the organizations they represent are listed on page 21. The grant applications recommended for funding by the peer reviewers and CTRP's SAC represent important and innovative projects that promise to advance the field of tobacco research in areas where more knowledge is needed to affect meaningful prevention and cessation of tobacco use, and prevention, diagnosis and treatment of tobacco-related diseases.

2001 CTRP DATA

As part of its first cycle of funding, CTRP was legislatively mandated to support a baseline evaluation survey to gather information about tobacco-related behavior and attitudes of children and adults in Colorado. A separate Request for Proposals for this contract was issued; following a review of submitted proposals by the CTRP Scientific Advisory Committee, the $1.5 million contract for the Baseline Study Award was granted to Arnold Levinson, Ph.D., of AMC Cancer Research Center. Future baseline studies will be funded from tobacco settlement funds awarded to the Colorado Department of Public Health and Environment. Since the majority of monies allocated to CTRP in subsequent years (minus the 5% allowed for administrative costs) will be designated for grants, there will be additional funds available for investigator-initiated research projects.

In response to the 2001 CTRP Call for Applications, the investigator-initiated research areas funded in the first cycle of awards drew from the broader research priorities set by the CTRP. The research areas listed below do not define the parameters of the research to be funded in future cycles. In other words, the types of projects supported by CTRP in any given cycle will reflect the particular disciplines of applications submitted, their relative scientific excellence, and the amount of funds available to CTRP that year. By no means is this year's Compendium meant to represent the definitive research portfolio supported by CTRP. Indeed, applications focused on those Research Priorities in which no awards were supported this year (e.g., policy research; health effects of secondhand smoke) will be particularly encouraged in future cycles.

For 2001, CTRP has awarded a total of $4.64 million for 14 grants to individual investigators at 6 research organizations. This funding level represents a "payline" of 23% of all applications submitted last year. The first cycle of awards supported by CTRP fell into the following research areas: Disease Diagnosis & Treatment, Nicotine Addiction, Prevention and Cessation, and Mental Health.

Award Distribution

Research Areas	Amount Funded (%)	Awards (% of total)
Disease Diagnosis & Treatment	5 (36)	$ 1,873,567 (40)
Nicotine Addiction	2 (14)	$ 126,720 (3)
Prevention and Cessation	6 (43)	$ 2,523,915 (55)
Mental Health	1 (7)	$ 113,250 (2)
Total	**14 (100)**	**$ 4,637,452 (100)**

In other words, slightly more than half of the awarded funds will support six studies that center on the social and behavioral factors underlying why individuals start to smoke, how cultural and family norms influence smoking prevalence, and what factors contribute to effective anti-smoking messages. Forty percent of the award monies will fund five projects focusing on tobacco-related disease processes, ranging from basic biological studies of the molecular and cellular changes that are critical to the initiation of disease, the development of new or refined diagnostic approaches to identify disease progression, and on potential therapies and/or novel drug delivery techniques. Two projects will investigate the underlying physiological mechanisms that may predispose individual susceptibility to nicotine addition or play key roles in its progression. Finally, one project will study what factors underlie maternal tobacco use and its effects on brain development.

This compendium lists the 14 funded grants in two sections. First, projects are grouped within research areas by principal investigator (in alphabetical order); a brief summary of each project is provided. Following the next section, the lay abstracts of each project, composed by the investigators themselves, provide additional details; these are grouped by award type and again are arranged in alphabetical order of the principal investigator's name.

Disease Diagnosis & Treatment

Greer, Robert O.

University of Colorado Health Sciences Center

Telomerase Expression in Tobacco Associated Oral Cancer

(Research Project)

Will evaluate a host of tobacco associated pre-cancerous and cancerous oral lesions in order to access potential malignant risk.

Huang, Mingxia

University of Colorado Health Sciences Center

Study of the Effect of the Tobacco Carcinogen Benzo(a)pyrene in *Saccharmyces cerevisiae*

(IDEA Project)

Utilizing budding yeast as an experimental model to investigate the consequences of exposure to the major carcinogen in tobacco, benzo(a)pyrene, this study will help our understanding of similar processes in human cells.

Panagiotidis, Michail I.

National Jewish Medical and Research Center

Epithelial Injury by Cigarette Smoke: Sulfur Metabolism

(Dissertation Award)

Will study how smoking may affect the DNA of lung cells so as to predispose a person to lung cancer and possibly cause depletion of vital nutrients to lung cells.

Sievers, Robert E.

University of Colorado at Boulder

CO_2-Assisted Nebulization for Drugs for Lung Ailments

(Research Project)

Will focus on two major tobacco-related diseases, lung cancer and emphysema, and will provide guidelines that allow formulations of new treatment compounds to be delivered into the lungs.

