COMMENT LETTERS RECEIVED

DRAFT MITIGATED NEGATIVE DECLARATION
for the
CITRUS VILLAGE PROJECT

2 PART carbonless FORM 9042

MEMORANDUM

TO Steve Chase

FROM Lisa Blois

DATE 1/11/08

SUBJECT 7386 Calle Real

MESSAGE

This was mailed to me by Ricardo Ramos. (No first address, just Goleta)

I don't know if you have seen this before.

PLEASE REPLY BY _____ NO REPLY NECESSARY _____

Adams 9042

Memorandum

Ricardo Ramos
Goleta Calif. 93117

Environmental Complaint Dept.
Calif. Environmental Protection Agency
1001 I Street
Sacramento, Calif 95812-2815

Jan 5 2008

I am writing to request that the EPA look into the undocumented soils issues on a site located within the City of Goleta at 7388 Calle Real and moniter this project to its conclusion.

This project, originally proposed in 2000 as the El Encanto Apts by The County of Santa Barbara and Community Housing Corporation has been redesigned and is now called Citrus Village, case number 04-226-GP-DP-RN–07-MND-004 but still uses the original Mitigated Negative Declaration which allows the reuse and recompaction of 30000 cubic yards of undocumented fill soil with no testing requirements for toxic or hazardous substances. I have included the original 2001 documentation.

This Mitigated Negative Declaration has been fatally flawed from the start using fraudulent data, withholding pertinent information, and stonewalling the concerned public when they tried to determine the truth about the soils issues.

Additionally the roadway safety, traffic count, and accident numbers have been manipulated and Santa Barbara County employees have perjured themselves at public meetings about these facts, numbers and data. I have included 2 internal SB County emails from 2000.

There are also drainage and runoff problems into the adjacent El Encanto creek.

In conclusion this project must have a full and complete EIR before it moves any farther forward to address the very serious environmental impacts and so every taxpayer will know the truth.

Sincerly

Ricardo Ramos

cc City of Goleta

LAW OFFICES OF
KITCHEN AND TURPIN

ERIC C. KITCHEN
A PROFESSIONAL LAW CORPORATION

DAVID C. TURPIN

SHARON C. KENNEDY

THE BALBOA BUILDING
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TELEPHONE 805-965-3079
FAX 805-966-0839

ekitchen@kitchenturpin.com
skennedy@kitchenturpin.com
rtyers@kitchenturpin.com

Via Facsimile and U.S. Mail

September 19, 2001

Ms. Laurie Weir
Executive Director
California Debt Limitation Allocation Committee
915 Capital Mall, Room 311
Sacramento, CA 95814

Re: Santa Barbara County El Encanto Heights Apartment Project/Revised Environmental Assessment Unknown Economic Impact: $1,902,900.00

Dear Ms. Weir:

Concerned homeowners of Goleta Valley have retained our law offices concerning the above referenced project. Goleta Valley is the site location designated by the Santa Barbara County Board of Supervisors for the El Encanto Heights Apartment Project. The project received final approval on August 13, 2001.

We were hired initially to file a CEQA challenge to the project upon the discovery that the Negative Mitigated Declaration initially required removal of a massive dumpsite of undocumented fill. However, the final Mitigated Negative Declaration allows recompaction of the soil without any method of verifying whether the fill material is hazardous or not.

Instead of doing a CEQA filing in Court, we contacted the environmental consultant, *Custom Environmental Services*, that issued a Revised Report and Findings, copy enclosed. It is our opinion this Revised Assessment and Conclusions requires a full re-examination of the feasibility and advisability for this project and its funding through CDLAC or CHFA or any other affiliated agency which issues bonds that require Securities Disclosures of unknown hazards and unknown and large economic impacts.

Our clients appeared before the Board and informed the Board that the site had been used for a dump for many years. Neighbors have provided statements that a local highway contractor used the dump for many, possibly twenty years. The soil reports boring sites do indicate soil that is darkly colored, an indicator of hydrocarbon spillage. The neighboring property is a former service station that is undergoing remediation. The site itself may have a flume that emanates from the neighboring site or is part of the contamination from the undocumented fill.

Interestingly, the initial Mitigated Negative Declaration states that the fill is to be "**removed.**" This is stated several times. However, the final Pacific Materials Report (the soils engineer) in recommendation Number 8, which is adopted in the final Mitigated Negative Declaration, states that the fill materials and on site removed soil (if free of expansive clay and organics) ***may be replaced in loose lifts of six inches and recompacted to 90%.***

When we learned of this information, I contacted the environmental consultants who did the initial site survey, Custom Environmental Services. That consultant reported they were never provided the soils reports, held by the County for about two years, and had they known of the soil report findings of undocumented fill, they would have recommended a Phase II report, or at a minimum an engineering geologist to test for organic or hydrocarbon substances before recompaction. That is now the recommendation with a worse case scenario disclosure.

