

26

Asthma Exacerbated at Work

Gregory R. Wagner

Department of Environmental Health, Harvard School of Public Health, Boston, Massachusetts, and NIOSH, Washington, D.C., U.S.A.

Paul K. Henneberger

Division of Respiratory Disease Studies, NIOSH/CDC, Morgantown, West Virginia, U.S.A.

INTRODUCTION

Exacerbation of asthma can result from exposures at home, at work, in the outdoor environment, and in public buildings. There is general agreement in clinical practice that troublesome home environmental exposures should be avoided; physicians often advise asthma patients to rid their homes of carpets, drapes, and other furnishings that might be repositories for allergens. Asthma-related exposures in the work environment are less frequently addressed, because exposures are often beyond the control of the individual patient and an employer may or may not accept their responsibility or have the ability to control exposures. The issue of an affected employee's right to workplace accommodation or compensation further clouds the issue.

Specific studies of asthma exacerbated by workplace exposures are limited in number. In many studies of work-related asthma, work-exacerbated asthma (WEA) is not clearly differentiated from asthma with a work-related onset. For example, the European Community Respiratory Health Survey classifies participants by their current or most recent job, for those who are no longer employed, or by the job they left due to health problems (1,2). The conditions of that job are then used to determine whether the subject was at risk for exposure to asthma agents; this information is used to determine whether the asthma was work-related. In a population-based study conducted in Norway, the researchers asked participants whether they had ever had respiratory symptoms in relation to work (3). While these studies are good for identifying the full impact of work on asthma, they do not separate cases characterized by work-related exacerbation of existing asthma from those characterized by work-related onset of asthma. Other investigators have defined and studied WEA as a separate subcategory within the larger category of occupational asthma or work-related asthma.

DEFINITIONS OF WEA

The American College of Chest Physicians published a consensus document on assessment of work-related asthma in 1995 (4). Based on the criteria presented in that document, people were considered to have work-aggravated asthma if they had a diagnosis of asthma and an association between asthma symptoms and work characterized by (i) the presence of asthma symptoms or related medication before entering a new occupational exposure setting and (ii) an increase in symptoms or the need for more or new medication after entering a new occupational exposure setting (4). A similar definition was proposed later in the 1990s as a surveillance case definition for use in the Sentinel Event Notification Systems for Occupational Risks (SENSOR) (5). The SENSOR criteria for work-aggravated asthma are (a) health-care professional's diagnosis consistent with asthma, (b) an association between symptoms and work, (c) asthma symptoms or treatment with asthma medication within the two years before entering a new occupational setting, and (d) increased asthma symptoms or increased asthma medication use upon entering a new occupational setting. There is a longitudinal component to these definitions of work-aggravated asthma. Asthma onset and the presence of asthma symptoms or related medication use must come before entering the new occupational exposure setting, and the condition must worsen after entering. Thus, the clinician or researcher must obtain, either prospectively or retrospectively, knowledge of asthma status before and after subjects enter a new occupational exposure setting.

Medical records may be useful to document an asthma patient's change in status. For example, in a study of recruits who entered the Israel Defense Force at the age of 18 to 21 years, baseline asthma status was established at the time of induction into the military and repeated clinical evaluations documented changes in asthma status over time (6). However, in the absence of medical records, determination of the progression of disease will often depend, at least in part, on subject or patient recall.

Another approach to the definition of WEA is to use self-reported data gathered by questionnaire in clinic- (7,8) or population-based (9–12) studies to determine whether work makes symptoms worse for people with asthma. The definitions for WEA varied to some extent among these studies, as indicated in Table 1. All of the studies were cross-sectional, with the exception of the one by Tarlo et al. (8) which was a retrospective review of data gathered from asthma patients over a 19-year period.