Silkoff, Philip E.

National Jewish Medical and Research Center

Inflammation, Oxidative Stress and Dysphasia in COPD

(Research Project)

Will identify subjects at high risk for lung cancer by examining phlegm for pre-malignant and malignant cells.

Nicotine Addiction

Breeze, Liam J.

University of Colorado Health Sciences Center

Nicotine Signal Transduction in the Central Nervous System

(Dissertation Award)

Will determine a mechanism by which nicotine can result in long-term changes in the brain and provide insights into the origins of nicotine addiction.

Dobelis, Peter

University of Colorado Health Sciences Center

Chronic Nicotine Effects in Mouse Hippocampal CA3 Region

(Postdoctoral Fellowship)

Will investigate chronic nicotine effects in the CA3 region of the hippocampus from inbred mice.

Prevention & Cessation

Helme, Donald W.

AMC Cancer Research Center

Colorado Anti-Tobacco PSA Message Sensation Value Project

(Research Project)

Will test the effectiveness of a brief media-based tobacco prevention program on producing changes in adolescents' attitudes toward smoking.

Klinnert, Mary D.

National Jewish Medical and Research Center

ETS Reduction Counseling for Families of Asthmatic Children

(Research Project)

Will implement an environmental tobacco smoke reduction program with the families of inner city asthmatic children.

Struhsaker Schatz, Mona C.

Colorado State University

Tobacco Use by Foster Care Youth and Professional Responses

(IDEA Project)

This study will examine the role of tobacco use among foster children and youth ages 10-17. Additionally, this study will examine how a respondent group of professional workers and foster parents help these foster children with smoking cessation and smoking prevention.

Swaim, Randall C.

Colorado State University

Prediction of Tobacco-Using Groups in Pre-Adolescent Youth

(Research Project)

Will help determine how prevention programs should be developed and have important implications for the design of culturally appropriate prevention programs.

Wagner, Carson B.

University of Colorado at Boulder

Theory and Research Confounds in Anti-Substance PSA Study

(Dissertation Award)

Will delineate the contemporary history of anti-substance PSA research and conduct empirical studies on their theoretical and methodological weaknesses toward highlighting areas in need of improvement.

Yousey, Yvonne K.

University of Colorado at Denver

Household Factors and Passive Smoke Exposure of Preschool Children

(Dissertation Award)

Will explore those factors which families identify as being important in developing rules about smoking in their homes and cars.

Mental Health

Ross, Randal G.

University of Colorado Health Sciences Center

Tobacco and Schizophrenia Affect Prenatal Brain Development

(IDEA Project)

Will lead to greater understanding of both the negative health effects of tobacco on early brain development and on the genetically-mediated prenatal brain development in those most at risk for later tobacco use.

LAY ABSTRACTS OF FUNDED PROJECTS

Research Projects

Telomerase Expression in Tobacco Associated Oral Cancer

Greer, Robert O.

University of Colorado Health Sciences Center

The evolution of oral cancer requires multiple steps in which a gradual accumulation of mutations alters cellular growth, proliferation and differentiation, and is expressed as a progression from normal tissue to precancerous tissue and ultimately to cancer. However, most cells with cumulative mutations do not progress to cancer. Therefore, investigators continually are searching for biomarkers for risk assessment of so-called precancerous lesions. The emergence of molecular biology with its new prognostic and, therapeutic tools represents an enormous opportunity in this arena. The use of biologic markers to screen patients who are at increased cancer risk may help to predict the probability of disease progression, aid in the diagnosis, assess the prognosis of the individual cancer patient, develop treatment protocols, and evaluate the response to therapeutic agents. We believe that it is prudent therefore to evaluate a host of tobacco-associated precancerous and cancerous oral lesions that have been reported to show progression to cancer, as well as lesions with questionable cancer potential including smokeless tobacco keratoses, in order to access potential malignant risk using a sensitive biomarker, the telomerase assay.

Telomerase is an enzyme that synthesizes telomeres, i.e., strands of DNA at the end of chromosomes that seem to serve as cell division "clocks." Telomerase is activated in most human cancer tissues but not in most normal tissues and tissues adjacent to cancer. Previous studies have shown that the lack of telomerase activity correlates with critically shortened telomeres and frequent spontaneous cancer remission. Thus the expression of telomerase is important and may be a rate-limiting step for cancer progression. Finally, because telomerase activity is detectable in the intermediate steps of progression to oral mucosal cancer, it may serve as a biomarker for cancer risk assessment in patients with certain oral precancers known as leukoplakias.