Custom Environmental Services has now issued a Revised Report and Findings, a copy of which I enclose for your records. Consistent with environmental analysis standards is a "worse case" scenario of an unknown expense related to the undocumented fill which could add an additional cost of **$1,902,900.00** to the project for removal of the contaminated fill, transport of the fill and replacement of the fill.

This is a very serious environmental issue. Importantly and materially from your perspective, this new issue is a disclosure issue as I surmise the County of Santa Barbara has not brought the revised Custom Environmental Services Revised Assessment and Conclusions to your attention.

Clearly, whatever underwriting agencies are working with CDLAC, whether CHFA or some other governmental affiliation authorized to issue bonds for funding these projects, this information is something that requires disclosure to any agency placing funds at risk to do this project, especially to the Securities and Exchange Commission and the bond issuers and underwriters.

If I can provide any additional information, please call me. In advance, I thank you for your attention to this very important matter.

Very truly yours,

KITCHEN AND TURPIN

By _____
Eric C. Kitchen

ECK/rpl
Enclosure
cc: All clients

LAW OFFICES OF
KITCHEN AND TURPIN

ERIC C. KITCHEN
A PROFESSIONAL LAW CORPORATION

DAVID C. TURPIN

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ekitchen@kitchenturpin.com
skennedy@kitchenturpin.com
rlyons@kitchenturpin.com

Via Facsimile and U.S. Mail

October 2, 2001

Mr. Steve Goggia
County Planner
Planning and Development
123 East Anapamu Street
Santa Barbara, Ca 93101

Re: El Encanto Heights Project/Request for Copy of Rincon Consultants Soils Reports

Dear Mr. Goggia:

As you know our law offices represent groups of concerned Goleta Valley Citizens who have opposed the El Encanto Heights Apartment Project.

Recently, in an article in the Valley Voice, you were quoted as saying that Rincon Consultants did a supplemental soil report that satisfied the County's concerns about the condition of the undocumented fill.

Under the *Freedom of Information Act*, I respectfully request a copy of any soil reports received by the County, (other than the two Pacific Materials Laboratory Reports and the two reports from Custom Environmental Services) including the Rincon Consultants' report referenced in the Valley Voice Article and any other geotechnical firm who investigated or opined on the condition of the soil or the identification of the non-native fill material at the site.

Until the article was published no one, including the prior consultant, Rosalie Skefich, was aware that any other soil report which identified the undocumented fill was done. Please provide the copies and send the copy cost to our law offices.

LAW OFFICES OF
KITCHEN AND TURPIN

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ekitchen@kitchenturpin.com
skennedy@kitchenturpin.com
rlyons@kitchenturpin.com

Via Facsimile and U.S. Mail

October 19, 2001

Laura Whittall-Scherfee
California Housing Finance Agency
1121 "L" Street, Suite 207
Sacramento, Ca 95814

Re: El Encanto Heights Apartments/Goleta Valley/Santa Barbara Community Housing Corporation

Dear Ms. Whittall-Scherfee :

Our law offices represent citizen groups in Goleta Valley opposed to the El Encanto Heights apartment project.

We had been retained to file a CEQA challenge to the decision of the Santa Barbara Board of Supervisors approval of the El Encanto Heights project, which passed on August 13, 2001.

Since then, we have investigated the soils conditions of the site and revealed that undocumented fill rests in the site to be developed. When the original environmental consultant was told of the existence of the two Soils reports that revealed the undocumented fill, she said that she had been misled and changed her conclusions by issuing a revised report dated September 11, 2001. Further, she has indicated that had she known of the undocumented fill she would most certainly have ordered a Phase II to determine the content of the undocumented fill.

In this September 11, 2001 response to the two withheld Soils Reports of Pacific Materials Laboratory, the original environmental consultants, Custom Environmental Services issued a Revised Report and Recommendations to have a geologic engineer on the site during excavation to determine if any hazardous waste is included within the site. I enclose that Revised report and Recommendations for your file.

Apparently, the Santa Barbara Community Housing Corporation, (SBCHC) as purchaser and developer, in response to the Revised Recommendations made by the original consultant, Custom Environmental Services, requested Rincon Consultants to issue a report by letter, dated October 16, 2001 concerning the undocumented fill material.

Initially, the County and the SBCHC refused to provide or issue to us a copy of the Rincon Report or the letter of October 16, 2001. However, with persistence we now have a copy and offer these criticisms of the Reports and letter of October 16, 2001.