FREQUENCY OF WEA

The frequency of WEA sometimes has been expressed as a percentage of some larger group of work-related asthma cases. From the surveillance of work-related asthma conducted by four states as part of the National Institute for Occupational Safety and Health-sponsored (NIOSH) SENSOR program, 19.1% of the cases registered during 1993–1995 were classified as work-aggravated asthma rather than new-onset asthma (5). From a case series of patients referred for occupational asthma to an occupational and environmental clinic in the United States, 27% were judged to have exacerbation of preexisting asthma (13). Tarlo et al. (14) investigated the status of 469 asthma claims that were accepted by the Ontario Workers' Compensation Board (WCB) during 1984–1988. The WCB group included 234 cases (49.9%) with

Table 1 The Frequency of Work-Exacerbated Asthma

Reference (location of study)	Setting	Definition of work-exacerbated asthma	% with work-exacerbated asthma (<i>n</i> = number with asthma)	
			Among adults with asthma	Among employed adults with asthma
Abramson et al. (9) (Victoria, Australia)	Community based	Self-reported: symptoms exacerbated by workplace conditions	20% (<i>n</i> = 159)	NA ^a
Henneberger et al. (7) (Colorado, U.S.A.)	HMO ^b	Self-reported: current work environment makes asthma worse	25% (<i>n</i> = 1461)	NA
Henneberger et al. (10) (Maine, U.S.A.)	Community based	Self-reported: coughing or wheezing is worse at work	18% (<i>n</i> = 88)	25% (<i>n</i> = 64)
Johnson et al. (11) (6 locations in Canada)	Community based	Self-reported: wheezing or dyspnea at or after work in current job	Wheezing 34% Dyspnea 31% (<i>n</i> = 106) ^c	NA
Saarinén et al. (12) (Finland)	Population based (from those enrolled in national health insurance)	Self-reported: asthma symptoms were caused or made worse by work at least weekly during the past month	NA	21% (<i>n</i> = 969)
Tarlo et al. (8) (Ontario, Canada)	Secondary- and tertiary-referral asthma clinic	Self-reported: worsening of asthma at work and workplace exposure to recognized aggravating factors (including emotional stress), but no likely workplace exposure to sensitizers	3.7% (<i>n</i> = 682)	8.1% (<i>n</i> = 310)

^aNA, not addressed in publication.^bHMO, health maintenance organization.^cLimited to those with adult-onset asthma who did not have probable or possible work-related onset of asthma.

aggravation of asthma (AA). In a publication based on the same 469 compensated cases, 68 (14.5% of the 469 total cases and 29.1% of the AA cases) were classified as accident-related AA, which was defined as asthma with worsening of symptoms after an acute accidental exposure to respiratory irritants (15).

Other researchers have identified adults with asthma by clinic records or population-based surveys and then estimated the percentage of the cases having work-exacerbated symptoms. As presented in Table 1, these estimates ranged from approximately 3.7% to 34% among adults with asthma and 8.1% to 25% among employed adults with asthma. This broad range of results may reflect, in part, differences among the studies in subject inclusion criteria and in outcome definition as well as in the source of subjects (Table 1). Some researchers studied asthma patients in a health care setting drawn from the general population, while others looked at people who work and report new symptom patterns associated with their work environment. For example, Tarlo et al. (8) examined the prevalence of work-related asthma among 682 adults (age greater than 17 years) who were diagnosed with asthma in a secondary- and tertiary-referral asthma clinic in Canada (8). Among the 50 cases who reported during their initial clinic visit that their asthma was worse at work, 25 were judged by the investigators to have work-related AA. These 25 cases represented 3.7% of all 682 cases and 8.1% of the 310 cases who had onset of asthma as adults and were employed at the time of the initial clinic visit (8). In a cross-sectional study conducted in a health maintenance organization (HMO) in the United States, 1461 adults with asthma completed a questionnaire (7). This cohort included any adult in the HMO population with a diagnosis of asthma, regardless of whether they were treated by their primary care physician or by a specialist. When asked, 25% of the HMO study participants reported that their asthma was made worse by their current work environment (7).

In a community-based study conducted in Australia, 20% of adults with asthma reported that their symptoms were exacerbated by workplace exposures (9). In a population-based study conducted in the state of Maine in the United States, 16 participants with asthma reported that their coughing or wheezing was worse at work; these participants represented 18.2% of all 88 asthma cases and 25% of the subset of 64 cases who were employed (10). In a study conducted in Finland, the researchers interviewed adults with asthma who had been granted reimbursement for asthma medication by the Finnish Social Insurance Institution (12). During interview, approximately 21% of 969 working adults with asthma reported work-related aggravation of respiratory symptoms at least once a week during the past month (12). The study by Johnson et al. (11) in Canada followed the model of the European Community Respiratory Health Survey. The article was focused on those who fulfilled criteria for probable or possible work-related onset of asthma, but also included findings for others with adult-onset asthma. Wheezing or dyspnea at or after work was reported by 34% and 31%, respectively, of this latter group (12).

