### Tobacco PIC

#### Counterplan: Public colleges and universities in the United States ought not restrict freedom of speech, except for prohibiting the tobacco industry from sponsoring social events held by any organization that receives university funding.

Rigotti et al 05 Nancy A. Rigotti, MD, Susan E. Moran, MD, MSCE, and Henry Wechsler, PhD “US College Students’ Exposure to Tobacco Promotions: Prevalence and Association With Tobacco Use” American Journal of Public Health 2005 January; 95(1): 138–144 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1449866/

Our findings have implications for universities, states, and communities. Colleges and universities should be alert to tobacco industry sponsorship of events on their campuses. As the American College Health Association and American Cancer Society recommend, colleges should ban the free distribution of tobacco products on campus, including distribution to fraternities and sororities, and prohibit tobacco industry sponsorship of social events held by any organization that receives college funds.10,24 States and communities already have a good reason for adopting smoking bans in bars and nightclubs: eliminating exposure to secondhand smoke.25,26 Our findings provide an additional rationale for adopting these policies: tobacco promotions are likely to be less successful in a smoke-free bar or nightclub, because smoking would not be modeled as an integral part of this social activity. Decoupling smoking and drinking will likely be an effective way to counteract the tobacco industry’s marketing strategies.

#### It competes—advertisement of commercial products is protected by the constitution.

US Courts “What Does Free Speech Mean?” <http://www.uscourts.gov/about-federal-courts/educational-resources/about-educational-outreach/activity-resources/what-does> JW

The First Amendment states, in relevant part, that: “Congress shall make no law...abridging freedom of speech.” Freedom of speech includes the right: Not to speak (specifically, the right not to salute the flag). West Virginia Board of Education v. Barnette, 319 U.S. 624 (1943). Of students to wear black armbands to school to protest a war (“Students do not shed their constitutional rights at the schoolhouse gate.”). Tinker v. Des Moines, 393 U.S. 503 (1969). To use certain offensive words and phrases to convey political messages. Cohen v. California, 403 U.S. 15 (1971). To contribute money (under certain circumstances) to political campaigns. Buckley v. Valeo, 424 U.S. 1 (1976). To advertise commercial products and professional services (with some restrictions). Virginia Board of Pharmacy v. Virginia Consumer Council, 425 U.S. 748 (1976); Bates v. State Bar of Arizona, 433 U.S. 350 (1977). To engage in symbolic speech, (e.g., burning the flag in protest). Texas v. Johnson, 491 U.S. 397 (1989); United States v. Eichman, 496 U.S. 310 (1990).

#### Tobacco companies use social events at universities to promote smoking—causes more regular tobacco use.

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Tobacco use among young adults in the United States is a growing public health concern. Cigarette smoking rates declined between 1993 and 2000 among all US adults except those aged 18 to 24 years.1 Among US college students, the prevalence of smoking rose dramatically during the 1990s before it declined slightly between 1999 and 2001.2–4 Smoking rates among young adults who do not attend college are higher than smoking rates among college students.4 Several factors account for young adults’ increased tobacco use. One factor is the aging of the cohort of adolescents whose smoking rates increased after 1991, but it does not explain all of the change.2–5 Another factor may be that young adults are initiating regular tobacco use in larger numbers.5 Young adults (aged 18–24 years) are the youngest legal targets of tobacco industry marketing. Internal tobacco industry documents show that tobacco marketing targets young adults.6–9 The industry envisions the uptake of smoking as a process that extends into young adulthood, during which time adolescents’ experimental or occasional smoking becomes solidified into a regular smoking habit.9 The tobacco industry has developed novel marketing strategies to promote this transition. A well-documented strategy is to sponsor social events at bars and nightclubs where free cigarettes and promotional items are distributed.6,7,9 Similar promotions take place at college social events sponsored by organizations such as fraternities and sororities.10 Bars and nightclubs have assumed greater importance for tobacco marketing since the 1998 Master Settlement Agreement between the tobacco industry and 46 states’ attorneys general, because the agreement limits the distribution of free cigarette samples to facilities that do not admit minors.6,9,11 Bars and nightclubs also are smoker-friendly environments for the tobacco industry, because they are among the few places where smoking is not generally restricted by clean-air laws.7 Promotional events at bars, nightclubs, and college social events aim to link alcohol with tobacco use and to make tobacco products a visible part of young adults’ social lives.6,7 The events reinforce brand visibility, allow the industry to reach specific target groups, and generate names for future marketing efforts.6,7,9 Promotions at social events have the potential to increase tobacco use by encouraging nonsmokers to try cigarettes, by encouraging experimental smokers to develop regular use, and by discouraging current smokers from quitting. There is no information about the extent of young adults’ exposure to these new tobacco promotions or about the impact of these promotions on young adults’ tobacco use. The potential impact could be substantial, because young adults are more susceptible to tobacco marketing than adults in older age groups.12 Colleges and universities provide a key channel for reaching young adults, because approximately one third of young adults attend college.13 Our study used data from a large nationally representative random sample of US college and university students to assess the prevalence of students’ exposure to tobacco promotions at bars, nightclubs, and campus social events and to explore the association between that exposure and smoking behavior. We hypothesized that students’ tobacco use before entering college might modify this association, because students who did not smoke regularly before college would be more susceptible to bar/nightclub promotions than students who entered college as regular smokers.