In the Rincon Phase I ESA and in the October 16, 2001 letter, Rincon under "Scope of Services" and "User Provided information," Rincon does not indicate it was provided any soils reports for its review. Two soils reports were done, in 1999 and in 2000. Why were these reports not given to Rincon Consultants?

Under the "Limitations" section of the Report, Rincon does not reveal that SBCHC has already purchased the property. In fact, the report indicates they are operating on an assumption that SBCHC has not yet purchased the property.

The ESA Report generated by Rincon does not say it has made any contact with past owners or current owners for interviews, which is a requirement of all Phase I ESA reports. Interestingly, the only contact was made just recently with John O'Shaugnessy and he did mention that the fill was from a subdivision west of the property. He does not indicate nor does the report indicate that the question was asked if any of the fill materials contained hazardous substances.

The only first hand "source" report of what constitutes the fill, is the statement from Susan Boehlje, the current Executive Director of the SBCHC. However, Susan Boehlje could not possibly have any first hand knowledge of the source of the undocumented fill. It is extremely unprofessional for an environmental consultant to rely upon a person who professes to have first hand knowledge when in fact she does not. The October 16, 2001 report quotes Ms. Boehlje as stating "... the fill dirt brought onsite was clean fill from a known source." Ms. Boehlje could not possibly have any first hand knowledge of this. It is outlandish and irresponsible for her to make this statement and for Rincon to rely upon it in concluding there is no hazardous materials on site.

Finally, it is clear that Ms. Boehlje provided only the first and not the second Rincon Soils Report to Rincon Associates. Why was the second report withheld?

There is no way that this property can be "cleared" for development without a Phase II report to discern the actual content of the undocumented fill.

The conclusions of Rincon are speculation at best. It is not scientific to conclude without actual testing, or at a minimum to have a site engineer during excavation determine the presence of hazardous materials before it is permanently placed into the ground as a future contaminate of the areas and its ground water resources.

We strongly protest this development as a government "cram" that is riddled with reports to justify what has already been decided as opposed to actually finding out the content of the undocumented fill before it is placed permanently into the ground and adversely affect the citizens' health and clean ground water.

Very truly yours,

KITCHEN AND TURPIN

By _____/s/_____
Eric C. Kitchen

ECK/kce

cc: Ms. Laurie Weir
Executive Director
California Debt Limitation Allocation Committee

CES — Environmental Compliance Consulting

CUSTOM ENVIRONMENTAL SERVICES

September 11, 2001

Steve Goggia, Planner
Santa Barbara County Planning Department
123 East Anapamu Street
Santa Barbara, CA 93101
805-568-2067 voice

re: Revised Environmental Assessment Conclusions
Proposed El Encanto Apartment Project
APN 077-490-039
7388 Calle Real
Goleta, California

Dear Mr. Goggia:

INTRODUCTION / BACKGROUND

Custom Environmental Services (CES) was retained by the subject Project applicant, Ms. Susan Boehlje, Executive Director of the Santa Barbara Community Housing Corporation (SBCHC) to conduct an environmental site assessment. Because the property was vacant of structures, I believed that a Transaction Screen Assessment (TSA) was appropriate to satisfy prospective buyer "due diligence". A proposal for a TSA was submitted on August 27, 1999 and accepted by SBCHC. The "yes / no" checklist, required for the TSA, and optional addendum (narrative) dated September 2, 1999 was submitted to SBCHC as a final report. The report was submitted on an expedited schedule to meet escrow conditions. SBCHC has not contacted CES since that time.

The purpose of an environmental Phase I Site Assessment or Transaction Screen Assessment is to identify "potential" environmental conditions that may result in a hazardous substance liability for the property or property owner. The assessment is an administrative investigation process conducted according to an industry standard that includes reviewing a minimum number of required resources. One of the reasons for conducting environmental site assessments during the escrow period of a property ownership transaction is so that if an environmental condition is identified, then responsibility for addressing and remediating the environmental condition can be assigned to either the existing property owner or new buyer.

Based on the findings of the Phase I Site Assessment, the consultant may recommend a Phase II Site Assessment (i.e., subsurface investigation). The Phase II process may include a geophysical survey, soil borings, and/or groundwater monitoring wells to confirm the presence of an environmental condition. In the original CES environmental TSA report, no additional

environmental site investigations (e.g., Phase II Site Assessments) were recommended based on the information collected up to that point in time.

Recently (August 30, 2001), I became aware that a soils report was conducted for the subject property. A soils report is typically requested before initiating any environmental site assessment, but, in this case, I was informed that one was not yet available. I have since reviewed an August 11, 2000 version of the Pacific Materials Laboratory report that documents findings from an August 19 and August 20, 1999 soil boring investigation. Based on the findings in the soils report— specifically the fact that the site has varying depths of artificial fill—I believe that I am obligated to modify the conclusions and recommendations in my original environmental TSA report.