Colorado Anti-Tobacco PSA Message Sensation Value Project

Helme, Donald W.

AMC Cancer Research Center

Recent estimates collected by the Colorado State Tobacco Education and Prevention Partnership (STEPP) show that 37% of high school students smoke, 23% of male students use smokeless tobacco (US Department of Health and Human Services, 1998), 20,000 children yearly become new daily smokers, and 3.4 million packs of cigarettes are illegally sold to children yearly in Colorado (Cummings, 1994). Tobacco use places a large burden on Colorado: Annual smoking-related health care expenditures are estimated at $930 million ($150 million of Colorado government Medicaid payments) (US DHHS, 1998). Annual expenditures for babies' health problems related to mothers smoking or exposure to secondhand smoke during pregnancy range from $17 to $49 million (Miller, 1998), and 4,400 adults die annually from smoking-related diseases (Adams & Melvin, 1998; US DHHS, 1997). Clearly, tobacco control is a priority for the health of Coloradoans.

One tool often used to reach audiences with anti-tobacco messages is the mass media through public health campaigns. Increasing the effectiveness of public health media campaigns is essential because of their potential for reaching vastly greater audiences than normally are reached through other methods. Although research over the last two decades has shown that the media can be used effectively in conjunction with interpersonal and institutional channels (e.g., schools), much is yet to be learned about how to make the media components of prevention campaigns most effective.

Televised Public Service Announcements (PSAs) continue to be the most popular media tool in prevention campaigns. Designing effective televised anti-smoking PSAs can be a difficult task. Our own research has established that connections between drug use, sensation-seeking, and particular message preferences are important keys to successful PSAs. Sensation-seeking—a biologically based personality trait characterized by novelty seeking and risk-taking—consistently has been shown to be associated with illegal drug use by teenagers and young adults, and with distinct preferences for innovative and exciting messages. We have demonstrated in laboratory studies that high sensation seeking young adults are more persuaded by anti-drug PSAs which are high in sensation value (HSV) (i.e., draw out strong sensory, emotional, and exciting responses) than by less stimulating messages (low sensation value or LSV) (Palmgreen, Lorch, Donohew, Rogus, Helm, Grant, 1991; Donohew, Palmgreen, Lorch, 1994; Palmgreen, Lorch, Donohew, Harrington, Dsilva, Helm, 1995). Other laboratory and field research we have conducted has shown that placing such PSAs in television programming also high in sensation value greatly enhances high sensation seekers' attention to and recall of prevention messages and the persuasive effectiveness of these messages.

This study examines the argument that the research on televised anti-drug PSA campaigns based on the sensory,

emotional, and need for excitement of high sensation seekers can be applied to tobacco use to produce media messages that achieve significant changes in tobacco-related attitudes, intentions, and prevention behaviors. The study proposed here will examine this argument among adolescents aged 12 to 14 years - a critical time when many experimental smokers start to become habitual tobacco users. The principle objectives of the study are to test the effectiveness of a brief media-based tobacco prevention program containing high sensation value messages on producing changes in adolescents': a) attitudes against smoking; b) intentions not to smoke; c) likelihood of acquiring additional advice on how to not smoke (prevent uptake or stop smoking) when compared to a program containing message lower in message sensation value (LSV).

ETS Reduction Counseling for Families of Asthmatic Children

Klinnert, Mary D.

National Jewish Medical and Research Center

Since the 1980's, studies of childhood asthma have shown higher percentages and greater severity among low-income minority children residing in large urban areas. Since then, studies have found that the high rates of asthma were due mainly to low income status, which in turn is related to social problems and environmental exposures that are increased in the inner city and are detrimental to asthmatics. Among the detrimental environmental exposures that may account for increased asthma problems among inner city children is the high exposure of many of these children to cigarette smoke. There has been clear evidence for some time that passive smoke exposure is associated with increased occurrence of asthma as well as increased severity and health care utilization for those children who have asthma. Based on a comprehensive national survey conducted from 1988-1991, 43% of children in the United States lived in a home with at least one smoker. In comparison, in the National Cooperative Inner-City Asthma Study of low-income urban children with asthma, baseline assessments showed 59% of homes included at least one smoker. Thus, poor urban children with asthma are exposed to high levels of cigarette smoke, which has significant effects on their asthma morbidity.