EXCESS FREQUENCY OF WEA

One way to address how often work contributes to the exacerbation of asthma is to compare high- to low-risk occupations. In the study of military recruits in Israel, researchers computed the cumulative incidence of asthma onset and worsening over the 30-month study period, which made it possible to compare these figures between different occupational categories (6). Among recruits with a history of rare and mild

attacks of dyspnea, normal spirometry, and response to exercise at military induction, worsening of asthma was greater among those in combat units ($78/370 = 21.1\%$) and maintenance units ($36/236 = 15.3\%$) than among those who performed clerical tasks ($11/193 = 5.7\%$). Thus, the excess cumulative incidence, presumably attributable to occupation, was 15.4% for those in combat units and 9.6% for those in maintenance units. Among recruits with mild asthma at induction, characterized by not requiring daily medications and having either mildly impaired spirometry or moderate bronchial hyperresponsiveness, $124/639 = 19.4\%$ of those in the maintenance units had worsening of symptoms compared to $113/606 = 18.6\%$ among those performing clerical tasks.

DISTINCTIVE FEATURES OF ADULTS WITH WEA

A few studies have compared adults with WEA to other adults with asthma or other adults with work-related asthma. Looking first at demographic features (Table 2), the two studies that made comparisons with all other adults with asthma agreed on two findings: The people with WEA were, on average, older but there was little difference by gender (7,12). These two studies did not agree on the impact of cigarette smoking, with one study finding no difference (12) and the other finding that cigarette smoking was more common among asthmatics with WEA (7). One study alone examined race, income, and education as risk factors and found being non-white, having lower income, and having less education were associated with work-exacerbation of asthma (7). When Goe et al. (16) used other adults with work-related asthma as the basis for comparison, they found that people with WEA were more likely to be female, young, nonwhite, and nonsmokers.

Table 2 Distinctive Demographic Features of Adults with Work-Exacerbated Asthma

Characteristics of adults with work-exacerbated asthma	Compared to other adults with asthma		Compared to other adults with work-related asthma
	Henneberger et al. (7)	Saarinen et al. (12)	Goe et al. (16)
Gender			
More women	No	No	Yes
Age			
Older	Yes	Yes	No
Younger	No	No	Yes
Race			
More nonwhites	Yes	NA ^a	Yes
Education			
Less	Yes	NA	NA
Annual income			
Lower	Yes	NA	NA
Ever smoked cigarettes			
More likely	Yes	No	No
Less likely	No	No	Yes

^aNA, not addressed in publication.

Table 3 Distinctive Health and Health Care Features of Adults with Work-Exacerbated Asthma

Adults with work-exacerbated asthma were more likely to have had:	Compared to other adults with asthma		Compared to other adults with work-related asthma
	Henneberger et al. (7)	Saarinen et al. (12)	Goe et al. (16)
Chronic bronchitis	Yes	NA	NA
Emphysema or COPD	Yes	NA	NA
Allergies	NA	NA	Yes
A family history of allergies or asthma	NA	NA	Yes
Onset of asthma as an adult	NA	Yes	NA
At least one treatment for asthma attack in past year	Yes	NA	NA
A need for asthma medication continuously in past 12 months	NA	Yes	NA
More days with asthma symptoms in past two weeks	Yes	NA	NA
More severe asthma, based on self-report	Yes	NA	NA

Abbreviations: COPD, chronic obstructive pulmonary disease; NA, not addressed in publication.

Distinctive health and health care features of adults with WEA are presented in Table 3. Each item presented is unique to one of the three studies already mentioned (7,12,16). These findings suggest that the people with work-exacerbated symptoms tend to have a more severe form of disease than other adults with asthma.