DISCUSSION

Because of the specific wording of Question 7 of the TSA questionnaire (i.e., "fill dirt...that originated from a contaminated site"), none of the question responses require modification. However, the fact that fill dirt of unknown origin was placed on the subject property would have been presented in the TSA addendum report conclusions. A recommendation for an environmental geologist to monitor the site grading activities would have been included. As a courtesy to the prospective property buyer, an estimate of the quantity of potentially impacted soil (worse-case scenario) and the estimated costs for transportation and disposal offsite as Class II landfill material would have been presented. If SBCHC had contacted CES with this new information, then we would have made our conclusions before the property ownership transaction was complete (February 1,2001) and responsibilities for environmental liability could have been assigned between the seller and the buyer.

CONCLUSIONS AND RECOMMENDATIONS

Since the property was purchased by SBCHC and the proposed project has been approved by the County Board of Supervisors, the main purpose of this environmental TSA report modification is to underscore the recommendations in the August 11, 2000 soils report and to present conclusions and recommendations that CES would have presented in our TSA had we known about the soils report findings.

(1) As specified in the Pacific Materials Laboratory report, all non-native soil at varying depths across the property up to 20 feet, must be removed (excavated), inspected, and assessed for compaction suitability before recompaction to at least 90%.

(2) An environmental geologist should be present during the site grading activities to assist in the determination of "suitable soil" for compaction. The environmental geologist will also identify needs for environmental monitoring (e.g., organic vapor field screening instruments)

and instruct the owner as to their CERCLA obligations for reporting a known hazardous materials release, if encountered.

(3) If hazardous materials are encountered and none of the soil can be re-compacted, a significant economic impact will be realized by the property owner as a potential for over 30,289 cubic yards (0.94 acres x 43,500 square feet x 20 feet) may be classified as Class I (hazardous waste) or Class II landfill material. Typical transportation and disposal costs for these materials to McKittrick Landfill in Bakersfield are approximately $50 per cubic yard, or a potential financial liability of over $1,514, 450. In addition, if import fill cannot be located in time to meet project schedule, then it must be purchased from Santa Barbara Sand for approximately $388,450.

Thank you for the opportunity to present these revised conclusions and recommendations based on newly discovered information.

Respectfully submitted,
CUSTOM ENVIRONMENTAL SERVICES

Rosalie A. Skefich

Rosalie A. Skefich
President / Chemical Engineer

cc: Susan Boehlje, Executive Director
Santa Barbara Community Housing Corporation

George Bisol
Santa Barbara County, Planning & Development, Grading Department

Peter Harmer
Santa Barbara County, Planning & Development, Grading Department

From: Steve Goggia
To: MAURICE, PAT
Date: 8/1/00 3:38PM
Subject: Re: El Encanto Apartamientos

how's this

>>> PAT MAURICE 07/24/00 09:51AM >>>
Steve,
I'm just returning from 2 weeks vacation and hope to get comments to you by August 1.

thanks,

P

>>> Steve Goggia 07/14/00 10:23AM >>>
I have not seen any revisions to the traffic study since this memo was sent. I do believe we've covered the right-of-way space for future expansions issues as best we can for now. I don't believe believe Pat's comments have been addressed.

I'm also reprinting an email sent to Bret, Pat and Jeff on 4/24. Jeff responded with a simple "yes". Bret and Pat had not responded. The Application will be called complete for processing very soon. I want assurances that the traffic report is acceptable. Please reply August 1st. thanks

4/24
Now that the Miramar smoke has cleared, I'm looking at the El Encanto Project. I have some questions regarding the traffic study (P&S 4/14/2000).

We know traffic on Calle Real exceeds the LOS C capacity. During the Mountain View Ranch fiasco, Bret had to testify that although the numbers were much higher, the roadway was still operating at acceptable levels. Can I assume this is still correct, and will only get better when Cathedral Oaks Segment 3 is opened?

I am confused by the Table on page 8: The Storke/Glen Annie/ Hollister intersection is going to operate at los F. How come there is no change to the PM Peak Hour with the 15-year forecast plus project (but there is with the existing plus project)? Is it because build out under the Community Plan sends so many trips to the intersection, the project trips are just a "drop in the bucket"?

Should we insert change in the V/C ratio is less than 0.01 and no CEQA Thresholds... to the last sentence in the top paragraph on page 9?

I want to be sure the project does not cause a significant CEQA impact.

What else do we need to do to get a bus stop on Calle Real in front of the project?

Are you satisfied with the findings of the report?

