

Director's Message

by Charles A. DiSogra, Dr.P.H., M.P.H.

Talent, creativity, diversity, economic strength, and political willpower keep California on the cutting edge of scientific, technological, and social progress. Consistently, many discoveries, ideologies, and movements originating in this state are soon followed by the rest of the country. One of the most cited examples is California's pioneering public health efforts in tobacco control. The changes in social norms regarding tobacco use and environmental tobacco smoke and the resultant 21% reduction in the prevalence of smoking are some of the outward measures of acknowledged success.1 However, the progress achieved in tobacco-related disease research, particularly the funding of unique innovative studies is a less recognized yet substantive success story.

Since 1990, TRDRP has competitively awarded \$351 million to support research on the prevention, causes, and treatment of tobaccorelated diseases in about 80 prestigious research centers across the state. The caveat in using tobacco tax dollars to fund research is that as program success in tobacco control reduces tobacco consumption, the total amount of tobacco tax dollars available for research is likewise reduced. This has been exacerbated over the past five years by the state's allocation of research dollars to cover shortfalls in the Department of Health Services' administrative budgets. As a result, TRDRP currently gets only 74% of the Proposition 99 funds designated for tobaccorelated disease research.

See "Director" page 4

Also in this Issue

Public Health and Big Tobacco	5
Stroke Funding Needed	8
Hopper Diversity Awards	10
TRDRP Update	12



Tobacco Research & The False Assumptions About Latinos

by Francisco O. Buchting, Ph.D.

"The long-awaited, often-anticipated Latino majority in California is no longer a theoretical, abstract, future possibility. It has arrived, as of the third quarter, 2001."

"From a long-term perspective, in passing this milestone, California has returned to its historic pattern of a Latino majority among its children, which was the norm for the state until the late 1850s."

The excerpts above are from a 2003 study from the Center of the Study of Latino Health and Culture that demonstrated beginning in the third quarter of 2001, more than half of the births in California were Latino.¹ Population projections for the state of California estimate that Latinos as a group, both adults and children, will become the majority between the years 2020 and 2030.²

The U.S. Census 2000 provided a snapshot of the dynamic growth of Latinos in the U.S. The number of Latinos in the U.S. grew from 22.4 million in 1990 to 35.3 million in 2000, an accelerated 60% increase when compared to a total U.S. population increase of 13.2%. In California, Latinos represent approximately 33% of the population, about 11 million people. California is reported to be the leader among the top 10 states with the largest number of Latino residents.³ With exceptional growth in the Latino population, both nationally and in California, and the diversity among Latinos (e.g., country of origin, place of birth, acculturation, education, SES, language dominance, etc.), some questions come to the forefront in tobacco-related research and tobacco control: Is it a problem to think and treat Latinos as a

Latinos Continued from cover

homogenous group? Is it correct to assume that surveillance and research results accurately reflect this diverse group? If Latinos are not a homogeneous group and if the assumption is false, one has to wonder if tobaccorelated studies are misleading. Do they fail to accurately capture the rate of tobacco use and its health and economic burden in the different Latino groups?

We have reached the tipping point where Latinos can no longer be treated as a homogeneous group in tobaccorelated research. Even though it may be expeditious and ease sampling and analytical issues in research methodology, it is inaccurate to portray tobacco use and its effects for a single category labeled "Latino" due to the growing heterogeneity in this population.

SURVEY SAYS—

For several years, the mantra has been that "When looking at smoking rates by race/ethnicity, Latinos have the second lowest rate in the U.S. and in California." This assertion is based on national⁴ and California⁵ surveys. But what does a 16.7% national or 14.8% California smoking rate for Latino adults really mean? Are Latinos in California really smoking less than Whites (18.9%), African Americans (18.1%), or American Indian/Alaska Natives (30.3%), but smoking more than Asians (13.1%)? Are these numbers giving a skewed picture of tobacco use for the Latino population in the U.S. and in California?

All one needs to do is to begin looking at group differences within the diverse Latino community and the notion that tobacco use in the Latino community is not as high as other groups becomes questionable. It is true that Latinos as a single category have the second lowest smoking rates, but that is assuming that looking at Latinos as a single category is valid.

By lumping the Latino population into one group, the assumption is made that all Latinos are the same. The fact is that not all Latinos in the U.S. are the same due in part to the Latin American country of origin, acculturation, number of generations in the U.S., and so on. Following are a few examples of how the conclusion that "Latinos have the second lowest smoking rate" can be problematic in research and in setting public health policy.

Gender and Group differences

Many researchers take gender differences into account in their studies. In this population, gender difference in smoking rates for males and females in the Latino community have been consistently found in both national surveys (22.7% Males, 10.8% Females, 16.7% overall)⁴ and California surveys (19.6% Males, 7.8% Females, 13.7% overall).⁶ In fact, the overall smoking rate for Latinos is driven down by the gender gap. This is true for other comparison groups. But what happens when you begin to look at country of origin by gender?

The smoking rate for the Latino population becomes a more complex issue when we look at the rate of smoking by different Latino groups as shown in Table 17 and by gender as shown in Table 2.7 In both Tables 1 and 2, it should be noted that people with Mexican ancestry self identify into two categories: Mexican American and Mexican/Mexicano. In Table 1, even though the overall smoking rate for Latinos is 18.3%, the range of smoking rates varies from 15.8% for Mexicans/ Mexicanos to 24.7% for Puerto Ricans. Table 2 also shows that there exist gender differences in smoking rates when

Table 1

Percentage of Current Cigarette Smokers among Latino Groups in United States, ages 18+

Characteristic Group	
Mexican American	70 19.7
Mexican/Mexicano	15.8
Puerto Rican	24.7
Cuban American	19.8
Other Latino	18.0
Overall	18.3
Source: National Health Interview St 2000, aggregate data	urveys, 1999 and

Table 2

Percentage of Current Cigarette Smoking by Gender and Latino Groups in United States, Ages 18+

Μ	F
%	%
24.9	15.1
22.5	8.3
28.5	21.7
22.1	14.0
	M 24.9 22.5 28.5 22.1

Source: National Health Interview Surveys, 1999 and 2000, aggregate data

looking at each Latino group as well as among Latinas. In fact, the low smoking rate for Latinas begins to take on new meaning depending on which group one is looking at, e.g., 8.3% smoking rate for Mexican women/Mexicanas compared to 21.7% for Puerto Rican women (which is almost equal to the national rate of 20.0% for white women).4 It seems that Mexican women/Mexicanas are the ones that are driving the overall smoking rates for Latinas to being the second lowest. A 2001 study by Perez-Stable et al. found differences in current smoking prevalence among Latinos by country of origin (Mexican American, Cuban American, Central American, South American, and Puerto Rican) and differences between and within males and females.8 Likewise, the January 30, 2004 MMWR reported differences in prevalence of cigarette use among four Latino populations (Mexican, Puerto Rican, Central or South American, and Cuban).9 A follow-up question is-Do U.S. born Latinos smoke more than foreign born Latinos?

Foreign born vs. U.S. born

When it comes to tobacco use, it matters whether a Latino is foreign born or U.S. born. In Table 1, the rate of smoking is lower for Mexican/Mexicano (15.8%) compared to Mexican Americans (19.7%).⁷ Similarly, in Table 2 the smoking rate is lower for Mexican/Mexicano than Mexican American between gender and within gender. A study by Baluja et al.(2003) looking at immigrant status and smoking found higher smoking rates for U.S.born Latinos (17.9%) compared to for-

Latinos Continued from page 2

eign-born Latinos (13.0%).¹⁰ The study also found gender difference in smoking rates for U.S.-born Latinas (14.4%) compared to foreign-born Latinas (6.7%).

With all Latinos combined making up one of the largest ethnic groups immigrating to the U.S., the impact of smoking norms in the country of origin needs to be considered. This is especially true since California has some of the most advanced tobacco control policies in the U.S. It should be of no surprise that smoking rates in Latin American countries vary by country from a low of 12.9% in Panama to a high of 49.5% in Uruguay. Similarly, gender differences for smoking rates exist within each Latin American country.¹¹

There are many other examples of observed differences in surveillance and research studies among the different Latino groups for cessation rates, smoking during pregnancy, youth uptake, and youth tobacco-use rates to name a few. The fact remains that by looking at Latinos as a heterogeneous group, the overall picture of tobacco for the Latino community dramatically changes.

IS THE TOBACCO INDUSTRY AHEAD, AGAIN?

