CHLAMYDIA INFECTIONS DURING PREGNANCY

To the Editor:

Saxer's article on *Chlamydia trachomatis (Saxer JJ: Chlamydia trachomatis genital infections in a community-based family practice J Fam Pract 1989; 28:41-47)* included a small sample of pregnant women with chlamydia infections. He made no recommendation on screening for treatment of chlamydia infections in pregnancy. Our practice is a community health center that sees both private and indigent patients. Of the 105 obstetric patients currently registered in our practice, 13 were found on routine screening to be positive for *Chlamydia trachomatis* by fluorescent antibody testing, a positive rate of 13%. None of the pregnant women had a positive gonorrhea culture. Twenty-seven percent of these patients had Medicaid and 10% had private insurance, the remainder being medically indigent. All patients were successfully treated with oral erythromycin, with cure documented.

Chlamydia infection in pregnancy has been associated with premature rupture of membranes and premature delivery.[1] These results suggest that, at least in the medically indigent population, screening for chlamydia infection should be performed as a routine part of prenatal care, and such infection should be treated.

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Reference

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The preceding letter was referred to Dr. Saxer, who responds as follows:

Dr. Devitt brings up another important point regarding *Chlamydia trachomatis* genital infections. *C trachomatis* is responsible for ectopic pregnancies, premature rupture of membranes, premature delivery, neonatal transmission, and even increased perinatal mortality. *C trachomatis* may be the most common perinatal infectious agent. Early treatment can prevent complications or transmission to offspring.[1,2]

Recommendations regarding screening are controversial and depend on several factors as outlined by Frame and Phillips.[3,4] Therefore, physicians should individualize their care to the populations that they serve.

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2. Schachter J, Grossman M, Sweet RL, et al: Prospective study of perinatal transmission of Chlamydia trachomatis. JAMA 1986; 255:3374–3377
3. Frame PS: A critical review of adult health maintenance, Part 2. Prevention of infectious diseases. J Fam Pract 1986; 22:417–422
4. Phillips RS, Aronson MD, Taylor WC, et al: Should tests for Chlamydia trachomatis cervical infection be done during routine gynecologic visits? Ann Intern Med 1987; 107:188–194

COMMUNITY-ORIENTED PRIMARY CARE

To the Editor:

I was pleased to see the Controversies in Family Practice feature focusing on the question "Is Community-Oriented Primary Care a Viable Concept in Actual Practice?" (*Frame PS: An affirmative view. O'Connor PJ: An opposing view. J Fam Pract 1988; 28:203–208).* I would like to expand upon some of the points raised by both Drs. Frame and O'Connor in their respective affirmative and opposing views.

Dr. Frame cited some of the previous work dating back to the 19th century and gave some examples of community-oriented primary care (COPC) activities from his own practice. One of the essential components of COPC I believe Dr. Frame did not adequately emphasize in his commentary pertains to the role of the community in identifying health problems and developing programs to address the problems.[1] The examples Dr. Frame cited from his own practice were primarily initiated and sustained by the physicians. Based on his descriptions, the alcohol and drug abuse program was the only one that involved broad-based community participation in a substantial way.

Dr. Frame appropriately acknowledged that COPC can "seem quite intimidating to the harried physician." If the physician is able to assemble a broad-based community group to work with him or her on a particular project, the enormity of the task is much less formidable. Responsibility for components of the project can be distributed among members of the group so that the success or failure of the project does not rest only on the shoulders of the physician.

The opposing view presented by Dr. O'Connor was also well stated. He appropriately indicated that few primary care physicians have received training specifically to do COPC. As we integrate the teaching of prevention more effectively into medical education and residency training, more physicians will acquire the skills necessary to do COPC. If the team approach referred to above is utilized, postgraduate training in public health as suggested by Dr. O'Connor will not be necessary for physicians to do COPC. Rather, the community group can include people with expertise in public health and epidemiology who can complement the clinical skills and insights of the physician.

Dr. O'Connor stated that the "competitiveness, diversity, and lack of coordination that characterize patient care in the United States medical care system make application of COPC a practical impossibility for most practicing physicians." On the contrary, the problems presently existing in the US health care system provide a unique opportunity for physicians to integrate the principles of COPC in their clinical practices. Physicians

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who work with the community and develop services to meet the needs of a defined population can distinguish themselves as community-responsive physicians.[1] Physicians from different practices can work together with the community on COPC projects, thereby avoiding the competition and ill-will about which Dr. O'Connor raises concern.

Dr. O'Connor pointed out that COPC has not been effectively tested in nonpoverty populations. The Kellogg Foundation and the National Rural Health Association have joined together to implement the National COPC Demonstration Project. Over the next few years, the results of this project will expand the research base of COPC as practiced in the offices of primary care physicians in the United States.