>>> PAT MAURICE 05/16/00 01:55PM >>>
Steve,
Enclosed are my comment for this project. what have you heard from the neighbors?

P

From: PAT MAURICE
To: Gabriel, Chris
Date: Tue, May 16, 2000 3:15 PM
Subject: el Encanto Apartamientos

Chris,
I understand the neighbors are concerned about collisions in front of the 7-11 between the proejct and Ellwood Station Road. All the more reason to do a collision history to have in your back pocket for the hearing.

Since the roadway is operating over capacity so you'll probably want to have that covered as well.

I also recommend having a diagram on hand to show the built-out section of Calle Real so that the PC is comfortable with the ROW we're giving away.

thanks,

P

CC: Goggia, Steve

Comments on Draft Mitigated Negative Declaration (DMND) for Citrus Village.
Case No. 04-226-GP;-DP,-RN07-MND-004
Submitted by Ingeborg E. Cox, MD

According to an article in the SB News Press dated August 28, 2002 Fire Capt. Maynard Yeaw stated that he won't approve any more plans with private streets narrower than 36 feet.
On page 9, of the DMND for Citrus Village, it states: "a 24 foot wide drive aisle would terminate in a hammerhead turnaround for emergency vehicles in front of building E".
If there is not enough parking, and also no enforcement, people will park their cars outside of designated areas.

When the street is narrowed because of the parked cars, what will happen during a medical emergency? You normally have police, fire and paramedics responding. How are they going to function in such a confined space?
The design of this Village is not considering this.
Think about the following scenario: Cardiac arrest in Building E.

There should be absolutely no modification of parking spaces. People that live there and do not have a parking space will go to the street. I invite you to come to Winchester Canyon Rd. to see the effects of not enough parking. Many vehicles, including trucks and RV's, are parked on the street. Street cleaning is impossible. Consequently, oil leaking is being washed into the storm drains ending into the ocean.

The design of this project does not integrate it into the existing neighborhood.
The project walls itself in.
The city of Goleta should integrate new housing into the existing neighborhood, not isolate them with walls from the existing neighborhoods.
You have an 8 'high retaining wall topped with a 3' high chain link fence. Why eleven feet of wall on the northern property boundary?

On page 39 it is mentioned that the California Air Resources Board (CARB) has developed guidelines designed to minimize sensitive receptor exposure. And it states that the guidelines mention **500 foot** setback from any freeway.

Medical studies mention a **500 meter** (~**1500 feet**) from a mayor roadway- which in this case is Highway 101- as the distance that has been studied. The major components of automotive pollution are: sulfur dioxide, carbon monoxide, nitrogen oxides, volatile organic compounds (benzene, ethylene) and polycyclic aromatic hydrocarbons. **Children** living **within 500 meters** of a freeway have substantial deficits in lung function and lung development.
Enclosed are articles from the medical literature addressing the effects of traffic exposure:

1) "Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study". The Lancet 2007
2) SB County APCD "On the Air" "Living near freeways harms children's lungs"

3) "Traffic related Air Pollution near busy roads The East Bay Children's Respiratory Health Study ". American Journal of Respiratory and Critical Care Medicine 2004
4) Cardiovascular implications of exposure to traffic air pollution during exercise QJM 2004

The residents will be exposed to polluted air from the freeway as well as the gasoline station. Also vehicles idling, or starting their engines at the 7- Eleven Store will contribute to more air pollution.

On page 88 it states that site specific traffic analysis were not prepared for the proposed project. The traffic has changed in the area. This development needs to have a current traffic analysis and not one dating back to March of 2005.
Also traffic analysis should be done at times when school is in session to get an actual traffic count.

We know that the nearby Glenn Annie intersection is already overburdened and most likely the existing LOS will go down to a level E or F.

Noise:
Page. 75 states results showed that future exterior noise levels at building sites will range from approx. 69dBA (Unit 3, Building B; Unit 7, Building D) to 74 dBA (Unit 1, Building A and Unit 5, Building C).

This proposal was the subject of a noise study done in 2001. It appears that no recent noise study was done. With the increase in traffic the city should demand a new noise study.
The noise caused by Union Pacific trains can be specifically distressing at night and should be taken into consideration.

Traffic noise is considered an environmental health problem.
There is an association between traffic noise and hypertension, sleep disturbances, cardiovascular risk if daytime levels exceed 65dB.
Children attending kindergarten where the traffic noise > 60dB have higher blood pressure.

This project needs to provide more data. What is the level of noise that the residents are going to be exposed to when Union Pacific trains pass at midnight? What is the noise level now from the freeway, as traffic has increased. Up to date data is needed.

Where are children going to play? The exterior noise is listed between 69 and 74dBA. This does not appear to be a site for children.