#### Empirics prove—college tobacco marketing increases the chance of tobacco use.

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To our knowledge, this is the first study that measured young adults’ exposure to a tobacco industry marketing strategy that has assumed greater prominence since the 1998 Master Settlement Agreement. During the first 6 months of the 2000–2001 school year, 8.5% of US college students attended a tobacco industry–sponsored social event where free cigarettes were distributed. Students at all but one of the 119 colleges surveyed reported attending these events. Bars and nightclubs were the most common settings, but students also reported attending events on college campuses, a site that has received less attention and that provides direct access to students. Our study shows that there is an association between attendance at these promotional events and tobacco use. It has been hypothesized that the tobacco industry’s new promotional strategies have contributed to the observed increase in young-adult tobacco use. To date, however, the evidence is only indirect; the introduction of these strategies corresponds temporally with the increase in smoking among young adults.5–7,9 We add to the evidence by showing an association between exposure to the new tobacco promotional events and current smoking. The association remained strong after we adjusted for potential confounding factors, such as the fact that smokers drink more alcohol and are more likely to go to bars. Furthermore, the effect of tobacco promotions on smoking behavior was modified by a student’s history of tobacco use before entering college. Nearly 80% of the students had not smoked regularly before 19 years of age. Among this group, students exposed to a tobacco promotional event had higher odds of being a current smoker at the time of our study. In contrast, students who were already smoking regularly when they entered college continued to smoke at high rates, and attending a tobacco promotional event had no effect on their smoking prevalence. This finding suggests that the tobacco industry sponsorship of social events may be encouraging the initiation or the progression of smoking among young adults.

#### Tobacco use causes massive negative health effects.

Saha et al 07 Sibu P Saha, MD MBA FICA, Deepak K Bhalla, PhD, Thomas F Whayne, Jr, MD PhD FICA, and CG Gairola, PhD “Cigarette smoke and adverse health effects: An overview of research trends and future needs” Int J Angiol. 2007 Autumn; 16(3): 77–83 <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2733016/> JW