The tobacco industry has realized that the projected numbers for Latinos represent an under-exploited market to the industry. For example, in 2001 Philip Morris ranked 10th among the top 60 U.S. Latino market advertisers with gross media expenditures totaling \$25 million. By 2002, only 12 months later, it is no accident that Philip Morris had become the second largest Latino market advertiser by more than doubling their gross media expenditure to \$64 million.12 What may be even more appealing to the tobacco industry is that the growing Latino market is a relatively young demographic group. For example, Latinos of Mexican origin have the largest proportion (38%) of individuals under the age of 18 of all other ethnic and Latino groups.13 An added bonus for the tobacco industry is that Latinos, as consumers, tend to be brand faithful.¹⁴

Furthermore, the tobacco industry continues to provide financial support and is highly visible in sponsorship to a broad range of Latino civic organizations, cultural events, and community festivals to position itself as a good corporate citizen in Latino communities.¹⁵ The reality is that the tobacco companies will definitely increase their aggressive targeting of all Latino groups in efforts to maximize their own market share.

THE RIGHT THING TO DO

There are many significant gaps in research on tobacco use and the different Latino groups in the Latino community. The need for the most accurate and robust research findings is great. Even though this article did not directly address the issue of lumping Latino youth into one category, analogous arguments can be made to the ones put forward for adult Latinos. Studies, focusing on the different Latino groups, both adults and youth, are needed to better understand tobacco use and the burden of tobacco-related diseases among these Latino groups. In addition, there is very little effort, if any, looking at Latino groups who are severely understudied, e.g., Latino migrant farm workers or LGBT Latinos. Likewise, international studies looking at tobacco use in Latin Americans and among Latino immigrants in the U.S. are needed.

It might be concluded that the tobacco industry has a better understanding of the market segmentation of current smokers, youth market, and future smokers in the Latino communities than is found in current tobacco-related research. The challenge for researchers and public health professionals is clear and can no longer be disregarded-the "Latino community" needs to be recognized and treated as the heterogeneous group they are. Not doing so means that future tobacco-related research and public health policy and programs to address tobacco use in the soon-to-be "Latino majority" in California would be grossly inadequate.

TRDRP has a history of funding Latino studies and continues to welcome applications in all research areas that focus on the heterogeneity of Latinos and the differences within these communities. The findings from these studies will be extremely valuable in order to gain the needed knowledge about the emerging majority of the combined Latino communities in California and will

See "Latinos" page 4

Latino or Hispanic?

Most individuals who can trace their family ancestry to Mexico, Central America, South America, and several islands in the Caribbean strongly prefer which term they use to describe themselves as either Latino or Hispanic. What is the difference and does it matter?

Hispanic is a descriptor that has replaced terms such as "Spanishsurnamed" and/or "Spanish-speaking" as a way to classify a segment of the population in U.S. government documents. People who consider their heritage linked to Spain also use it. The use of the term Latino (men) or Latina (women) is an ethno-social-political movement who see the term Hispanic as claiming association with Spanish conquistadores/colonialists and also as an imposed U.S. government term. Latino is seen as more politically correct because it acknowledges the ancestral roots in America, it incorporates people of Latin American heritage whose primary language is not Spanish (e.g., Brazil), and many women prefer the gender-specific term "Latina" as a way of creating a sense of unity and sisterhood.

Hispanic or Latino/Latina? It does matter!

Latinos Continued from page 3

assist in tobacco-control efforts for these diverse communities and for the state.

The author wishes to acknowledge and personally thank Dr. Ralph Caraballo from the Centers for Disease Control and Prevention, Dr. Lourdes Baezconde-Garbanati from the University of Southern California, Dr. Vilma Cokkinides from the American Cancer Society, Nora Manzanilla from the Office of the Los Angeles City Attorney, and Cecilia Portugal from the Hispanic/ Latino Tobacco Education Partnership for their generous contribution, sharing of data, and expertise to this article.

References

- Hayes-Bautista, D.E., Hsu, P., Perez, A., Kahramanian, M.I., 2003. The Latino Majority has Emerged: Latinos Comprised more than 50 Percent of all Births in California. Center for the Study of Latino Health and Culture, UCLA.
- State of California, Department of Finance, Population Projections by Race/Ethnicity, Gender and Age for California and Its Counties 2000-2050, Sacramento, California, May 2004.
- 3. U.S. Census Bureau, Census 2000. www.census.gov
- CDC. Cigarette Smoking Among Adults -United States, 2002. MMWR May 28, 2004, 53(20), 427-431.
- California Department of Health Services, Tobacco Control Section, March 2003. Age-adjusted smoking prevalence among California adults by race/ethnicity group.
- California Health Interview Survey, 2001. www.chis.ucla.edu
- Caraballo, R., 2002. Tobacco Use Among Latinos Living in the U.S. Paper presentation at the Hispanic/Latino Conference on Tobacco Prevention and Control.
- Perez-Stable EJ, Ramirez A, Villareal R, Talavera GA, Trapido E, Suarez L, Marti J, McAlister A., 2001. Cigarette smoking behavior among US Latino men and women from different countries of origin. American Journal of Public Health, 91(9), 1424-1430.
- CDC. Prevalence of Cigarette Use Among Racial/Ethnic Populations -United States, 1999-2001. MMWR January 30, 2004, 53(3).
- Baluja K.F., Park J., Myers D., 2003. Inclusion of Immigrant Status in Smoking Prevalence Statistics. American Journal Public Health, 93(4), 642-646.
- Tobacco Control Country Profiles, Carrao, M.A., Guindon, G.E., Sharma, N., Shokoohi, D.F. (eds). American Cancer Society, Atlanta GA, 2000.

- 12. Hispanic Business Magazine. Top 60 Advertisers in the Hispanic Market, 2001 and 2002.
- U.S. Census Bureau, Census 2000. www.census.gov
- 14. Gordon, K.T. ¿Se Habla Español? Entrepreneur magazine - June 2003.
- Tobacco Corporate Donations In California 2002-2004 Tobacco Industry Monitoring Evaluation - The TIME Project - University of Southern California.

Director Continued from cover

The effect of this budget squeeze is that the scientific advancements and discoveries essential to future progress are systematically slowed in California due to reduced TRDRP funding opportunities. Consequently, California scientists and their home institutions are being deprived of a competitive edge in the very competitive national research economy. TRDRP funding initially helps to support some of California's most innovative scientific ideas with a relatively low investment of tobacco tax dollars. This in turn potentially allows California scientists to leverage that investment in conjunction with their scientific findings to obtain substantially larger awards from federal and private sources for continuation research. Those larger awards, most from the National Institutes of Health, also create more jobs in the state. In effect, reduced state tobacco tax funding for research is not just a blow to scientific progress but is also bad for the research business in California

The short-term solution is clear: The state's Tobacco Education and Research Oversight Committee has called for an end to this diversion of research funds and for the full 5% of Proposition 99 tobacco tax dollars to be appropriated to TRDRP as intended by California's voters and the state legislature. This restoration of the diverted funds would enhance the economic competitiveness of California's research institutions and their scientists. More importantly, it will give additional assurance that progress in the prevention, causes, and treatment of tobacco-related diseases will not be further impaired.

The need for scientific progress is one of the key reasons for the original passage of Proposition 99 and for the creation of TRDRP. A degree of moral imperative is attached to this point, since the immediate future provides an ironic yet somewhat predictable situation. With less tobacco-related research being funded (due to successes in tobacco control), hoped-for medical advances may consequently be longer in the making. The irony is that the most likely beneficiaries of this research are the aging generation of voters who passed Proposition 99 some fifteen years ago. These voters include the smokers, ex-smokers, and those who had been exposed to secondhand smoke who wanted more research to be done. Today they are seeing progressively fewer tobacco-tax dollars going to TRDRP to do that potentially beneficial research. Diverting any of these already shrinking research dollars leaves the Proposition 99 voters shortchanged.

Referrences

 Gilpin EA, White MM, White VM, Distefan JM, Trinidad DR, James L, Lee L, Major J, Kealey S, Pierce JP. "Tobacco Control Successes in California: A Focus on Young People, Results from the California Tobacco Surveys, 1990-2002." La Jolla, CA:University of California, San Diego; 2004. (Prepared for the California Department of Health Services Tobacco Control Section)

STRANGE BEDFELLOWS:





Public Health and Big Tobacco

by Phillip Gardiner, Dr.P.H.