The residential units located to the North are going to be exposed to noise levels at 95dB This is not acceptable.

Fire:
There is at present inadequate fire protection. Pg. 82 lists fire station 11 as the primary station serving the project site. The ratio of service for Station 11 is **7,198 people**, or nearly **twice** the **acceptable maximum level of 4,000 people** per station.
This puts residents in jeopardy should there be a large fire as there is no adequate coverage.

The city of Goleta should demand adequate fire protection for its residents.

The proposed project should contribute money for a new fire station and the project must be postponed until the fire station has been constructed and is operable.

8/28/02

Fire chief: Goleta streets not safe

Lanes too narrow

By MORGAN GREEN
NEWS-PRESS STAFF WRITER
e-mail: mgreen@newspress.com

Fire Capt. Maynard Yeaw can't escape the image of firefighters unable to save a life because a car parked on a narrow street blocks their engine.

He's the man in charge of seeing that all plans for new subdivisions allow for County Fire Department engines to reach fires and medical emergencies.

Citing hundreds of new homes, condos and apartments built recently on substandard narrow private streets in the county, he says: "It's time to draw the line in the sand."

Capt. Yeaw says he won't approve any more plans with private streets narrower than 36 feet. That width leaves room enough for parked cars along both curbs and 20 feet for fire engines to pass, the requirement under the California Fire Code.

The unincorporated county and other jurisdictions with county fire service, such

Please see **STREETS** *on A8*

Fire Department will OK nothing less than standard

■ **STREETS**
Continued from Page A1

as the city of Goleta, have final say on housing proposals. "But we're going to oppose anything less" than standard streets.

The policy shift is a big change.

For the past several years, the Fire Department has regularly signed off plans showing private streets so narrow that parking must be banned on one or both sides to allow emergency vehicles to pass. Some streets are as narrow as 20 feet.

Small streets, Capt. Yeaw said, are an easy way for developers to make room on their sites for more units. And in the midst of the local housing crisis, planning commissions "have been under enormous political pressure to put in more housing."

A routine county practice has been to permit substandard streets so long as other fire safety precautions are taken, such as fitting the housing with internal sprinkler systems.

But the majority of calls are medical, Capt. Yeaw said.

"A sprinkler system doesn't do you any good if you're having a heart attack," he said.

Senior Fire Department officials said they do not recall a specific instance when property or life was lost because an engine couldn't negotiate one of the private substandard streets. However, they said, it could happen at any time.

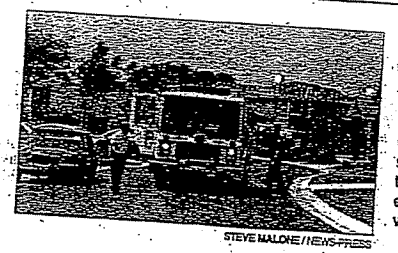
The problem is that obstructive illegal parking along the narrow streets is common, Capt. Yeaw said. Other obstructions crop up as well, such as construction materials and garbage and recycling bins.

"We see the same result time and time again," Capt. Yeaw said. "It's a reasonable expectation that's what you'd continue to see."

The Fire Department has the authority to cite and tow, but does not have the resources to do so, he said.

In the city of Goleta, where land is costly and housing demand is high, builders have erected several subdivisions, from luxury homes to low-income affordables, with substandard private streets.

"The council has expressed concerns," said Rick Gomez, interim city planning manager. Policies that allow the practice are under review by city engineers and planners, with an eye toward changes.



STEVE MALONE / NEWS-PRESS

County Fire Capt. Maynard Yeaw, left, says roads such as this one in the Orchard Park subdivision are too narrow for emergency vehicles.

But, substandard streets have also cropped up in the North County, where land is relatively plentiful and inexpensive.

Out jogging earlier this year in Orcutt near his home, Fire Department Operations Division Chief Jim Harrison ran into a new project on Ajay Street where a half dozen large homes were clustered at the dead-end of a substandard lane.

"That should never have been allowed," he said. The street was so jammed with illegally parked vehicles it made it nearly impossible for a pedestrian to pass by.

"I sent somebody to talk to those people three different times," he said.

The County Planning Department is in charge of processing subdivision projects. County Planning Director John Patton could not be reached for comment Tuesday.

But Second District Supervisor Susan Rose said she is delighted the Fire Department is addressing the issue.

Requiring standard streets does not preclude more housing that is affordable. It just means public safety shouldn't be sacrificed, she said.

At least one developer, Bill Ehrlich, said mandatory 36-foot streets will not crimp plans by the Larwin Development Company for between 1,100 to 1,500 units of housing units on 262 acres of Bishop Ranch in Goleta.