CLINICAL APPROACH TO A PATIENT WITH WEA

An argument for differentiating between asthma caused by specific sensitizers and WEA is the presumed difference in prognosis with continued workplace exposures. The progression of asthma severity as the result of persistent exposure to sensitizing agents is well recognized, and is the basis for the clinical recommendation to remove someone from exposure, as soon as possible, after diagnosis of occupational asthma (17). Continued exposure to lower levels of the sensitizer and more careful clinical monitoring will often not prevent worsening of the patient's asthma. With irritant-induced or exacerbated work-related asthma, the patient might be able to continue working with lowered exposures and better control of symptoms with medication and not suffer a progression in asthma severity. However, the long-term prognosis for such asthma patients requires additional study (17). Clinical approaches to WEA should include:

- Careful evaluation to ensure that the diagnosis is accurate and is not a misdiagnosis of occupational asthma due to specific sensitizing agents;
- Minimization of exposures triggering exacerbations, preferably through engineering controls;

- Workplace accommodation through job placement away from known triggers in work areas with the least potential exposure to irritants;
- Occasional supplemental use of personal respirators where necessary to protect against unusual conditions. Individuals using respirators must be fit tested and instructed in the use and care of the devices as part of a comprehensive respiratory protection program.
- Referral, as appropriate, to sources of information about relevant social support programs.

CHALLENGES IN STUDYING WEA

Most cross-sectional studies have relied on self reporting to establish WEA status among adults with asthma (7,9–12). With self-reporting goes the opportunity for misclassification due to under- and/or overreporting. Workers with preexisting or quiescent asthma who become sensitized to a new work agent are often erroneously thought to be suffering exacerbations of their preexisting asthma. This is not only a challenge in epidemiologic investigations but is also particularly important clinically, as the newly sensitized individuals should be removed from the exposure as quickly as possible.

Some researchers have gone beyond self-reports of WEA. The studies that utilized data from a surveillance program, like SENSOR in the United States (16), or from an asthma clinic, like the study from Ontario, Canada (8), had a more intimate knowledge of the patients, and presumably used that knowledge to judge the veracity of self reports. Another approach was pursued by Milton et al. (18) in a pilot study of new-onset work-related asthma, but it could also be used to investigate WEA. Specifically, the WEA case definition could include both the subject's self report of WEA and the judgment of experts that the subject had experienced occupational exposures that could exacerbate asthma. This would have the advantage of setting a higher standard for WEA case status than has regularly been used in cross-sectional studies.

The need for immediate health care, whether at an emergency department in a hospital, a critical care clinic, or the office of the subject's doctor or increased use of medication are reasonable "objective" indicators of asthma exacerbation. Such visits or medication changes are documented in medical and pharmacy records, eliminating the need to rely on self-reporting alone for the worsening of symptoms. This approach requires access to medical records, which could be monitored prospectively or reviewed retrospectively. There remains the challenge of establishing that conditions at work led to the need for acute care.

Some have suggested that the exacerbation of asthma by workplace exposures might be relatively benign and not contribute to the worsening of the individual's underlying condition (19). One far from benign consequence of WEA is leaving work, either temporarily or permanently. A question about having to leave work due to respiratory health problems has been used in the questionnaires of the European Community Respiratory Health Survey. Moreover, in the province of Ontario in Canada, workers can receive compensation for absences of only a few weeks as the result of WEA. While this endpoint does not directly document an increase in airway inflammation or bronchial hyperresponsiveness, it is easy to collect by questionnaire and is a measure of the impact of occupational exposures on asthma.

With enough clinical data, it would be possible to document that someone with asthma has experienced an increase in airway inflammation or bronchial hyperresponsiveness. This would require repeated clinical follow-up, and also require the determination of whether changes were related to work rather than some other source of harmful exposures. Researchers could also determine the severity level of asthma in patients over time and whether changes were related to workplace exposures.

CLASSIFICATION OF WEA

There have been disagreements within the occupational respiratory research community on how to classify WEA. In an editorial published in 1998, Wagner and Wegman noted that occupational asthma is usually defined "by the specific response to an agent capable of provoking sensitization" (20). They argued that this definition of occupational asthma should be broadened to include "preexisting asthma exacerbated by workplace environmental exposures," noting that this condition can result in significant disability, and that broadening the definition of occupational asthma would motivate prevention. Other researchers, commenting on the editorial, argued that WEA should not be considered as occupational asthma, but should be included in the broader category of work-related asthma (19). The commenting authors justified a more restrictive definition of occupational asthma on the basis of different mechanisms of disease, different medical approaches, and the need to treat the conditions differently in social insurance (workers' compensation) systems. These apparent differences have diminished over time as the diverse mechanisms of asthma initiation and exacerbation are explored. Recognition that WEA is an important source of work disability and a target for prevention is increasing, and the need for social insurance and compensation systems to provide assistance for people with WEA is starting to be addressed.