As part of the Global Burden of Disease Study carried out by the Harvard University School of Public Health in 1997 (4), it was projected that mortality and morbidity from tobacco use will increase by almost threefold worldwide in 20 to 25 years. Similar predictions have been made by the Oxford University Center headed by Sir Richard Doll, who was one of the first researchers to link cigarette smoking with lung cancer in the 1950s (5,6). Cancer, cardiovascular diseases and chronic obstructive pulmonary disease continue to be the main health problems associated with cigarette smoking. An extensive database has accumulated, which has consistently documented a relationship between smoking and these specific diseases. The strength of the association is further demonstrated by measuring the RR and the presence of a dose-response relationship (ie, direct relationship between the intensity of exposure to cigarette smoke and the risk of disease). According to a 2004 Centers for Disease Control and Prevention report (3), approximately 2600 people die of cardiovascular disease in the United States every day, which translates into one death every 33 s. Furthermore, the likelihood of dying from heart disease increases fourfold as a result of smoking. The cost of heart disease and stroke in terms of health care expenses and lost productivity was estimated at US$351 billion in the United States alone in 2003. An analysis by European health experts (7) determined that in developed countries as a whole, tobacco is responsible for 24% of all male deaths and 7% of all female deaths; these figures rise to over 40% in men in some countries of central and eastern Europe and to 17% in women in the United States. The average decreased life span of smokers is approximately eight years. Among United Kingdom doctors followed for 40 years, overall death rates in middle age were approximately three times higher among physicians who smoked cigarettes than in nonsmokers. In those United Kingdom physicians who stopped smoking, even in middle age, a substantial improvement in life expectancy was noticed. These same experts found that worldwide, smoking kills three million people each year and this figure is increasing. They predict that in most countries, the worst is yet to come, because by the time the young smokers of today reach middle or old age, there will be approximately 10 million deaths per year from tobacco use. Approximately 500 million individuals alive today can expect to be killed by tobacco and 250 million of these deaths will occur in the middle age group. Tobacco is already the biggest cause of adult death in developed countries. Over the next few decades tobacco is expected to become the biggest cause of adult death in the world. For men in developed countries, the full effects of smoking can already be seen. Tobacco causes one-third of all male deaths in the middle age group (plus one-fifth in the old age group) and is the cause of approximately one-half of all male cancer deaths in the middle age group (plus one-third in the old age group). Of those who start smoking in their teenage years and continue smoking, approximately one-half will be killed by tobacco. One-half of these deaths will be in middle-aged individuals (35 to 69 years of age) and each will lose an average of 20 to 25 years of nonsmoker life expectancy. In contrast, the total mortality is decreasing rapidly and cancer mortality is decreasing slowly in nonsmokers in many countries. Throughout Europe in the 1990s, tobacco smoking caused three-quarters of a million deaths in the middle age group. In the Member States of the European Union in the 1990s, there were over one-quarter of a million deaths in the middle age group directly caused by tobacco smoking, which included 219,700 deaths in men and 31,900 in women. There were many more deaths caused by tobacco at older ages. In countries of central and eastern Europe, including the former Union of Soviet Socialist Republics, there were 441,200 deaths in middle-aged men and 42,100 deaths in women. Several epidemiological studies examining the factors responsible for the interindividual differences in the susceptibility to tobacco-related cancers and cardiovascular diseases are being performed in the United States, Europe and Japan. Although still not common practice, many of the newer studies are employing molecular genetic assays in conjunction with epidemiology to identify genotypes susceptible to disease development and select suitable biomarkers of tobacco smoke exposure. The frequency of investigations in the area of cigarette smoke composition and chemistry decreased during the last decade. Nonetheless, there are ample data to suggest that cigarette smoke is a highly complex mixture that contains approximately 4800 different compounds (8). Approximately 100 of these compounds are known carcinogens, cocarcinogens and/or mutagens. The complex mixture also contains gases such as ozone, formaldehyde, ammonia, carbon monoxide, toluene and benzene, and about 1010 particles of different sizes in each mL of mainstream smoke. In addition, a number of other toxic, mutagenic, tumour promoter and/or cocarcinogenic substances have been identified in both mainstream and sidestream cigarette smoke over the years. Many chemical and biological assays of smoke condensates have also documented the presence of potent inhibitors of carcinogenesis in smoke. Such a complex chemical composition of smoke has made it difficult to determine the active constituent(s) responsible for the tobacco-related health risks of smoking and has led to studies of individual constituents of smoke such as polycyclic aromatic hydrocarbons (PAH), nitrosamines and nicotine. Thus, over the years, various individual groups of smoke constituents have been the focus of research at different times. For example, studies of PAH were in vogue during the 1970s and 1980s, followed by nitrosamines in the 1990s. Tobacco alkaloids have long been studied because of their pharmacological activity and have attracted increased attention because of their suspected role in addiction, smoking behaviour and cessation. However, it is also being realized now that the health effects of this complex mixture are likely to result from a combined effect of these chemicals through multiple mechanisms rather than as result of the effects of a single smoke constituent. The mixture contains compounds belonging to almost every class of