He said infill projects on small sites might have problems. But with such a large piece of land as Bishop Ranch, he can vary the housing types and cluster homes around driveways and still build through streets of standard size.

Effect of exposure to traffic on lung development from 10 to 18 years of age: a cohort study

W James Gauderman, Hita Vora, Rob McConnell, Kiros Berhane, Frank Gilliland, Duncan Thomas, Fred Lurmann, Edward Avol, Nino Kunzli, Michael Jerrett, John Peters

Summary

Background Whether local exposure to major roadways adversely affects lung-function growth during the period of rapid lung development that takes place between 10 and 18 years of age is unknown. This study investigated the association between residential exposure to traffic and 8-year lung-function growth.

Methods In this prospective study, 3677 children (mean age 10 years [SD 0·44]) participated from 12 southern California communities that represent a wide range in regional air quality. Children were followed up for 8 years, with yearly lung-function measurements recorded. For each child, we identified several indicators of residential exposure to traffic from large roads. Regression analysis was used to establish whether 8-year growth in lung function was associated with local traffic exposure, and whether local traffic effects were independent of regional air quality.

Findings Children who lived within 500 m of a freeway (motorway) had substantial deficits in 8-year growth of forced expiratory volume in 1 s (FEV_1, −81 mL, p=0·01 [95% CI −143 to −18]) and maximum midexpiratory flow rate (MMEF, −127 mL/s, p=0·03 [−243 to −11]), compared with children who lived at least 1500 m from a freeway. Joint models showed that both local exposure to freeways and regional air pollution had detrimental, and independent, effects on lung-function growth. Pronounced deficits in attained lung function at age 18 years were recorded for those living within 500 m of a freeway, with mean percent-predicted 97·0% for FEV_1 (p=0·013, relative to >1500 m [95% CI 94·6–99·4]) and 93·4% for MMEF (p=0·006 [95% CI 89·1–97·7]).

Interpretation Local exposure to traffic on a freeway has adverse effects on children's lung development, which are independent of regional air quality, and which could result in important deficits in attained lung function in later life.

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See Comment page 535

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Introduction

Both cross-sectional[1–5] and longitudinal[10–15] studies have shown that lung function in children is adversely affected by exposure to urban, regional air pollution. Evidence has emerged that local exposure to traffic is related to adverse respiratory effects in children, including increased rates of asthma and other respiratory diseases.[16–26] Cross-sectional studies in Europe have shown that deficits in lung function are related to residential exposure to traffic.[27,28–32] However, does traffic exposure have an adverse effect on lung-function development in children? The answer to this question is important in view of the extent of traffic exposure in urban environments and the established relation between diminished lung function in adulthood and morbidity and mortality.[33–35]

We investigated the association between residential exposure to traffic and 8-year lung-function development on the basis of cohort data from the Children's Health Study. We also studied the joint effects of local traffic exposure and regional air quality on children's lung development.

Methods

Participants

The Children's Health Study recruited two cohorts of fourth-grade children (mean age 10 years [SD 0·44], one in 1993 (cohort 1, n=1718) and the other in 1996 (cohort 2, n=1959). All children were recruited from schools in 12 southern California communities as part of an investigation into the long-term effects of air pollution on children's respiratory health.[7,34,40] A consistent protocol was used in all communities to identify schools, and all students targeted for study were invited to participate.[40] Overall, 82% (3677) of available students agreed to participate. Pulmonary-function data were obtained yearly by trained field technicians, who travelled to study schools to undertake maximum effort spirometry on the children, using the same equipment and testing protocol throughout the study period. Details of the testing protocol have been previously reported.[7,15] Children in both cohorts were followed up for 8 years.

A baseline questionnaire, completed at study entry by each child's parent or legal guardian, was used to obtain information on race, Hispanic ethnic origin, parental income and education, history of doctor-diagnosed asthma, in-utero exposure to maternal smoking, and household exposure to gas stoves, pets, and environmental tobacco smoke.[40] A yearly questionnaire, with similar structure to that of the baseline questionnaire, was used to update information on asthma status, personal smoking, and exposure to environmental tobacco smoke. For statistical modelling, a three-category socioeconomic status variable was created on the basis of total household income and education of the parent or guardian who completed the questionnaire. High socioeconomic status (23% of children, n=823) was defined as a parental