Denver has many asthmatic children from low-income families. The Denver Health & Hospital Authority (DHHA), which serves as the main safety net health care provider toDenver's children of low-income families, sees over 2,000 children with asthma each year. Among these children, asthma severity is high, with increasing numbers of ED visits and hospitalizations. The goal of the proposed study is to implement an environmental tobacco smoke (ETS) reduction program with the families of these inner city asthmatic children. To assess whether the program is effective, the study will be designed to have one group that receives counseling and another group that receives no counseling. The non-counseling group will receive the usual message about decreasing ETS that all families of asthmatics receive at Denver Health. Families that agree to be in the study will have an equal chance of being assigned to either group. Counselors will go to the homes of the families assigned to the counseling group to work with families on ways to decrease the amount of tobacco smoke to which their asthmatic child is exposed. Emphasis will be on changing smoking behavior patterns, rather than on smoking cessation. Families will receive help in planning and implementing these behavior changes, and in rewarding themselves for success. Counselors will also increase families' knowledge about asthma and the ways in which it is made worse by cigarette smoke. This counseling approach has been used successfully with middle class families of asthmatics as well as with low-income ethnically diverse mothers of young children.

Subjects will be 132 low income, racially diverse children ages 5 to 13 with asthma, who are seen for their asthma at DHHA and who are exposed to cigarette smoke in their homes. Ethnically, the sample is expected to be about 75% minority, with about 25% bilingual or monolingual Spanish speaking. There will be 3 home-based evaluation sessions: at the beginning, after the counseling, and one year after the counseling has ended. At each evaluation, parents will be asked to report the number of cigarettes they have smoked in the child's presence. In addition, the children will be asked to provide a urine sample, which will be tested to see how much nicotine they have in their system as a result of being around cigarette smoke. Also, parents will be asked to report on their child's asthma symptoms in the past 2 weeks. The children will be asked to do a breathing test, which involves blowing into a tube attached to a computer to measure how well they are breathing. As another way to see if the smoke reduction counseling makes a difference, the number of emergency room visits and hospitalizations a child has had for asthma in the year after counseling will be compared with the number from the year before the study.

CO_2 Assisted Nebulization for Drugs for Lung Ailments

Sievers, Robert E.

University of Colorado at Boulder

Smoking is the leading cause of both lung cancer and emphysema. Lung cancer is the major cause of cancer mortality. Emphysema is also a serious disease in which the walls of the lung are permanently damaged and weakened, leading to severe problems with breathing. In both of diseases the initial site of damage is located in the interior of the lung. Thus, to treat or prevent these diseases, it is necessary that the chemicals used for treatment reach the lungs' interior. If these chemicals are given systemically in the diet or by injection, relatively high doses are needed, to assure that an effective level of the chemicals reach the lungs' interior. Therefore, there is a great risk of causing side effects in other organs and tissues.

Recently, in studies in animals, it has been shown both

for lung cancer and emphysema that direct delivery of active compounds to the lung's interior via inhalation can greatly reduce risk of systemic side effects and increase effectiveness. These studies investigated the treatment of emphysema with a protein therapeutic and the chemoprevention of lung cancer with anti-inflammatory compounds.

Fine dried powders of compounds are ideal vehicles for delivery of therapeutic or preventative agents into the lungs. With a simple inhaler device, these powders can be easily and conveniently administered. Also, as dried powders such compounds are much more stable than they would be in liquids. This is especially true for protein-based therapeutics, which are very easily damaged irreversibly in liquids. A commercial therapeutic product must be stable for at least 18-24 months and be able to withstand exposure to extreme conditions such as freezing temperatures. Such stability can be achieved readily in dried powders, but rarely in liquids.

The University of Colorado has developed and patented a novel method to make fine dried powders of therapeutic and chemopreventative agents. This process uses supercritical carbon dioxide (i.e., CO_2 at high pressure) to convert drug substances to very fine powders. Our goals in the current proposal are to investigate this method for making powders of the protein therapeutic for emphysema and the anti-inflammatory chemopreventive compound for lung cancer. First, we will conduct experiments with a small-scale laboratory drying apparatus. Then, we will design, build and test a large scale drying unit that is suitable for production of sufficient material for clinical trials. Finally, throughout the research we will investigate the effects of different processing conditions and chemical compositions on the properties of the dried powders and the stability of the drugs contained in them. This work will define the general rules that govern production of these powders. These rules will provide guidance for the production of powders from different classes of therapeutic or preventative compounds, and hence, will make development of such products rapid and efficient.

The major goals of the current proposal are two-fold. First, there is a translational component in which we will investigate the use of our novel supercritical carbon dioxide-assisting bubble drying (CAN-BD) process to prepare dried powders, which are suitable for pulmonary delivery, of selected compounds. We will also develop methods to scale up the production of the powders to a level that would be sufficient to produce quantities of powders needed for clinical trials. The compounds to be investigated, all of which should have the greatest efficacy and reduced systemic side effects via direct lung delivery, are: 1) alpha-1 antitrypsin, which is used in treatment of emphysema; and 2) 5-lipoxygenase and cyclooxygenase inhibitors, and budesonide, which have application for chemoprevention of lung cancer.