CONCLUSIONS

Exacerbation of asthma can result from exposures at home, at work, in the outdoor environment, and in public buildings. Asthma-related exposures in the work environment are less frequently addressed, because exposures are often beyond the control of the individual patient and an employer may or may not accept responsibility or have the ability to control exposures. The issue of an affected employee's right to workplace accommodation or compensation further clouds the issue. The incidence of asthma exacerbated at work among employed adults with asthma ranges between 8% and 25%, depending on the epidemiologic criteria utilized. Under specific exposure conditions, an excess cumulative incidence of WEA may be found in certain occupations. Compared to other adults with asthma, workers with WEA on average are older, and in one study this problem was more likely to be found in nonwhites and those with less education and lower incomes. Continued exposure to lower levels of the agents responsible for WEA must be avoided in the same manner as new-onset asthma. However, it is possible that some of these patients may continue working with reduced exposures and adequate use of controller medications without suffering progression of asthma severity, although close monitoring over time is necessary.

Most cross-sectional studies have relied on self-reporting to establish the presence of WEA. However, visits to hospitals, critical care clinics, or the office of the subject's doctor with a need for increased medication are reasonable "objective indicators" of asthma exacerbation. Current research demonstrates that WEA is an important source of work disability and an appropriate target for prevention. Additional outcomes research can help define how to address this problem more effectively.

DIRECTIONS OF FUTURE RESEARCH

- Prospective surveillance of newly hired workers (preferably apprentices) with histories of currently controlled asthma or asthma in remission in workplaces where asthma triggers are likely to be encountered.
- Outcomes in the above studies vis a vis: (i) number and frequency of asthma flares, (ii) the effects of workplace exposure on asthma control or severity—both short term and long term, (iii) the extent of temporary or permanent time lost from work as a result of exposure, (iv) the health consequences of continuing employment at such worksites, (v) the responses to various interventions, and (vi) amount of time required to resolve and reestablish control of asthma.
- Health surveillance programs should fine tune annual incidence data by including a separate category of WEA.
- Further examination of the economic, social (e.g., family), and productivity impact of work-exacerbated and new-onset asthma over time.
- Comparison of the incidence of new onset asthma and WEA in industries before and after introduction of optimal environmental controls.

REFERENCES

1. Fishwick D, Pearce N, D'Souza W, et al. Occupational asthma in New Zealanders: a population based study. *Occup Environ Med* 1997; 54: 301–306.
2. Kogevinas M, Anto JM, Soriano JB, Tobias A, Burney P. The risk of asthma attributable to occupational exposures: a population-based study in Spain. *Am J Respir Crit Care Med* 1996; 154:137–143.
3. Bakke PS, Gulsvik A. Work-related asthma: prevalence estimates by sex, age and smoking habits in a community sample. *Int J Tuberc Lung Dis* 2000; 4:649–656.
4. Chan-Yeung M. Assessment of asthma in the workplace. ACCP consensus statement. *Chest* 1995; 108:1084–1117.
5. Jajosky RA, Harrison R, Reinisch F, et al. Surveillance of work-related asthma in selected U.S. states using surveillance guidelines for state health departments, California, Massachusetts, Michigan, and New Jersey, 1993–1995. *MMWR CDC Surveill Summ* 1999; 48(3):1–20.
6. Katz I, Moshe S, Sosna J, Baum GL, Fink G, Shemer J. The occurrence, recrudescence, and worsening of asthma in a population of young adults: impact of varying types of occupation. *Chest* 1999; 116:614–618.
7. Henneberger PK, Hoffman CD, Magid DJ, Lyons EE. Work-related exacerbation of asthma. *Int J Occup Environ Health* 2002; 8:291–296.
8. Tarlo SM, Leung K, Broder I, Silverman F, Holness DL. Asthmatic subjects symptomatically worse at work: prevalence and characterization among a general asthma clinic population. *Chest* 2000; 118:1309–1314.