If COPC proves to be a viable model for the provision of primary care services, its implementation in this country may have a meaningful role in reshaping the health care delivery system in the United States

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Reference

1. Nutting PA (ed): Community-Oriented Primary Care: From Principle to Practice. Health Resources and Services Administration (Rockville, Md.). HRSA publication No. HRS-A-PE86-1. Government Printing Office, 1987, pp 6–9, 357–406

SMOKING CESSATION

To the Editor:

Gilbert et al (*Gilbert JR, Wilson DMC, Best JA, et al: Smoking cessation in primary care: A randomized controlled trial of nicotine-bearing chewing gum. J Fam Pract 1989; 28:49–55*) demonstrated a well-implemented research study on the effectiveness, or rather noneffectiveness, of the addition of nicotine-bearing chewing gum to physician-based support sessions. It would have been of interest to see the effect of a longer term of

support during that first year of cessation from smoking of patients known to have a high relapse rate.[1]

From my experience, very little, if any, effect may be obtained with the addition of nicotine gum if the patient does not believe, due to experience, hearsay, or otherwise, that the gum will be effective. More important, an individual who believes that he or she is worthless, for example, and so deserves to die an early death, will not likely benefit from the support or expertise on the part of most health professionals to quit smoking successfully. This is perhaps one extreme; however, an individual's health beliefs will impact on the performance of health-related behavior.[2,3] A smoker's perceptions of her susceptibility to smoking-related diseases (cancer, emphysema, bronchitis) and the potential seriousness of these diseases, as well as the motivation for general positive health and the perceived efficacy of preventive health measures (quit smoking!), will comprise a more total picture of health belief.[2,3] This is to say that much will be added to studies of smoking cessation with the addition of controlling for the important confounding variable of health beliefs.

Finally, this experiment would be improved with the addition of double-blinding, at least including another group, ie, "placebo-gum and support," with the patients and physicians blind to the type of gum. This will address more directly the issue of the effectivenss of nicotine-bearing gum.

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References

1. American Thoracic Society: Cigarette smoking and health. Am Rev Respir Dis 1985; 132:1133–1136
2. Becker MH (ed): The health belief model and personal health behavior. Health Educ Monogr 1974; 2:326–473
3. Weinberger M, Greene JY, Mamlin JJ, Jerin MJ: Health beliefs and smoking behavior. Am J Public Health 1981; 71:1253–1255

The preceding letter was referred to Dr. Gilbert and colleagues, who respond as follows:

Dr. Armetta has made comments relevant to studies of physician-initiated smoking cessation in general, and our study, in particular. We wish to respond to several issues which she raised.

Length of support: By far the greatest degree of recidivism occurs in the first 3 months after quitting. Our experience is that a relatively small proportion of patients take advantage of all follow-up visits even up to 2 months after quitting. We anticipate the rate of compliance with follow-up would drop fairly dramatically beyond this point. More important, we were trying to develop a maneuver that was reasonably affordable, and the addition of further follow-up might make the cost prohibitive.

Belief in benefit: We agree that a patient who does not believe the gum to be effective will be unlikely to comply with the suggested prescription.

Health beliefs: Patients' health beliefs and feelings of self-worth likely influence their chances of quitting. It is unlikely that patients who are low in self-esteem or who believe they are not susceptible to smoking-related disease will be influenced by a nonintensive maneuver. Again, we would emphasize that the study is focused on the efficacy of relatively nonintensive maneuvers. Even if health beliefs do influence outcome, randomization should solve the problem of equality of groups, particularly when sample size is large.

Placebo control of gum: A placebo control and blinding are preferable to a no-gum control because of the possibility of placebo effects. In the trial which we reported, one would expect the lack of a placebo to enhance the effect of gum. That nicotine gum is no better than a no-gum control enhances our belief that nicotine gum is unlikely to be effective in a general practice setting.

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SERUM CHOLESTEROL AND GLYCOSYLATED HEMOGLOBIN

To the Editor:

I wish to applaud the study conducted by Urberg and Rajdev (*Urberg M, Rajdev K: A correlation between serum cholesterol and glycosylated hemoglobin in nondiabetic humans. J Fam Pract 1989; 28:269–274*) in which they found a significant relationship between serum cholesterol and levels of glycosylated hemoglobin in nondiabetics. The discussion from this study and the corresponding commentary by Neighbor have wide-reaching implications and raise numerous corollary questions and comments.

First, the impairment of glucose metabolism should be thought of as a continuum with the constellation of these impairments better thought of, and referred to, as *glucopathies.*

Second, this study lends support to Yudkin's[1] contention that there is more epidemiological correlation between the intake of sugar and coronary artery disease than with the intake of animal fats, as propounded by Keys.[2]

Third, can levels of glycosylated hemoglobin be used as an early indicator of premature atherosclerosis in asymptomatic individuals?