Second, we will use mechanistic studies with these model compounds to define the "rules" that govern effects of processing and formulation conditions on particle properties (e.g., size and agglomeration) and active compound stability during processing and storage in the dried solid. The results from this work will provide guidelines that will allow formulations of new compounds that are delivered via fine powders into the lungs to be developed rationally and rapidly.

Inflammation, Oxidative Stress and Dysphasia in COPD

Silkoff , Philip E.

National Jewish Medical and Research Center

Chronic tobacco abuse causes chronic obstructive pulmonary disease (COPD), and lung cancer. COPD is a lung disease in which large amounts of phlegm are produced, the airways become obstructed, and the air sacs become destroyed, a condition known as emphysema. In the US, there are an estimated 15 million patients with COPD. This year, more than 160,000 people will die of lung cancer.

It is well recognized that persons with COPD are much more likely to develop lung cancer. However, the common causative factors which lead to both COPD and lung cancer have not been identified. In COPD, the airways are inflamed and the lung produces chemicals called oxidants, which damage structures in the lung. Airway inflammation in COPD is characterized by infiltration of inflammatory white blood cells that produce many chemical substances and oxidants. These chemicals and oxidants digest the lung tissue causing COPD and may cause changes in DNA, the genetic material of cells, leading to the development of lung cancer.

At National Jewish, we will identify subjects at high risk for lung cancer, by examining phlegm for pre-malignant and malignant cells and obtaining a CAT scan of the lungs. Our study proposes to examine the connection of airway inflammation and oxidant production with pre-malignant or malignant changes detected in sputum from these subjects. We expect that those subjects with pre-malignant or malignant cells in their sputum will have more severe airway inflammation and higher oxidant production compared to those without such changes.

This study is important because identification of a link between inflammation, oxidant production and precancerous or cancerous states will strengthen the search for effective drugs to combat lung cancer. While quitting smoking is the best preventive measure, most lung cancers develop in ex-smokers, suggesting that the factors leading to cancer persist after smoking cessation. Furthermore, many patients are unable to stop smoking. Effective drugs may include those that suppress inflammation, or neutralize oxidant damage to the lung.

Prediction of Tobacco-Using Groups in Pre-Adolescent Youth

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It is well known that peer influence is a key factor that leads to both tobacco and other forms of substance use among youth. It is also clear that family influence, school performance, and personal characteristics of youth help determine whether youth begin using tobacco. What is not known is how peer groups that protect youth from tobacco use, and those that encourage tobacco use, develop.

In the present study, students in ethnically diverse (primarily Mexican American and non-Hispanic White) schools will be given surveys to complete. The students will be surveyed first when they are in the 4th, 5th, or 6th grade. Over the following three years, students will complete surveys that ask them about their tobacco use and attitudes toward tobacco use, as well as other personal characteristics. They will also complete surveys that ask them who their five best friends are. This survey will be used to track friendship patterns and how they lead either to social groups that do not use tobacco, or social groups that do use tobacco. Teachers and parents will also complete surveys about the youth. The information collected will be used to determine what characteristics of children and what characteristics of the peer groups they belong to either promote or discourage tobacco use. For example, it might be determined that youth who do poorly in school are more likely to use tobacco only if they belong to a tightly-knit group of friends. Alternatively, youth who are isolated from other children may use tobacco only if they have a number of personal risk factors such as having parents they do not feel close to and who use tobacco.

This project will help us determine how prevention programs should be developed. Although altering the behavior of adolescents in a deviant peer group is extremely difficult, if we could identify the factors that lead up to membership in these peer groups we could perhaps intervene before the child enters a social network that supports and encourages tobacco use. In addition, this project will be conducted in schools that have large proportions of both Mexican American and non-Hispanic White youth. Too often research studies are conducted only among majority White youth and conclusions are drawn that may not apply to minority youth. This project will determine whether its findings apply to both cultural groups or whether there are important differences between these groups in how tobacco use develops. These results will have important implications for the design of culturally appropriate prevention programs.