The age old adage, "politics makes strange bedfellows," was never truer than in the battle lines that were drawn and the camps established in support and against Senate Bill 2461. The Kennedy/ DeWine Bill (S2461) passed the Senate 78 to 15 as a rider to HR4520, the Foreign Sales Corporation/Extra-territorial Income Act (FSC/ETI). The FSC/ETI is a bill intended to generally reduce a broad range of punitive tariffs on American exports and when coupled with the Kennedy DeWine rider, would give the Food and Drug Administration (FDA) regulatory powers over the manufacturing and marketing of cigarettes.1 However, House and Senate conferees denuded the tax bill of FDA authority. something not entirely unexpected. Still, the real story was the 21st century alliances that were formed: Philip Morris and the American Public Health Association calling the Senate vote "historic," and the New York Times, some tobacco researchers, and RJ Reynolds denouncing it, the whole tobacco control scorecard was re-arranged, albeit temporarily. Below, the main provisions of S2461 are reviewed along with the unique cast of characters in favor and against this legislation.

A Shotgun Marriage

Some could argue that passage of S2461 was historic in that it was the first time

that either house of government voted in favor of regulating tobacco by the FDA. In its broad strokes, the bill would have given the FDA the authority to regulate the sale, distribution and advertising of cigarettes and other tobacco products. Moreover, the FDA could bar advertising aimed at children, end vending machine sales, make more conspicuous warning labels mandatory, and prohibit unverified reduced health risk claims. Significantly, tobacco companies would have to give the government a list of all ingredients and additives in tobacco products and the FDA could outlaw certain additives. While the bill did not allow the FDA to eliminate nicotine, the bill would have allowed it to mandate the reduction of this substance, the active addicting ingredient in tobacco smoke.2

The regulatory authority for the FDA was contained in a rider to the FSC/ETI that not only includes FDA oversight provisions, but also provides a whopping \$12 billion "buyout" for tobacco farmers. Co-author, Senator Mike DeWine (R-Ohio), claimed that the legislation, now defunct, "was a shotgun marriage. . . . that made sense."2 Senator Jim Bunning (R-Kentucky) who has historically opposed government regulation of tobacco stated unequivocally that, "I think FDA regulation is a very steep price to pay for a buyout, [but] if that's the only way to get my growers relief, this senator will vote to pay it."2 S2461 would have required cigarette manufacturers to

pick up the tab to buy out tobacco farmers, although \$3 billion in remaining Phase II payments from industry to farmers would be stopped.

On the House of Representatives side of Capitol Hill, a companion bill, HR4433, was distinctly different than the Senate version of the Bill. The House bill not only would have provided for a \$9.6 billion grower "buyout" financed by the United States Treasury (read: your tax dollars) as opposed to the tobacco industry, but it also didn't contain any language giving the FDA regulatory authority over tobacco products. Under HR4433, however, the \$3 billion in remaining Phase II payments from industry to farmers would still be required.

Public Health and Philip Morris Hand in Hand?

The Senate bill was hailed by everyone from Representative Henry Waxman (D-California) and the American Public Health Association (APHA) to Philip Morris and Smith Barney. With this starting line-up, the proposed legislation has indeed spawned very strange bedfellows. "This is not only a win; it is a win, win, win," said Georges Benjamin, M.D., F.A.C.P., executive director of APHA. "This amendment is a victory for public health, a victory for taxpayers and a victory for farmers. We applaud the leadership in support of taxpayers and public health shown by the sponsors



Bedfellows Continued from page 5

of this amendment."3 The Campaign for Tobacco Free Kids (CTFK), speaking for the American Cancer Society (ACS), American Lung Association (ALA), and the American Heart Association (AHA) dubbed the legislation, "the strongest, most bipartisan and most comprehensive bill ever introduced to grant the FDA authority over tobacco products."4 Aligned with these public health giants is Philip Morris, the tobacco industry leader. Not to be outdone by the praise that was heaped on the proposed legislation, Steven C. Parrish, Senior Vice President, Corporate Affairs, Altria Group, stated that, "Altria Group and our domestic tobacco company, Philip Morris USA, enthusiastically endorse passage of a final FSC/ETI bill that contains the DeWine/Kennedy FDA proposal in its entirety, and we will strongly oppose any and all amendments to this language during the upcoming House/ Senate Conference Committee."

In a joint statement, ACS, ALA, AHA and the CTFK acknowledged that "A few compromises were necessary in order to achieve such bipartisan support, and while we had hoped to expand states' rights to give them unfettered authority to regulate tobacco marketing, that was not possible in order to reach an agreement with Congress."⁴ This coalition of the Republican-controlled Senate, Democratic health advocates Waxman and Kennedy, tobacco farmers, Philip Morris and the major organizations in the public health field was indeed an historic and unprecedented alliance in the fight for FDA regulatory control over tobacco products.

Tobacco Researchers Denounce Senate bill; RJ Reynolds does too!

While the Senate bill was clearly seen by some as an advance for the tobacco control movement, there were others who were quite dismayed. Michael Siegel, M.D., M.P.H., a professor at Boston University's School of Public Health, and a noted tobacco control researcher, in a lengthy and scathing critique argued that S2461 would have given the FDA "rather narrow authority, with its hands tied in terms of banning tobacco products, eliminating nicotine, establishing a prescription-only access system, raising the legal age of purchase, and regulating the sale of tobacco in certain types of establishments."6

In a series of fact sheet emails, Dr. Siegal critiqued S2461. Siegel argued that the Senate Bill missed the target altogether: "It requires FDA to ban any tobacco product that contains a severely harmful chemical defect, but explicitly prevents FDA from doing the same with tobacco products that contain thousands of severely harmful chemicals, so long as those chemicals are ordinarily contained in tobacco products. It provides for stringent regulation to prevent adulterated or misbranded products, but leaves the "pure" and "properly branded" deadly products largely unregulated. It requires that manufacturers report any adverse health effects of its products that are UNEX-PECTED, but the EXPECTED 450,000 or so deaths per year due to these products require no special attention."⁷

Dr. Siegel goes as far to say that S2461 would not have saved lives, if passed, it would probably lead to increases in tobacco-related deaths! Siegel asserted: "This legislation is likely to result in increased, not decreased deaths from tobacco products, for the following reasons: It will make it virtually impossible to research, develop, introduce, and market new potentially less hazardous tobacco products; it will undermine current and future litigation; and, It will reduce the public's perception of the inherent harms of cigarettes."8 Siegel contends that the constellation of public health groups who supported this legislation had essentially sold out to Philip Morris; they were supporting legislation that would increase tobaccorelated deaths.

Another tobacco researcher, Michael Cummings, Ph.D., research scientist in cancer control and epidemiology at Roswell Park Cancer Institute, echoed Dr. Siegel's point of view, stating: "It [the legislation] requires FDA to ban any tobacco product that contains a severely harmful chemical defect, but prevents FDA from doing the same with tobacco products that contain thousands of severely harmful chemicals, so long as those chemicals are ordinarily contained in tobacco products. It bans the presence of strawberry, grape, orange, clove, cinnamon, pineapple, vanilla, coconut, licorice, cocoa, chocolate, cherry, or coffee in cigarettes, but does not inherently disallow the presence of hydrogen cyanide, carbon monoxide, N-nitrosodimethylamine, benzene, radioactive polonium 210, or nitrogen dioxide."9

Well, if some tobacco researchers did not think the bill was ready for primetime, some of Philip Morris's friends in the tobacco industry were also denounc-

Bedfellows Continued from page 6

ing it, albeit for their own reasons. Mincing no words, Tommy Payne, executive vice president of external relations for R.J. Reynolds Tobacco Company stated: "If Congress is serious about giving tobacco growers financial relief, it will adopt the House version of the tobacco quota buyout bill."¹⁰ Furthermore, speaking about the Senate version Payne asserted that: "The Kennedy/ McConnell amendment is ill-conceived, imperils the viability of a tobacco quota buyout and creates an overwhelming competitive advantage for Philip Morris."¹⁰

The New York Times and the Wall Street Journal added their voices to the opposition. The Times agreed with the Public Health Community that FDA oversight of tobacco is long overdue; however, their editorial page considered the current bill "ill advised . . . the price for getting senators from tobacco-growing states on board is an unseemly \$12 billion handout to tobacco growers, who have already been coddled for far too long by protectionist quotas meant to keep out cheaper foreign-grown tobacco ... It creates a disastrous precedent for a nation that direly needs to start dismantling other crop supports, both for domestic budgetary reasons and to comply with international trade laws."11 Interestingly, the Wall Street Journal stated unequivocally that "what's wrong with the tobacco deal approved by the Senate last week, [is] that its biggest fan is America's biggest cigarette-maker, Philip Morris."12