Fourth, insofar as glycosylated hemoglobin is an indicator of impaired glucose metabolism, is there a serum counterpart to indicate *abnormal* lipid metabolism with tissue binding and deposition?

Fifth, since diabetes is thought of as a model for the aging process by gerontologists,[3] should primary care physicians use glycosylated hemoglobin not only as a marker for cardiovascular diseases but as a biomarker of physiological aging?

Sixth, would glycosylation inhibitors, such as aminoguanidine as studied by Cerami et al,[4] not only prevent diabetic complications but also atherogenesis and in turn the process of aging?

Urberg and Rajdev touch upon exciting and fundamental problems in medicine of particular interest to fam-

ily physicians concerned with health promotion and disease prevention. Further studies are needed and should be encouraged.

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2. Keys A: The diet and the development of coronary artery disease. J Chronic Dis 1956; 4:364–380
3. Kohn RR: The question of accelerated collagen aging in diabetes mellitus. In Korenman SG: Endocrine Aspects of Aging. New York, Elsevier, 1982
4. Cerami A, Vlassara H, Brownlee M: Glucose and aging. Sci Am 1987; 256:90–96

To the Editor:

I read with interest the article entitled "A Correlation Between Serum Cholesterol and Glycosylated Hemoglobin in Nondiabetic Humans."[1] While this article suggests a relationship between elevation of serum cholesterol and elevation in glycosylated hemoglobin, several major flaws of this study should be brought to the attention of the reader.

As mentioned in the commentary by Dr. Neighbor, there is a potential for a significant selection bias in this article. There is no mention of random selection of subjects. No description of the study population is included. In addition, a single fasting glucose determination may not be sufficient to rule out diabetes in this patient population. Thus, we may not be able to apply the results noted in this study to our own nondiabetic primary care populations.

Another important flaw not addressed in the study was the lack of control for significant confounders. Confounders known to be associated with both cholesterol and diabetes (diet, body mass, or age) should have been either addressed at the time of patient selection, or dealt with in the analysis of the data.

In light of these significant flaws, we should use the study presented here to raise the question of whether a true association exists between serum cholesterol and glycosylated hemoglobin in nondiabetic patients. Perhaps this article will prompt these and other researchers to study this question in a more comprehensive and controlled fashion.

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Reference

1. Urberg M, Rajdev K: A correlation between serum cholesterol and glycosylated hemoglobin in nondiabetic humans. J Fam Pract 1989; 3:269–274

The preceding letter was referred to Dr. Neighbor, who responds as follows:

The paper "A Correlation Between Serum Cholesterol and Glycosylated Hemoglobin in Nondiabetic Humans" by Martin Urberg and Kalpana Rajdev[1] identifies a significant strongly positive correlation between stable glycosylated hemoglobin and total blood cholesterol in nondiabetic humans. A similar relationship was identified in a larger study conducted by Elizabeth Barrett-Connor et al in 1984.[2] This paper was not cited by Urberg and Rajdev and did not appear in the initial literature search I conducted in preparing the commentary for their article.

In the study by Barrett-Connor et al the relationship between glycosylated hemoglobin and plasma cholesterol was examined in 558 euglycemic adults aged 40 to 79 years who had no history of diabetes. Pearson's product moment correlation for glycosylated hemoglobin and choelsterol ws 0.12 ($P<.05$). A similar correlation was noted between glycosylated hemoglobin and low-density lipoprotein cholesterol. Neither correlation coefficient changed significantly after adjustment for age and body weight.

This correlation between glycosylated hemoglobin and cholesterol is much less than the correlation of 0.63 identified by Urberg and Rajdev. What is the reason for this difference? Possible reasons include the differences in the range of total cholesterol values,the exclusion of outlier values in the study by Barrett-Connor et al, and the lack of adjustment for age and body weight in the study by Urberg and Rajdev. Additionally, the relatively large differences in the variances of both glycosylated hemoglobin and total cholesterol in the two studies would result in different correlation coefficients even though the true slope of the fitted line describing the relationship is identical.[3] However, a simple calculation of the slopes from the values of r and variances of glycosylated hemoglobin and total cholesterol in the two studies reveals greatly different calculated slopes.

The key difference that may account for difference in findings between the two studies is the difference in the type of glycosylated hemoglobin measured. Barrett-Connor et al measured total glycosylated hemoglobin by an ion-exchange chromatographic method, whereas Urberg and Rajdev measured stable glycosylated hemoglobin using resin affinity chromatography. The more precise measure of recent glucose metabolism used by Urberg and Rajdev probably accounts for the finding of a stronger correlation. This highlights the importance of considering differences in methods of measurement when comparing studies involving glycosylated hemoglobin.

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