IDEA Projects

Study of the Effect of the Tobacco Carcinogen Benzo(a)pyrene in *Saccharmyces cerevisiae*

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Of all the cancer-related deaths in the United States, lung cancer is the leading cause. Every year more people die of lung cancer than breast cancer, colon cancer, and prostate cancer combined. Tobacco smoking is the single most important risk factor for lung cancer. More than 60 carcinogens (cancer-causing chemicals) have been identified in tobacco smoke. Benzo(a)pyrene, which is present at 20 to 40 ng (1 nanogram equals one hundredth millionth of a gram) per cigarette, is the best studied and one of the strongest carcinogens known by far. Benzo(a)pyrene itself is not a very active compound, but it poses risks to a normal cell in our body in several ways. It is converted into an active carcinogen named BPDE inside the cells by enzymes that normally works in the metabolic pathways. BPDE can bind directly to DNA and cause errors in the genetic information coded by DNA. Cancer arises when accumulation of errors in genetic information inside certain cells eventually lead to their uncontrollable growth and propagation. One other way benzo(a)pyrene can cause damage is that it can induce an increase of free radicals inside the cell, and these free radicals can inflict damage to both DNA and other important molecules of a cell such as proteins and lipids.

We want to understand what happens when normal cells are exposed to benzo(a)pyrene and what defense mechanisms are activated to fight against its damaging effects. Such understanding will aid the design of drugs to fight lung cancer. We choose to use the baker's yeast, a single cell organism that is much simpler than human, in our experiments. It grows fast and is relatively inexpensive to work with, and modern biological techniques are readily available. More importantly, many of the mechanisms are similar between yeast and human cells, so what we have learned from yeast studies can lay the foundation for future studies on human.

Tobacco and Schizophrenia Affect Prenatal Brain Development

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Prenatal tobacco exposure has direct long-term effects on the ability to inhibit behavior, whether this behavior is measured by parental report, neuropsychological tests, or physiological response to auditory stimulation. The deficits in inhibition are described for post-natal periods from infancy to late school age, yet the effect of tobacco exposure is presumed to be prenatal, where tobacco exposure impairs normal brain development. Despite this assump-

tion, there is no direct evidence that the effects of prenatal tobacco can be identified prenatally, primarily because there has been no attempt to measure inhibitory functions at this stage of our development. This proposal expands the measurement of physiological response to auditory stimuli into the prenatal and early postnatal period (aim #1), during a time that the brain development associated with inhibitory processes is occurring. This proposal will examine the effect of prenatal tobacco exposure on prenatal and early postnatal brain inhibitory processes (aim #2). This novel approach will increase our understanding of when, during prenatal development, the health risks of tobacco use on brain development are occurring.

An additional focus of this proposal centers around genetic vulnerability to severe psychotic mental illnesses, such as schizophrenia. Of all groups studied, cigarette smoking is highest in individuals with severe mental illnesses such as schizophrenia. Not only do over 80% of people with schizophrenia smoke, they smoke "harder" (sucking more nicotine out of a cigarette than other smokers) and have greater difficulty quitting. This very high level of cigarette use is presumed to be secondary to the role of nicotine in the development of the illness. One gene that is likely involved in schizophrenia is a gene that makes a brain receptor responsive to nicotine. People with schizophrenia have many fewer of these nicotinic receptors. One of the reasons people with schizophrenia smoke so much is that they are trying to get a lot of nicotine into their body and highly activate their limited number of receptors. This nicotinic receptor also has effects on how individuals respond to simple sounds, especially the ability to inhibit responding to sounds with no relevant information, and this response can be measured in newborn infants and maybe even before birth. This impaired ability to inhibit response to auditory stimuli is similar to the deficits found with prenatal tobacco exposure, perhaps because tobacco acts prenatally on the same nicotine receptor that is deficient in genetic vulnerability to schizophrenia. Aim #3 of this proposal focuses on whether the effects of genetic risk for schizophrenia and prenatal tobacco are additive or interactive, where the effect of tobacco is even greater in genetically vulnerable individuals.

This proposal should lead to greater understanding of both the negative health effects of tobacco on early brain development and on the genetically-mediated prenatal brain development in those most at risk for later tobacco use.

Tobacco Use by Foster Care Youth and Professional Responses

Struhsaker Schatz, Mona C.

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Over 500,000 children and youth live in substitute care because of abuse and neglect; roughly 10,000 children are fostered in Colorado each year. In order to increase our knowledge about the role of tobacco use among special populations, this tobacco use research study examines the role of tobacco use among foster children and youth, aged 10-17. These youth are often excluded from research studies or not identifiable in generalized study populations, yet their psychological, emotional, familial, and social problems may contribute to a higher rate of use of tobacco products than among the general youth population. A second part of this study will examine how a respondent group of professional workers and foster parents help these foster children with smoking cessation and smoking prevention.