Hence, the lineup of who opposed the Senate Bill included the Republican controlled House of Representatives, some tobacco researchers, RJ Reynolds, the New York Times and the Wall Street Journal. Joining this disparate group was the Smokefree Pennsylvania, a tobacco control organization. They, along with others have lobbied "Congress to eliminate both . . . FDA regulation [and] tobacco quota buyout proposal . . . from the FSC/ETI legislation. Fair and effective FDA legislation can be crafted next session of Congress."¹³

Divergent Views in the Tobacco Industry

Why did Philip Morris support FDA oversight and RJ Reynolds did not; what was in it for Altria? Dr. Siegel argues that S2461 was essentially a Philip Morris bill: "Regardless of the national health groups' defense of the proposed FDA legislation, the fact of the matter is that the health groups are now supporting a Philip Morris bill. In particular, the Campaign for Tobacco-Free Kids last year vehemently criticized an almost identical bill that it referred to as the "Philip Morris-backed FDA bill," called it "ineffective," called it "weak, loophole-filled legislation."6 All agree that Philip Morris had some hand in writing parts of S2461 and most financial observers are clear what Altria's interests are. Smith Barney tobacco analyst Bonnie Herzog stated that "Although there are several steps that need to be completed before this is signed into law, this is very positive news for the tobacco stocks, particularly Altria Group."14

Ms. Herzog noted that if the FDA did gain power to regulate the tobacco industry, Philip Morris would benefit in the following ways: "barriers [would be created] to entry for some of the deep discount manufacturers, thereby further entrenching the big manufacturers; level the playing field among the major manufacturers; [and] possibly help the industry in fighting future litigation or possibly help prevent future litigation." Furthermore, FDA regulation of tobacco would be particularly positive for Philip Morris USA because "Regulatory guidance could help Philip Morris with the credibility of its reduced risk cigarette product, which if regulated by the FDA could represent an alternative to smoking."14

The Wall Street Journal was of the same opinion. Writing on their editorial page they stated that "[Philip Morris] the tobacco giant knows that FDA regulators would severely restrict marketing, which gives it an advantage over lesser-known rivals and lower-priced competitors. Put another way, the federal government would become not only a partner of tobacco but a partner of tobacco monopolists."¹²

In short, the greater restrictions faced

by new, smaller and upstart cigarette manufactures would have given a competitive edge to the industry leader, Philip Morris; the proposed legislation would cast their current market position in stone. It is no wonder that RJ Reynolds said: "The Kennedy/McConnell

With Philip Morris and the American Public Health Association calling the Senate vote "historic," and the New York Times, some tobacco researchers, and RJ Reynolds denouncing it, the whole tobacco control scorecard has been re-arranged.

amendment is ill-conceived, imperils the viability of a tobacco quota buyout and creates an overwhelming competitive advantage for Philip Morris."¹⁰

FDA Authority: Run Aground on the Rocks of Special Interest

Regardless of the strange bedfellows that came together to support or oppose S2461, House and Senate Conferees put the kibosh on FDA oversight of tobacco products. The conference committee showered corporations with \$145 billion in new tax cuts, including a \$10 billion buyout for tobacco farmers. Clearly, the main focus of these congressmen and women was on business interests not public health; FDA oversight of tobacco products became just one among many bargaining chips at the negotiation table. While Philip Morris may have supported the legislation, Senator John McCain felt the deal cut by the conferees to be "a disgrace," and "a complete sellout to the tobacco companies."15

The dropping of FDA oversight from the FSC/ETI has probably brought to an end the unique coalitions that flourished briefly this past summer in the run up to the congressional votes on FDA authority. While some in the tobacco control movement hailed the defeat of FDA regulation in its current form, still, there is no guarantee that new legislation, stronger or not, can be crafted for the next session. Moreover, it is not clear at

by Kamlesh Asotra, Ph.D.

How does tobacco smoking cause cerebrovascular disease and increase risk for stroke? Are men and women, young and old, of different racial/ethnic populations in equal peril from tobacco? What are the main challenges concerning stroke research? Why is stroke research seriously under-funded and what efforts should be made by federal and voluntary funding agencies to rectify low funding for stroke research in the United States? This article attempts to illuminate these burning issues that currently face research on stroke and its causation by tobacco smoking.

Tobacco and Cerebrovascular Disease

Tobacco is the second major cause of death in the world, and is responsible for 10% of all deaths killing approximately 5 million people each year. Tobacco accounted for nearly 3 million deaths in 1990, increasing its toll to 4 million deaths in 1998, and it is estimated that by 2030, it will cause 10 million deaths annually worldwide.2 It is mind-boggling that this mayhem, unleashed on humanity with the introduction of commercial tobacco to the world by early settlers and traders in North America some 5 centuries ago has become a global public health disaster. Each of these deaths, one every 6.5 seconds, is caused by a variety of tobacco-related diseases predominantly cancer, heart disease, pulmonary disease and cerebrovascular disease culminating in stroke.

Tobacco use accelerates development of both cardiovascular and cerebrovascular diseases by promoting atherosclerosis - a condition of arterial wall hardening due to plaque build-up that causes diminished blood flow - in the arteries that supply blood to the heart muscle or to the brain, respectively. The relationship between smoking and atherosclerosis was observed in the early 1900's by Buerger³ in young male smokers and an association between cerebrovascular disease and extracranial atherosclerosis was reported by Gowers as early as 1875.⁴ Since then, a number of risk factors for cerebrovascular disease culminating in incidents of stroke have been identified (see below).

The precise mechanisms by which tobacco smoking causes cerebrovascular disease culminating in stroke are poorly understood. It is generally believed that tobacco smoke causes oxidation of low density lipoprotein (LDL), and this formation of oxidized LDL is one of the earliest events that initiate the process of plaque formation in systemic and carotid arteries. TRDRP-funded research has demonstrated that tobacco smoke rapidly damages the "good" cholesterol transport particle known as the high density lipoprotein (HDL) by inhibiting its enzyme component, called LCAT (lecithin: cholesterol acyltransferase) which is responsible for packaging cholesterol into HDL particles and which also facilitates the removal of cholesterol from the arteries.^{5, 6} Tobacco smoke results in compromised protective function of HDL, lowered HDL levels, and elevated LDL/HDL ratio in chronic smokers, increasing their risk for atherosclerotic disease in the arteries. Exposure of endothelial cells to cigarette smoke condensate increases the levels of inflammatory cytokines suggesting a complex pro-inflammatory response to tobacco smoke constituents.7

Types and Causes of Strokes

Stroke, the third leading cause of death, is caused by the interruption of blood supply carrying oxygen and nutrients to the brain due to rupture or blockage of an artery to the brain. About 700,000 individuals suffer from stroke annually in the U.S.A., and at any given time there are nearly 3 million stroke survivors in this country. On average, someone in the United States suffers a stroke every 53 seconds, and every 3.3 minutes someone dies of one.8 Two-thirds of stroke survivors suffer moderate or severe impairment. An immediate consequence of stroke is brain injury in the region deprived of blood flow, resulting in loss of consciousness and unilateral paralysis, affecting speech and vision.

The most common variety of stroke

TOBACCO-CAUSED CERE Urgent Need for



"The evidence is sufficient to infer a stroke" - The United States S

called an ischemic stroke is caused by blockage of arterial blood flow due to a blood clot or due to fatty deposits in the blood vessel wall (plaque). This type accounts for over 85% of all cases of stroke. There are two clinical forms of ischemic stroke: (a) embolic stroke, caused by a blood clot formed somewhere in the body (usually the heart) that cannot pass through the narrow bore of an artery, thereby blocking blood circulation to the brain, and (b) thrombotic stroke, the more common variety of ischemic stroke, occurs when blood flow is reduced due to blockage of one or more arteries.9 Large vessel occlusive disease, commonly due to atherosclerotic involvement of the common and internal carotid arteries, can cause carotid ischemic stroke. Small vessel occlusive disease is thought to be non-atherosclerotic narrowing of small end-arteries in

BROVASCULAR DISEASE: Increased Research Funding



Il relationship between smoking and n General's report for 2004.¹

the brain. Cardiogenic stroke occurs due to blood clots arising from the heart, typically as a result of atrial fibrillation or ischemic (i.e., atherosclerotic) heart disease.^{10,11} Strokes caused by bleeding within the brain – called intracerebral (within the brain) and subarachanoid (between the skull and the brain) hemorrhagic strokes - have a higher risk of fatality causing greater functional impairment among survivors than the ischemic strokes. Stroke is not one disease but a heterogeneous group of disorders reflecting differences in pathological mechanisms.12,13 The mortality of ischemic stroke is 15-30% within the first 30 days. Hemorrhagic stroke has a graver prognosis, with a 30-day mortality rate of 40-80%. Treatment and rehabilitation of stroke patients costs nearly \$53.6 billion annually in the United States.14