chemicals that are toxic and protective, agonist and antagonist, carcinogenic and anticarcinogenic, and exists in the gaseous as well as the particulate phase. Extensive studies on the chemical constituents of tobacco smoke and their relationship to disease were published by Hoffmann and Hoffmann of the American Health Foundation (8). Newer studies have largely focused on the comparative chemistry of mainstream and sidestream smoke. Interest in the free radical chemistry of smoke has resurfaced due to the realization that smoke-induced oxidative injury may play an important role in the etiology of a variety of tobacco-related diseases. Pioneering studies on the free radical chemistry of tobacco smoke, performed in the laboratory of William Pryor at the Louisiana State University (9), identified short- and long-lived radicals in mainstream and sidestream cigarette smoke, and implicated them in various smoking-associated disease etiologies. Go to: TOBACCO-RELATED CARDIOVASCULAR DISEASE Cardiovascular diseases, and atherosclerosis in particular, are the leading causes of death in industrial societies. The predominant underlying cause of coronary artery disease (CAD) is atherogenesis, which also causes atherosclerotic aortic and peripheral vascular diseases. Cigarette smoking, independently and synergistically with other risk factors such as hypertension and hypercholesterolemia, contributes to the development and promotion of the atherosclerotic process. Various studies have shown that the risk of developing CAD increases with the number of cigarettes smoked per day, total number of smoking years and the age of initiation, thus indicating a dose-related response. In contrast, cessation of smoking is reported to reduce mortality and morbidity from atherosclerotic vascular disease. The mechanisms through which smoking influences the development and progression of atherosclerosis are poorly understood at present, but recent studies point to an adverse effect of smoking on endothelial and smooth muscle cell functions as well as thrombotic disturbances produced by tobacco smoke (10,11). With the use of modern ultrasonographic techniques, three independent studies performed in the United States, Europe and Australia have demonstrated that both active and passive smokers exhibit impaired endothelium-dependent vasoregulation (12–14). Some degree of recovery of endothelial function in ex-passive smokers who have stayed away from smoke-contaminated environments further supported a secondary role of smoke in endothelial dysfunction (15). Evidence has been presented that tobacco-related impairment of endothelial function may be related to its adverse effects on endothelial nitric oxide (NO) synthase (16,17). An association between a genetic polymorphism of the endothelial NO synthase gene and the predisposition of smokers to CAD was reported (18,19). Additionally, studies report that smoke interferes with L-arginine and NO metabolism, resulting in reduced NO formation (20). Upregulation of the expression of endothelial cell adhesion molecules (CAMs) such as vascular CAM-1 and intercellular CAM-1 by smoke condensates, and stimulation of leukocyte and endothelium attachment by exposure to cigarette smoke was demonstrated (21). Cigarette smoke extract has been shown to induce expression of CAMs (22). However, the expression of a specific adhesion molecule is determined in vivo and the relationship between various events is poorly understood. Exposure to tobacco smoke is known to increase oxidative stress in the body by various mechanisms, including depletion of plasma antioxidants such as vitamin C. At least two studies have been performed to determine the role of oxidative stress in increasing leukocyte-endothelial interactions that precede the development of atherosclerosis in smokers. One study showed that a high intake of vitamin C by smokers significantly reduced the adhesiveness of their monocytes to endothelial cells (23). However, in a second study, sera from young smokers was collected before and after a single oral supplementation with vitamin C and L-arginine (a substrate for NO production). The sera were tested for promotion of the adherence of human monocytes to human umbilical vein endothelial cell monolayers. It was shown that while oral L-arginine caused reduction in such leukocyte adherence, no reduction was seen with vitamin C supplementation (24). This suggested that the NO levels may be important in smoking-induced leukocyte-endothelial interactions, at least during the early stages. Neither NO nor any other markers of oxidative stress were measured in either of these studies. The levels of 8-hydroxydeoxyguanosine, an oxidized DNA product, and F2-isoprostane, an oxidative arachidonic acid product, were found to be elevated in passive smokers (25,26). Oxidation of low-density lipoprotein (LDL), which is a gold standard risk factor of the atherosclerotic process, was also found to be elevated in smokers, as determined by the presence of increased levels of autoantibodies against oxidized LDL. It was further demonstrated that dietary supplementation with a lipid-soluble antioxidant, α-tocopherol, significantly reduced plasma levels of oxidized LDL autoantibodies (27). Similarly, intake of a mixture of antioxidants was found to increase the resistance of smoker LDL to oxidative modification (28) and reduce the plasma levels of 8-hydroxydeoxyguanosine in passive smokers (25). These studies have thus identified newer, more specific markers of oxidative stress that can be used as biomarkers of oxidant injury and used for the development of dietary and/or pharmacological interventions against disease development. Relatively few studies related to cardiovascular effects of cigarette smoke have been performed in rodent models. Such animal studies are, however, needed to delineate the role of different mechanisms in promoting atherosclerotic disease and for developing appropriate interventions. Go to: TOBACCO-RELATED CANCERS Tobacco carcinogenesis has remained a focus of research during the past 10 years, and various epidemiological and experimental studies have not only confirmed the major role of tobacco smoke exposure in lung and bladder cancers, but have also reported on its association with cancers of various other sites, such as the oral cavity, esophagus, colon, pancreas, breast, larynx and kidney. It is also associated with leukemia, especially acute myeloid leukemia.