9. Abramson ML, Kutin JJ, Rosier MJ, Bowes G. Morbidity, medication, and trigger factors in a community sample of adults with asthma. *Med J Aust* 1995; 162:78–81.
10. Henneberger PK, Deprez RD, Asdigian N, Oliver LC, Derk S, Goe SK. Workplace exacerbation of asthma symptoms: findings from a population-based study in Maine. *Arch Environ Health* 2003; 58:781–788.
11. Johnson AR, Dimish-Ward HD, Manfreda J, et al. Occupational asthma in adults in six Canadian communities. *Am J Respir Crit Care Med* 2000; 162:2058–2062.
12. Saarinen K, Karjalainen A, Martikainen R, et al. Prevalence of work-aggravated symptoms in clinically established asthma. *Eur Respir J* 2003; 22:305–309.
13. Wheeler S, Rosenstock L, Barnhart S. A case series of 71 patients referred to a hospital-based occupational and environmental medicine clinic for occupational asthma. *West J Med* 1998; 168:98–104.
14. Tarlo SM, Liss G, Corey P, Broder I. A workers' compensation claim population for occupational asthma: comparison of subgroups. *Chest* 1995; 107:634–641.
15. Chatkin JM, Tarlo SM, Liss G, Banks D, Broder I. The outcome of asthma related to workplace irritant exposures: a comparison of irritant-induced asthma and irritant aggravation of asthma. *Chest* 1999; 116:1780–1785.
16. Goe SK, Henneberger PK, Reilly MJ, et al. A descriptive study of work-aggravated asthma. *Occup Environ Med* 2004; 61:512–517.
17. Friedman-Jimenez G, Beckett WS, Szeinuk J, Petsonk EL. Clinical evaluation, management, and prevention of work-related asthma. *Am J Ind Med* 2000; 37:121–141.
18. Milton DK, Solomon GM, Roseillo RA, Herrick RF. Risk and incidence of asthma attributable to occupational exposure among HMO members. *Am J Ind Med* 1998; 33:1–10.
19. Malo J-L, Chan-Yeung M. Comment on the editorial. Occupation asthma: prevention by definition. *Am J Ind Med* 1999; 35:207.
20. Wagner GR, Wegman DH. Occupational asthma: prevention by definition. *Am J Ind Med* 1998; 33:427–429.

NIOSH LIBRARY SYSTEM
MORGANTOWN LIBRARY
1095 WILLOWDALE ROAD
MORGANTOWN, WV 26505

ASTHMA IN THE WORKPLACE

And Related Conditions

THIRD EDITION

edited by

I. Leonard Bernstein

*University of Cincinnati
Cincinnati, Ohio, U.S.A.*

Maira Chan-Yeung

*University of British Columbia
Vancouver, British Columbia, Canada*

Jean-Luc Malo

*Université de Montréal
Montreal, Quebec, Canada*

David I. Bernstein

*University of Cincinnati
Cincinnati, Ohio, U.S.A.*



Taylor & Francis

Taylor & Francis Group
New York London

Published in 2006 by
Taylor & Francis Group
270 Madison Avenue
New York, NY 10016

© 2006 by Taylor & Francis Group, LLC

No claim to original U.S. Government works
Printed in the United States of America on acid-free paper
10 9 8 7 6 5 4 3 2 1

International Standard Book Number-10: 0-8247-2977-3 (Hardcover)
International Standard Book Number-13: 978-0-8247-2977-6 (Hardcover)

This book contains information obtained from authentic and highly regarded sources. Reprinted material is quoted with permission, and sources are indicated. A wide variety of references are listed. Reasonable efforts have been made to publish reliable data and information, but the author and the publisher cannot assume responsibility for the validity of all materials or for the consequences of their use.

No part of this book may be reprinted, reproduced, transmitted, or utilized in any form by any electronic, mechanical, or other means, now known or hereafter invented, including photocopying, microfilming, and recording, or in any information storage or retrieval system, without written permission from the publishers.

For permission to photocopy or use material electronically from this work, please access www.copyright.com (<http://www.copyright.com/>) or contact the Copyright Clearance Center, Inc. (CCC) 222 Rosewood Drive, Danvers, MA 01923, 978-750-8400. CCC is a not-for-profit organization that provides licenses and registration for a variety of users. For organizations that have been granted a photocopy license by the CCC, a separate system of payment has been arranged.

Trademark Notice: Product or corporate names may be trademarks or registered trademarks, and are used only for identification and explanation without intent to infringe.

Library of Congress Cataloging-in-Publication Data

Catalog record is available from the Library of Congress

informa
Taylor & Francis Group
is the Academic Division of Informa plc.

Visit the Taylor & Francis Web site at
<http://www.taylorandfrancis.com>