The thickness of the carotid artery inner wall is significantly greater in smokers than non-smokers, particularly 60-year-olds and above,¹⁵ leading to carotid artery occlusion. Over half of former smokers show greater reduction in internal carotid artery occlusion than current smokers, with the more pronounced difference in older people.¹⁶

A precursor and predictor of stroke, called mini-stroke or transient ischemic attack (TIA), is a neurologic deficit lasting less than 24 hours and is attributed to focal cerebral or retinal ischemia.17 Although many cases of TIA never come to medical attention, it is estimated that more than 5 million Americans are diagnosed with a TIA annually.18 The causes of true TIAs, such as atrial fibrillation, carotid artery disease, and large- smallartery disease in the brain, are identical to those of stroke. Cigarette smoking is reportedly a major preventable risk factor for possibly 200,000 to as many as 500,000 cases of TIA that come to medical attention each year.17

Tobacco Smoking and Stroke

The Framingham Heart Study was the first to assess the relation of smoking to the type of stroke, number of cigarettes smoked and the beneficial effect of smoking cessation.¹⁹ It concluded that smoking was a significant independent factor contributing to the risk of stroke in general, and to brain infarction specifically. The risk of stroke has been directly related to the number of cigarettes smoked. Heavy smokers (>40 cigarettes/day) have a relative risk of stroke 2-4 times greater than non-smokers,^{19, 20} and twice that of people who smoke less than 10 cigarettes/day. The large cohort study of 22,071 US male physicians showed that individuals smoking >20 cigarettes/day had nearly three times the risk of total non-fatal stroke and 50% higher chance of fatal stroke than nonsmokers.²¹ However, other studies show within 5 years after stopping, stroke risk can be reduced to that of non-smokers.¹⁹

Gender and Racial/Ethnic Group Susceptibility to Stroke

The female-to-male mortality ratio differs for stroke subtypes by ethnicity and age in the United States.²²⁻²⁷ Although the fatality rates for hemorrhagic and ischemic stroke are similar for men and women,²⁸ more women than men are admitted to nursing home for stroke disabilities.29 Prospective studies have suggested an increased risk for total hemorrhagic stroke, intracereberal stroke and subarachanoid stroke among men and women who are current smokers; the risk increases with the number of cigarettes smoked.^{30, 31} Generally, women have a lower risk of stroke than men at ages less than 65 years, but women have a higher risk than men at ages of 65 years and above.

The deaths attributed to intracerebral hemorrhagic strokes among Blacks, American Indian/Alaska Natives, Asian/Pacific Islanders, and Hispanic men and women are higher than those among White men and women. The proportion of death due to subarachanoid strokes was greater among American Indian/Alaska Natives, Asian/Pacific Islanders and Hispanic women than Black or White women.25, 26 An analysis of sex-specific age-standardized death rates for ischemic stroke (n=507, 256), intracerebral hemorrhagic stroke (n= 98,709) and subarachanoid hemorrhagic stroke (n=27,334) among Whites, Blacks, American Indian/Alaska Natives, Asians/Pacific Islanders, and Hispanics revealed that overall 81.7% women and 77.7% men succumbed to ischemic stroke, 13.7% women and 18.5% men succumbed to intracerebral hemorrhagic stroke, and 4.6% women and 3.8% men died of subarachanoid hemorrhagic stroke.26

Other Risk Factors for Stroke

Beside cigarette smoking, other risk factors for stroke include, hypertension, valvular heart disease, atrial fibrillation, hyperlipidemia, diabetes mellitus, and a family history of stroke. In addition, elevation of plasma homocysteine, low circulating levels of folic acid and vitamin B6, periodontal disease, and chronic bronchitis are all independent risk factors. Severe, acute ischemia in nerve *See "Funding" page 13*

Enhancing Diversity in Tobacco-Related Disease Research The Cornelius Hopper Award Supplements

by Shana Amenaghwon, M.P.A.

California's diverse populations present both unique challenges and valuable opportunities for tobacco-related disease research. California is literally teeming with special populations. California is rapidly becoming majority Latino, a heterogeneous group of cultures and smoking traditions from throughout South America, Central America, Mexico and the Caribbean. Moreover, there are significant Asian populations including Chinese, Filipinos, Vietnamese, Hmong, Koreans and a growing number of persons from the Indian sub-continent. California is also home to the largest urban American Indian and African immigrant populations in the United States. California's Russian population is also significant, representing the second greatest concentration of this group in the United States. Each of these diverse populations have varied and diverse tobacco use traditions and disease incidences that need to be explored.

Unfortunately, researchers with personal experience in these communities or those who are currently focusing their research in these areas are few and far between. Recognizing the inherent need for scientific talent to address the concerns of under-represented communities, TRDRP created the Cornelius Hopper Diversity Award Supplements (CHDAS) in 2000 to honor the legacy of Dr. Cornelius L. Hopper, former Vice President of Health Affairs, University of California, Office of the President (UCOP).

The Legacy

Dr. Cornelius L. Hopper, the first African-American Vice President of the UCOP, retired after a 20-year tenure spanning from 1979 to 2000. Initially, he was the Special Assistant to the President, and three years later he was named the Vice President of Health Affairs. Dr. Hopper earned his M.D.



Dr. Cornelius L. Hopper Vice President–Health Affairs, Emeritus

degree from the University of Cincinnati College of Medicine. After training in internal medicine, neurology and several years as a faculty member in the Department of Neurology at the University of Wisconsin, he became the Vice President for Health Affairs at Tuskegee Institute in 1971, where he created the first National Health Service Corps Field Station in the southeastern United States and the first rural community based area health education center.

During his tenure as the Vice President for Health Affairs at UCOP, Dr. Hopper was responsible for a system of 14 health professions schools, five major teaching hospitals and a budget of more than \$2.5 billion. He was instrumental in establishing statewide research programs in AIDS, geriatrics, breast cancer and tobacco-related diseases that awarded more than \$300 million to California researchers. He also developed the Wellness Lecture Series and coordinated the expansion of primary care training opportunities in the UC system. He has served on a variety of boards including the Board of Regents at

Oakland's Samuel Merritt College, and he is a California Health Manpower Policy Commissioner. Most recently he has evaluated a number of post-Soviet hospital partnerships established by the American International Health Alliance with USAID funding.

The Award

The overall aim of the CHDAS award is to enhance the trainees' experience and qualification for tobacco research careers and to expand and strengthen the infrastructure for tobacco research in California by assisting the development of research, including historically underrepresented communities. Currently active TRDRP funded principal investigators are encouraged to mentor qualified trainees with this \$15,000 supplement. To date, TRDRP has funded 30 CHDAS awards to 25 principal investigators at 14 institutions totaling \$764,272. (See www.trdrp.org "CHDAS Awardees" for the complete list.)

The CHDAS Experience

Recipients receive invaluable experience and exposure to research of tobaccorelated diseases, and many have used the award as a means to further their career and educational goals. Not only do the CHDAS recipients benefit from the award, the principal investigators also benefit by the opportunity to augment their staff support assisting the progress of their research. Here are just a few of their stories:

Cara Booker, one of the program's first year recipients in 2000, worked with Dr. Jennifer Unger on her TRDRP supported project entitled "Accultutraion, media, peers, parents and adolescent smoking" at University of Southern California (USC). She applied for the award as a public health master's candidate and indeed earned her M.P.H. with an emphasis on biostatistics and epidemiology in 2003. She is now in a

CHDAS Continued from page 10

Ph.D. program at USC where she continues her research and expects to graduate in the spring of 2005. Her dissertation will be a cross national/cross-cultural study focusing on stressful life events and adolescent smoking. She is awaiting the peer-reviewed publication of her manuscript where she is the first author and has several more possible publications in progress. She explains, "I feel that the CHDAS was really instrumental in allowing me to discover ... my passion in the public health and research field."

Dr. Unger explains, "One of the best features of the CHDAS program is that it helps graduate students carve out their own research niches. Graduate students typically work as research assistants on our TRDRP-funded projects, but usually it's not until the dissertation stage that they have the opportunity to propose and test their own hypotheses. The CHDAS program gives them the opportunity to pursue their own ideas, while they still have support from the PI and resources of the parent grant...Cara's dissertation is a direct extension of our TRDRP research...The CHDAS award gave her an excellent introduction to tobacco research. I'm confident that she will become a leader in the field."