Postdoctoral Fellowships

Chronic Nicotine Effects in Mouse Hippocampal CA3 Region

Dobelis, Peter

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Nicotine is the active component of tobacco that supports chronic tobacco use. Nicotine interacts with a specific class of receptor proteins in the brain termed the nicotinic acetylcholine receptors, of which several types are known to exist. These nicotine receptors are located in several brain regions, one of which is the hippocampus, an area critical for learning and memory and thought to be involved with certain aspects of nicotine addiction.

This study will investigate chronic nicotine effects in the CA3 region of the hippocampus from inbred mice. This region regulates the activity of the hippocampus and is rich with nicotinic receptors. Inbred mice are used as an animal model because they are genetically identical (thus decreasing experimental variability), and many studies of nicotine's effects have used them as subjects, providing baseline information for this study.

The first part of this study will determine which types of nicotinic receptors are associated with the various cell types known to exist in the CA3 region. There are several different cell types in the hippocampus, each type having a specific role in hippocampal function. There are also at least two types of nicotinic receptor expressed in the hippocampus, and previous studies in the CA1 region of the hippocampus suggest these receptors are found on some cell types and not others. Since the organization of the CA3 region differs from that of CA1, and since CA3 drives hippocampal activity, determining which type of nicotine receptor is associate with which cell type is crucial to understanding how these receptors effect hippocampal function. The results obtained from these studies will lay the foundation for the experiments determining chronic nicotine effects on hippocampal function.

The second part of this study will determine how chronic nicotine treatment effects these receptors and the cells they're associated with, and how this leads to changes in

hippocampal function. Numerous studies have shown chronic nicotine exposure results in changes in the number and function of nicotinic receptors and in nicotinic receptor-mediated release of neurotransmitters. However, little is known about how chronic nicotine exposure changes the way cells in the brain communicate with each other. By gaining a better understanding of how chronic nicotine changes brain function, treatments may be developed that prevent or reverse these changes and thus possibly preventing addiction to nicotine.

Dissertation Awards

Nicotine Signal Transduction in the Central Nervous System

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Tobacco use is the most prevalent public health problem in Colorado. The basis of this problem is nicotine addiction. Effective treatment of tobacco use therefore requires an understanding of the underlying mechanism of nicotine addiction. The actions of nicotine are mediated by proteins found on nerve cell membranes called neuronal nicotinic acetylcholine receptors (nAChRs). As a result, understanding nicotine addiction requires an understanding of the mechanisms through which nAChRs can cause long-term changes in brain function. Long-term changes in nerve cells are thought to be a result of a maintained change in the amount and type of proteins expressed in the cell. This change can ultimately result in altered behavior. One place where protein expression is regulated is the cell nucleus. The nucleus contains deoxyribonucleic acids (DNA), which carry the specifications for proteins that are made in the cell. Portions of the DNA are transcribed into ribonucleic acids (RNA), which can then be translated into proteins.

In this proposal, we wish to determine if nicotine can cause a change in the amount of a protein called brain-derived neurotrophic factor (BDNF). BDNF has been shown to have important effects on the function of nerve cells. If nicotine increased BDNF levels it could help explain some of the long-term effects of nicotine in the brain. We can measure the amount of BDNF using a technique called reverse-transcription polymerase chain reaction (RT-PCR) and in situ hybridization.

In addition, we also wish to determine the intermediate steps that connect nAChRs on the cell membrane to the DNA encoding BDNF in the nucleus. There are some clues that suggest a potential mechanism. First, it is known that changes in protein expression are dependent on changes in calcium levels within the cell. Therefore, we wish to determine the calcium dependency of nicotine-induced changes in BDNF expression. Second, calcium can activate a protein called the calcium/cyclic 3',5'-adenosine monophosphate response element binding protein (CREB) and increase BDNF expression. CREB binds to DNA and in part, regulates what proteins are expressed in a cell. Consequently, we would like to know if nicotine could activate CREB. Calcium does not activate CREB directly; rather it acts through a family of intermediate proteins called protein kinases. As a result, we are also interested in determining what members of this protein family could be activated by nicotine in nerve cells. We will use techniques called Western blotting and immunocytochemistry to determine the effect of nicotine on both CREB and protein kinases. These techniques utilize antibodies that recognize the activated CREB or protein kinase in the nerve cells.

This proposal will, for the first time, determine a mechanism by which nicotine can result in long-term changes in the brain and provide insights in to the origins of nicotine addiction. Knowledge of these mechanisms will allow development of drugs that will be useful in the treatment of tobacco use.

Epithelial Injury by Cigarette Smoke: Sulfur Metabolism

Panagiotidis, Michail I.

National Jewish Medical and Research Center

Cigarette and other tobacco smoke can cause a variety of lung and heart diseases. Because smoke contains a large number of toxic, reactive chemicals called "free radicals", it has severe effects on sulfur containing amino acids and antioxidants in the lung and in the body. The damaging reactions of these free radicals and loss of these antioxidants are thought to play an important role in many lung (chronic obstructive lung disease (COPD), chronic bronchitis, and possibly lung cancer) and cardiovascular (heart attack, stroke) diseases related to smoking. Smoking can deplete some of these nutrients (amino acids) and antioxidants in the short run (right after smoking) and then there can be an increase in them later on as the lung and the body try to compensate for the shortage. In this project, we will study how these changes happen and how they may affect the DNA of lung cells so as to predispose to lung cancer and possibly to cause depletion of vital nutrients to lung cells. Such a depletion or deficiency could occur in this situation because it appears that there may be a "tug-of-war" going on between two ends of an important biochemical pathway - one end which producing an important antioxidant called glutathione and the other end which produces an important sulfur-containing molecule called SAM (S-adenosylmethionine). SAM is an important building block for cells and many of their components. We do not yet know which end of this pathway "wins" in a cigarette smoker with a marginal diet and a poor intake of sulfur-containing foods. Our studies will help us determine this in a cell model for exposure to cigarette smoke performed in our laboratory.

These events also may be pertinent to other processes in

the lung such as cell growth and could be relevant to why infants born to cigarette smokers have smaller airways and are predisposed to asthma. Finally, another event related to these processes is the elevation of an amino acid called homocysteine in the blood of smokers. This homocysteine may predispose these individuals to cardiovascular diseases like heart attack and stroke.

Theory and Research Confounds in Anti-Substance PSA Study

Wagner, Carson B.

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Traditionally, research on public service announcements (PSAs) has looked at the ways in which they can stimulate rational behavior, usually among young people, that will prevent them from abusing such substances as cigarettes, alcohol, and illicit drugs. The problem with this is that a good deal of theory and research suggests that unreasoned, "gut feeling" reactions may be more important in defining behavior. There is emerging evidence, in fact, which suggests that PSAs can make adolescents curious to experiment with substances. The current research is examining the extent to which we can use communication to alter unreasoned responses and avoid unintended outcomes of PSA consumption. The research focuses on the ability of various types of public service ads to do so situationally, and it highlights theoretical and methodological oversights in prior PSA research toward explaining the gaps that exist between ad intentions and outcomes. Our preliminary experimental results suggest anti-substance ads are better at changing well-reasoned responses, while "gut feeling" modification is harder to detect than theory implies. Although the processes we demonstrate lend promise for using ads to protect youth who face decisions under optimal conditions, our research shows that PSAs might be less useful for dissuading adolescents who do not always have the ability and motivation to think through rationally decisions about cigarettes, alcohol, and other drugs.

Household Factors and Passive Smoke Exposure of Preschool Children

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Environmental tobacco smoke exposure (also known as passive smoke exposure) is harmful to pre-school children as it is a source of illness for them. An estimated nine to twelve million children in the United States are exposed to second hand smoke in their homes. While their exposure is involuntary, research shows that they are particularly susceptible to health risks occurring from second hand smoke. Most children of smokers are exposed at home. Efforts to prevent environmental tobacco smoke exposure focus on parents and household members and the factors and forces that influence their tobacco use.

The dilemma of how environmental tobacco smoke can be reduced in the home is a public health concern and deserves further study. Information is readily available on the unhealthy effects of smoke exposure, but little is known about how smoking-related practices by family and household members affect children. Tobacco control policies that reduce smoke exposure have been used effectively in workplaces, restaurants and other public places. How similar policies might be developed and used in homes is little understood. Understanding factors that contribute to household policies regarding smoking is necessary if effective methods for reducing tobacco smoke exposure of children are to be developed and implemented.

This research will explore the factors that families identify as being important in developing rules about smoking in their homes and cars. Families who receive services from school based health centers, and who have children age 0-4 living at home, will be selected for study. Parents will be interviewed to discover how they develop rules for smoking in their homes. Once identified, a survey will be developed and given to other families with young children to determine how these factors influence the smoking polices which operate in the home. The saliva or urine of children in these families will be checked for levels of cotinine, a byproduct of nicotine. The cotinine levels will be compared to the smoking policy in the home. Understanding the reasons for smoking policies will help researchers and health professionals develop better programs and interventions with parents for preventing and/or reducing smoke exposure, thereby reducing the risk of young children for illness.

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