Darya Soto, M.D. was a 2001 CH-DAS recipient training under the direction of Dr. George Caughey on his TRDRP supported project entitled, "Human tryptase gene expression: Role in COPD" at the University of California, San Francisco. Dr. Soto applied for the 2001 CHDAS as a practicing physician seeking to expand her career in lung cancer research. She is currently an assistant adjunct professor of medicine at UCSF department of medicine researching lung cancer using mouse models of adenocarcinoma. She has received other grants since her CHDAS. Most recently, she received a Faculty Development Award from the National Cancer Institute. Dr. Soto states, "The award and mentoring by Dr. Caughey did assist in my career development. They aided in initiating my current research studies."

Dr. Caughey adds, "The CHDAS

program provides a terrific opportunity for established investigators in tobaccorelated disease to incubate the interest and careers of individuals not adequately represented in this field. CHDAS support was just what Dr. Soto needed to jump-start her career in lung cancer research and to obtain the results needed to win larger-scale, longer-term support from the National Cancer Institute."

Andrea Castillas, a 2002 CHDAS recipient worked under the mentorship of Dr. Randolph Hastings on his TRDRP support project entitled, "Novel regulatory mechanisms for lung cancer growth" at the Veterans Medical Research Founand lung cancer. As a result, this award has given me the opportunity to meet and collaborate with leading researchers in the area of lung cancer. In the laboratory, it has assisted me in learning new techniques such as immunohistochemistry, quantitative real time PCR and ...[data] collection methods in clinical research."

Dr. Hastings further explains, "The obvious benefit of the award for me as a principal investigator was to provide support for a qualified individual to work in my laboratory. Andrea began in the lab with a project for her post-baccalaureate program. The supplement

LOOKING FORWARD

TRDRP is proud of this award and is especially proud of and salutes all of the Cornelius Hopper awardees and their mentors. We urge TRDRP principal investigators to mentor all individuals who are interested in pursuing tobacco-related disease research and to encourage qualified individuals to apply for Cornelius Hopper Diversity Award Supplements. Applications for these awards will be invited in the 3rd week of April 2005. Please visit www.trdrp.org for CHDAS application requirements.

dation of San Diego. Andrea applied as a UCSD Human Development B.A. graduate with a minor in biology interested in the biological sciences and exploring M.D. and Ph.D. programs.

She continues to work with Dr. Hastings and is also a hospital assistant at the UCSD student-run free clinic teaching medical student's clinical laboratory, where part of her work is on tobacco-related diseases such as lung cancer. She has received an NIH Minority International Research Training Program Scholarship, which has given her additional experience in biomedical research. She is currently applying to medical schools with research opportunities. Andrea states, "I am very thankful that Dr.Hastings gave me the opportunity to apply for this award... working with Dr. Hastings inspired me to follow a career in research...As a recipient of the award, Dr. Hastings has sent me to [attend] symposiums, meetings, and conferences to learn more about research

allowed her to continue working, to learn more, and to develop additional techniques in that project for a longer period of time. In addition, she could spend more hours in that lab because of the financial support. Because she wrote the application for CHDAS (with my help), she had a larger stake and sense of ownership of her project than she might have felt if I had simply hired her as an employee. Finally, the opportunity to go to the TRDRP meetings and present her findings was a valuable experience. The process of applying for the award was straightforward, and I would encourage other investigators to apply if they have appropriate candidates."

I would like to extend a special thanks to Dr. Hopper, Dr. Asotra, Dr. Bowen and Dr. Gardiner for their support and expertise without which this article would not have been written.

TRDRP UPDATE

49 Grants Awarded in the 2004 Funding Cycle

TRDRP awarded 49 grants to individual investigators at 27 California institutions in the 2004 funding cycle. The proportion of applications funded improved over last year from 23.8% to 26.3% even though there were fewer available funds this year (\$17 million vs.\$18 million) and fewer applications (186 vs. 244). This improvement is due to the fact that almost all applicants were responsive to TRDRP's primary research areas introduced in this cycle and that almost one out of three of the 189 applications reviewed were ranked as either excellent or outstanding. Unfortunately, several proposals that were scored as "excellent" by TRDRP's study sections could not be funded due to insufficient funds. Another \$5 million would have been needed to fund all of these excellent proposals. Funding levels varied due to the different number of applications received for various award mechanisms. The number and percent of applications funded by award mechanism are listed below.

	Applications Reviewed	Number of Funded	Percent Funded
Award Mechanism			
Research Project – Primary Area	91	24	26%
Research Project – Complementary Area	7	1	14%
Innovative Developmental Exploratory (IDEA)	26	5	19%
New Investigator	19	6	31%
Postdoctoral Fellowship	21	7	33%
Dissertation	9	4	44%
Community-Academic Research	9	1	11%
School-Academic Research	4	1	25%

A complete list of grant recipients and the abstracts describing their research projects is published in the 2004 Compendium of Awards available on the TRDRP website (www.trdrp.org). All funded investigators are mailed a copy; other interested parties may obtain printed copies upon request.

Cornelius Hopper Diversity Award Supplements

This year marked the fifth year of funding for the Cornelius Hopper Diversity Award Supplements (CHDAS). In 2004, six currently-funded TRDRP investigators will receive CHDAS supplements to their grants to mentor trainees (see box). For further information on CHDAS see related article on page 10.

CHDAS Trainee	Principal Investigator	Institution
Marc Adams, M.P.H.	Dr. Melbourne Hovell	San Diego State University
Justin Hernandez, B.S.	Dr. Richard Olmstead	Brentwood Biomedical Research Institute
Pamela Jones, M.P.H., B.S.N.	Dr. Ruth Malone	University of California San Francisco
Yaneth Rodriguez, B.S.	Dr. Steve Sussman	University of Southern California
Claradina Toya, B.A.	Dr. Jennifer Unger	University of Southern California
Jessica Zulema-Borja, B.A.	Dr. Ricardo Munoz	University of California San Francisco

Funding Continued from page 9

tissue triggers cellular changes that can rapidly cause irreversible damage (infarction). A penumbra of ischemic, electrically silent tissue develops around the infarct zone that may be salvageable by restoration of blood flow.

There has been a lot of interest in learning if stroke has some genetic determinants. Although genetic determinants of the common forms of stroke are largely unknown, some mutations in specific genes causing rare forms of stroke have been reported,³² including brain hemorrhage.³³ The first main locus associated with stroke, called STRK1, was mapped to chromosome 5q12.³⁴ Recently, two genes have been identified in the pathogenesis of stroke: A gene called PDE4D, encoding the enzyme phosphodiesterase 4D, is reported to be important in ischemic stroke,35 and another gene encoding 5-lipoxygenase activating protein confers risk of myocardial infarction and stroke.36

Why Progress in Stroke Research Has Been Slow?

There are several reasons why stroke research throughout the world has been lagging behind that for other major diseases such as cancer and cardiovascular disease. Stroke has usually been perceived as a disease of the elderly that is largely untreatable and difficult to study. As previously described, stroke is a very heterogeneous disorder comprising a number of different syndromes with different etiologies. Also, stroke has a large number of risk factors that are commonly shared by cardiovascular disease. A possible shortage of experts in stroke research in the United States could also be a reason for a slow pace of progress.

An internet search revealed that Cerebrovascular Disease and Stroke Centers exist at several major public and private universities and medical centers in California. Many of these centers, including those at UCLA, UCSF, Stanford and USC, among others, have active research programs in different aspects of stroke research. Collaborative efforts among stroke researchers at these Centers and the well known cadres of scientists engaged in research on tobacco-related diseases within California could unravel novel molecular mechanisms by which tobacco causes cerebrovascular disease as well as develop facile diagnostic and effective therapeutic/interventional technologies for stroke in the near future.

The Report of the Stroke Progress Group, the National Institutes of Neurological Disorders and Stroke,³⁷ published in 2002 not only identified various reasons that make stroke research the challenge it has been thus far, but also recommended the research and scientific made available for stroke research in 2003 by the NIH, with projections of \$342 million and \$352 million in years 2004 and 2005, respectively.41 Total NIH funding for tobacco research in 2003 was \$531 million,⁴¹ although it is not clear what proportion of this sum was spent on tobacco-related disease research, tobacco use cessation or disease prevention. The American Heart Association has been funding stroke research since the 1950's. The American Heart Association and the American Stroke Association awarded \$39.2 million on stroke research nationally and \$4.07 million in California in 2003.42

"Four million unnecessary deaths per year, 11,000 every day. It is rare - if not impossible - to find examples in history that match tobacco's programmed trail of death and destruction. I use the word 'programmed' carefully. A cigarette is the only consumer product which when used as directed kills its customer" Dr. Gro Harlem Brundtland, Director-General Emeritus, World Health Organization.²

priorities for stroke research for the next 5-10 years. Unfortunately, this Report is silent on the need to support research on the causation of stroke by tobacco.

Funding for Stroke Research

Despite the high human and financial costs of stroke globally, stroke research has been significantly under-funded as compared with heart disease and cancer in the United States,³⁸ the United Kingdom,³⁹ and across Europe.⁴⁰

The cost of cancer in the US was estimated at more than \$150 billion in 2002. The NIH actually invested \$5.4 billion in cancer research in fiscal year 2003, with estimated expenditure of \$5.6 billion and more than \$5.7 billion to be made in 2004 and 2005, respectivelv.⁴¹ In contrast, the cost of heart disease and stroke in 2003 was estimated to be \$351 billion: \$209 billion for health care expenditures and \$142 billion for lost productivity from death and disability. Of this total, the cost of stroke was estimated to be approximately \$53.6 billion.¹⁴ While almost \$2.3 billion were spent by the NIH on cardiovascular disease in 2003, only \$330 million were

Considering that stroke is the 3rd major cause of death in the USA with the paltry current funding, there is an obvious need to boost federal funding for stroke research. Currently, only the National Heart, Lung, and Blood Institute and the National Institute of Neurological Disorders and Stroke offer funding opportunities for stroke research. It will be prudent to provide sufficient federal funding to 'jump start' stroke research, preferably at the level of 3% of the cost of disease burden, comparable to that for cancer, in light of the fact that with the expected increase in the aging population, the incidence of stroke is likely to rise proportionately. This objective can be easily met if more Institutes within the NIH that traditionally fund research in the area of tobacco, aging, drug addiction, diabetes and genetics, also entertain and fund grant applications on tobacco-caused stroke in increasing numbers.

TRDRP's Commitment for Stroke Research in California

Cerebrovascular disease including stroke, has been a legislatively mandated, prior-

See "Funding" page 14

Funding Continued from page 13

ity area of research for TRDRP since its inception in 1989. However, over the past several years, TRDRP has received only a small number of grant applications on cerebrovascular disease as compared with those on cardiovascular disease or cancer. One reason for this may be due to possible shortage of research groups in California engaged in stroke research until recently. This, in turn, may be dictated by the availability of limited

The National Stroke Association has recommended adoption of the term brain attack for stroke, by analogy with the familiar heart attack. This description is intended to emphasize to the public both the location of the lesion and the urgency of the need for assessment and treatment.

federal research funds for stroke research. As mentioned previously, there exists a unique cadre of scientists in California that constitute perhaps the best tobacco-related disease research enterprise in the United States. Also, there exist several Cerebrovascular Disease and Stroke Centers in California now. Together, this research infrastructure and the scientists in California are fully capable of taking the lead role in answering outstanding questions regarding tobacco and stroke, as they have done in other tobacco-related diseases. TRDRP continues its commitment in providing funds for tobacco-caused cerebrovascular disease research which may lead to improved diagnostic and therapeutic approaches for stroke among Californians. It is our hope that the new research findings from TRDRP's support of stroke research will enable California scientists to generate additional funding from federal and non-profit funding agencies for the creation and viability of a much-needed stroke research enterprise in California. TRDRP encourages applications focused on

cerebrovascular disease from all researchers in California for various award mechanisms.

Acknowledgments: The author is grateful to Mr. Michael Edell, Director of Research, American Heart Association Western Affiliate, for providing 2003-04 data on research funding for stroke both across the USA and in California. Sincere thanks are also due to my colleagues Drs. M.F. Bowen, F. Buchting, C. DiSogra and P. Gardiner for critical reading of the manuscript and their editorial suggestions.

References

- U.S. Department of Health and Human Services. The Health Consequences of Smoking: A Report of the Surgeon General (2004). (http://www.cdc.gov/tobacco/).
- Excerpt from Dr. Gro Harlem Brundtland's address to the WHO International Conference on Tobacco and Health, Kobe City, Japan, 15th November, 1999. http://www.who.int/directorgeneral/speeches/1999/english/19991115_kobe html.
- Buerger, L. (1908). Thromboangiitis obliterans: a study of the vascular lesions leading to presenile spontaneous gangrene. Am. J. Med. Sci., 208:567.
- Gowers, W.R. (1875). On a case of simultaneous embolism of central retinal and middle cerebral arteries. Lancet, ii:794.
- Bielicki, J.K., Forte, T.M., and McCall, M.R. (1995). Gas-phase cigarette smoke inhibits plasma lecithin-cholesterol acyltransferase activity by modification of the enzyme's free thiols. Biochim.Biophys.Acta., 24;1258(1): 35-40.
- Bielicki, J.K. (2003). Diagnostic tools of HDL damage by tobacco smoke. Final Progress Report of the TRDRP grant award.
- Nordskog, B.K., Blixt, A.D., Morgan, W.T., Fields, W.R., and Hellmann, G.M. (2003). Matrix-degrading and pro-inflammatory changes in human vascular endothelial cells exposed to cigarette smoke condensate. Cardiovasc. Toxicol., 3(2):101-117.
- American Stroke Association. www.strokeassociation.org.
- Caplan, L.R. (2000). Caplan's Stroke: A Clinical Approach. Butterworth-Heinemann, Boston.
- Adams, H.P., Jr. et al. (1993). Classification of subtypes of acute ischemic stroke. Definitions for use in a multicenter clinical trial. TOAST. Trial of Org 10172 in Acute Stroke Treatment. Stroke, 24: 35-41.
- Fisher, C.M. (1982). Lacunar strokes and infarcts: A review. Neurology, 32: 871-876.
- Alberts, M.J. (1999). Genetics of Cerebrovascular Disease. Futura, New York.
- Hassan, A., and Markus, M.J. (1995). Genetics and ischemic stroke. Brain, 123: 1784-1812.
- Heart Disease and Stroke Statistics 2004 Update. American Heart Association, pp.13-16 (www.americanheart.org).

- Howard, G., Burke, G.L., Szklo, M., Tell, G.S., Eckfeldt, J., Evans, G. et al. (1994). Active and passive smoking are associated with increased carotid wall thickness. The atherosclerosis risk in community study. Arch.Inter. Med., 154: 1277-1282.
- Tell, G.S., Polak, J.F., Ward, B.J., Kittner, S.J., Savage, P.J., and Robbins, J. (1994). Relation of smoking with carotid artery wall thickness and stenosis in older adults. The cardiovascular health study. Circulation, 90: 2905-2909.
- Johnston, S.C. (2002). Transient ischemic attack. N. Engl. J. Med., 347 (21): 1687-692.
- TIA/mini strokes: public knowledge and experience – Roper Starch Worldwide survey: Roper Starch Worldwide. Englewood, Colorado: National Stroke Association (2000). 55.
- Wolf, P.A., D'Agostino, R.B., Kannel, W.B., Bonita, R., and Belanger, A.J. (1988). Cigarette smoking as a risk factor for stroke. The Framingham Study. JAMA, 259: 1025-1029.
- Wannmathee, S.G., Shaper, A.G., Whicup, P.H., and Walker, M. (1995). Smoking cessation and the risk of stroke in middle-aged men. JAMA, 274: 155-160.
- Robbins, A.S., Mansfield, J.E., Lee, I., Satterfield, S., and Hennekens, C.H. (1994). Cigarette smoking and stroke in a cohort of US male physicians. Ann.Intern.Med., 1210: 458-462.
- 22. Gillum, R.F. (1995). Epidemiology of stroke in Native Americans. **Stroke**, 26: 514-521.
- Gillum, R.F. (1995). Epidemiology of stroke in Hispanic Americans. Stroke, 26: 1707-1712.
- Gillum, R.F. (1999). Stroke mortality in blacks: disturbing trends. Stroke, 30: 1711-1715.
- Ayala, C., Greenlund, K.J., Croft, J.B., Keenan, N.L., Donehoo, R.S., Giles, W.H., Kittner, S.J. and Marks, J.S. (2001). Racial/ethnic disparities in mortality by stroke subtype in the United States, 1995-1998. Am. J. Epidemiol., 154: 1057-1063.
- Ayala, C., Croft, J.B., Greenlund, K.J., Keenan, N.L., Donehoo, R.S., Malacher, A.M., and Mensah, G.A. (2002). Sex differences in US mortality rates for stroke and stroke subtypes by race/ethnicity and age, 1995-1998. Stroke, 33: 1197-1201.
- Frey, J.L., Jahnke, H.K., and Bulfinch, E.W. (1998). Differences in stroke between White, Hispanic and Native American patients. The Barrow Neurological Institute Stroke Database. Stroke, 29: 29-33.
- The CROSS Group. Epidemiology of aneurismal subarachnoid hemorrhage in Australia and New Zealand: incidence and case fatality from the Australian Cooperative Research on Subarachnoid Study (ACROSS). (2000).
 Stroke, 31: 1843-1850.
- Leibsom, C.L., Ranson, J.E., Brown, R.D., O'Fallon, W.M., Hass, S.L., and Whisnant, J.P. (1998). Stroke-attributable nursing home use. Neurology, 51: 163-168.
- Kurth, T., Kase, C.S, Berger, K., Schaeffner, E.S., Buring, J.E., and Gaziano, M. (2003). Smoking and the risk of hemorrhagic stroke in men. Stroke, 34: 1151-1155.

Bedfellows

Continued from page 7

all when or if FDA oversight authority over tobacco products will make its way back to the top of the congressional agenda. On the other hand, since the tobacco farmers buyout took place without FDA oversight, the pressure is already mounting on future tobacco control proponents and future legislators to craft legislation that rightfully gives the FDA jurisdiction over tobacco.

References

- Copy of the Senate bill can be found at: http://ash.org/fda
- Dewar, H. "Senate Backs Compromise on Tobacco; Regulation of Industry Paired with Buyout." Washington Post, Friday, July 16, 2004. http://www.washington post.com/ac2/wp-dyn/A52184-2004Jul15?language=printer
- Benjamin, G. "American Public Health Association Applauds Passage of Historic Tobacco Control Amendment." American Public Health Association (APHA). Press Release, Friday, July 16, 2004, http://www.apha.org/news/press/2004/ tobacco_control_amendment.htm
- 4. Campaign for Tobacco Free Kids, American Cancer Society, American Heart Association, American Lung Association. "FDA Regulation of Tobacco Products: Why It's Needed and What It Will Do." Joint Press Release, June 1, 2004, http://www.tobaccofreekids.org/reports/ fda/summary.shtml
- Gonzales, Lisa. "Altria Group and Philip Morris USA Applaud Senate Passage of Bill Granting the FDA Regulatory Authority of Tobacco Products." Altria Corporate Services, Inc. Press Release, Thursday, July 15, 2004 http://biz. yahoo.com/bw/040715/155954_1. html
- Siegel, M. "Let's be clear: S2461 is a Philip Morris bill.: Email posting, Tuesday, June 1, 2004, http://www. smokefree.net/bgannounce/messages/
- Siegel, M. "Critical Analysis of S2461: FDA Tobacco Legislation Senate Amendment 3563 (Kennedy-DeWine Amendment) to S1637, the Foreign Sales Corporation/Extraterritorial Income Act (FSC/ETI); Part I, Flaws in Overall Regulatory Context of S2461." Email posting, Saturday, July 17, 2004, http://www.smokefree.net/bgannounce/ messages/

- Siegel, M. "Critical Analysis of S2461: FDA Tobacco Legislation Senate Amendment 3563 (Kennedy-DeWine Amendment) to S1637, Foreign Sales Corporation/Extraterritorial Income Act (FSC/ETI); Part II, Why S2461 Will Be Detrimental to the Public's Health." Email posting, Saturday, July 17, 2004, http://www.smokefree.net/bgannounce/messages/
- Cummings, M. "Analysis of the FDA tobacco Legislation." Email posting, Email posting, Saturday, July 17, 2004, bill@smokescreen.org.
- Singleton, J. "Senate Tobacco Quota Buyout/FDA Legislation Would Hurt Growers, Industry." R.J. Reynolds Tobacco Company Press Release, Thursday, July 15, 2004, http://www.rjrt.com/NR/ NRreleases_rjrtview.asp?postID=947
- NY Times Editorial. "The Stink y Tobacco Deal." New York Times Corporation, Friday, July 16, 2004, http://www.truthout. org/docs_04/071704E.shtml

- Wall Street Journal Editorial. "Congress's Marlboro Men." Wall Street Journal Corporation, Tuesday, July 20, 2004, http://no-smoking.org/july04/07-21-04-5.html
- Godshall, B. "Campaign Against FDA Tobacco Legislation." Smokefree Pennsylvania, Email posting, Wednesday, July 21, 2004, http://www.smokefree.net/ bg-announce/messages/247115.html
- 14. Herzog, B. "Senate Passes Tobacco Buyout and FDA Regulation Legislation Amendment." Smith Barney Tobacco Analyst, Monday, July 19, 2004, http://www.smokefree.net/bgannounce/ messages/247131.html
- Andrews, EL. "Negotiators Approve Big Tax Cuts for Business." New York Times, October 7, 2004.

Funding Continued from page 14

- Kurth, T., Kase, C.S, Berger, K., Gaziano, M., Cook, N.S., and Buring, J.E. (2003). Smoking and risk of hemorrhagic stroke in women. Stroke, 34: 2792-2795.
- Joutel, A. et al. (1996). Notch3 mutations in CADASIL, a hereditary adult-onset causing stroke and dementia. Nature, 383: 707-710.
- Palsdottir, A. et al. (1988). Mutation in cystatin C gene causes hereditary brain hemorrhage. Lancet, 2: 603-604.
- Gretarsdottir, S., et al. (2002). Localization of a susceptibility gene for common forms of stroke to 5q12. Am. J. Hum.Genet., 70:593-603.
- Gretarsdottir, S., et al. (2003). The gene encoding phosphodiesterase 4D confers risk of ischemic stroke. Nat Genet., 35:131-138.
- Helgadottir, A., et al. (2004). The gene encoding 5-lipoxygenase activating protein confers risk of myocardial infarction and stroke. Nat Genet., 36:233-239.
- The Report of the Stroke Progress Group, National Institutes of Neurological Disorders and Stroke. (2002). (www.ninds.nih.gov).

- Toole, J.F. and Toole, W.W. (1984). Federal funding for research in stroke and trauma a clinical investigator's viewpoint. Stroke, 15:168-171.
- Rothwell, P.M. (2001). The high cost of not funding stroke research: a comparison with heart disease and cancer. Lancet, 357: 1612-1616.
- Pendlebury, S.T., Rothwell, P.M., Algra, A., Ariesen, M-J., Bakac, G., et al. (2004). Underfunding of Stroke Research: A Europe-Wide Problem. Stroke, 35: DOI 10.1161/01. STR.0000140632.83868.a2.
- U.S. Department of Health and Human Services. National Institutes of Health. Estimates of Funding for Various Diseases, Conditions, Research Areas. (Update of August 17, 2004). (www.nih.gov/news/fundingresearchareas/htm).
- Personal e-mail communication from Mr. Michael Edell, Director of Research, American Heart Association, Western Affiliates, dated August 30, 2004.



Tobacco-Related Disease Research Program University of California—Office of the President 300 Lakeside Drive, 6th Floor Oakland, CA 94612-3550 1952

December 2004 Newsletter

Burning Issues

is published by TRDRP Office of Health Affairs University of California Office of the President 300 Lakeside, 6th Floor Oakland, CA 94612-3550 Phone: (510) 987-9870 Fax: (510) 835-4740 e-mail: trdrp@ucop.edu

www.trdrp.org

TRDRP Staff

Director Charles A. DiSogra, Dr.P.H., M.P.H.

Research Administrators Kamlesh Asotra, Ph.D. M.F. Bowen, Ph.D. Francisco O. Buchting, Ph.D.

Phillip Gardiner, Dr.P.H.

Administrative Coordinator Teresa E. Johnson

Grant Analysts

Shana Amenaghawon, M.P.A. Christine Tasto, M.P.A.

Administrative Staff

Leslie Bertrand Jewel Charles Carlin Colbert Sharon L. Davis

HOLD THE DATES

December 4-8, 2004 THE AMERICAN SOCIETY FOR CELL BIOLOGY 44TH ANNUAL MEETING Washington, DC

> January 20, 2005 TRDRP APPLICATION DEADLINE Oakland, CA

February 16-19, 2005

SECOND INTERNATIONAL CONFERENCE ON WOMEN, HEART DISEASE AND STROKE Orlando, FL

> October 12-13, 2005 2005 TRDRP Investigators Meeting Los Angeles, CA