	Regional pollutant effect*	p	Local freeway distance (m)						
			<500	p	500–1000	p	1000–1500	p	p for interaction†
1000–1800 h ozone	−13	0·821	−81	0·012	−41	0·165	−33	0·275	0·51
Nitrogen dioxide	−109	0·003	−80	0·012	−41	0·166	−33	0·279	0·81
Acid	−111	0·002	−80	0·013	−41	0·164	−33	0·285	0·54
PM_{10}	−111	0·013	−81	0·012	−42	0·158	−32	0·287	0·24
$PM_{2·5}$	−100	0·009	−80	0·012	−41	0·160	−33	0·285	0·40
Elemental carbon	−101	0·001	−80	0·012	−42	0·156	−33	0·282	0·63

*Pollutant effects are the difference in 8-year FEV_1 growth from lowest to highest observed community-average concentration of the pollutant, specifically: per increase of 37·5 ppb ozone (1000–1800 h), 34·6 ppb of nitrogen dioxide, 9·6 ppb of acid vapour, 51·4 μg/m³ of PM_{10}, 22·8 μg/m³ of $PM_{2·5}$, and 1·2 μg/m³ elemental carbon. Distance effects are the difference in 8-year growth relative to those living >1500 m from a freeway. †A test of whether freeway-distance effect is modified by regional concentration of the pollutant. PM_{10}=particulate matter <10 μm aerodynamic diameter. $PM_{2·5}$=particulate matter <2·5 μm aerodynamic diameter.

Table 3: Joint effect of regional pollution and local distance to a freeway on 8-year FEV_1 growth

Reduced lung-function growth was independently associated with both freeway distance and with regional air pollution (table 3). Statistically significant joint models of regional pollution with distance to freeway were seen for nitrogen dioxide, acid vapour, elemental carbon, and particulate matter with aerodynamic diameter less than 10 μm and less than 2·5 μm. Ozone was not associated with reduced lung-function growth. There was no significant evidence of effect modification (interaction) of local traffic effects with any of the regional pollutants.

A subset of 1445 children were observed over the full 8 years of the study, from age 10 to 18 years. In this group, we noted significant deficits in 8-year FEV_1 growth and MMEF growth for those who lived within 500 m of a freeway (table 4). At 10 years of age, there was some evidence of reduced lung function for those who lived closer to a freeway than those who did not, although none of the differences between distance categories was statistically significant. However, by 18 years of age, participants who lived closest to a freeway had substantially lower attained FEV_1 and MMEF than those who lived at least 1500 m from a freeway.

These deficits in average FEV_1 and MMEF translated into pronounced deficits in percent-predicted lung function at 18 years of age (figure). There was a trend of lower percent-predicted lung function for children who lived closer to a freeway than for those who lived further away. The effect was most pronounced for those who lived less than 500 m from a freeway, with average percent predicted values of 97·0% (95% CI 94·6–99·4) for FEV_1 (p=0·013 relative to >1500 m) and 93·4% (89·1–97·7) for MMEF (p=0·006).

		Lung function		8-year growth
		Age 10 years	Age 18 years	Difference* (95% CI)
		Difference* (95% CI)	Difference* (95% CI)	
FVC	Freeway distance			
	<500 m	−17 (−70 to 37)	−85 (−192 to 22)	−69 (−160 to 22)
	500–1000 m	−12 (−61 to 37)	−54 (−151 to 43)	−42 (−125 to 41)
	1000–1500 m	−30 (−80 to 21)	−81 (−181 to 19)	−52 (−137 to 33)
FEV_1	Freeway distance			
	<500 m	−23 (−73 to 28)	−121 (−219 to −23)	−98 (−182 to −15)
	500–1000 m	−32 (−78 to 14)	−93 (−183 to −4)	−61 (−137 to 15)
	1000–1500 m	−34 (−81 to 14)	−78 (−170 to 14)	−44 (−122 to 34)
MMEF	Freeway distance			
	<500 m	−57 (−169 to 56)	−230 (−432 to −28)	−173 (−327 to −19)
	500–1000 m	−92 (−195 to 10)	−105 (−289 to 79)	−12 (−152 to 128)
	1000–1500 m	−45 (−150 to 60)	−151 (−340 to 38)	−106 (−250 to 38)

*Difference in 8-year lung function or growth relative to children living >1500 m from a freeway.

Table 4: Cumulative effect of residential distance in the 1445 children with full 8-year follow-up

Discussion

This study shows that residential proximity to freeway traffic is associated with substantial deficits in lung-function development in children. 8-year increases in both FEV_1 and MMEF were smaller for children who lived within 500 m of a freeway, than for those who lived at least 1500 m from a freeway. Freeway effects were seen in subsets of non-asthmatic and non-smoking participants, which is an indication that traffic exposure has adverse effects on otherwise healthy children. Deficits in 8-year growth resulted in lower attained FEV_1 and MMEF at 18 years of age for participants who lived within 500 m of a freeway than for those who lived further away. Since lung development is nearly complete by age 18 years, an individual with a deficit at this time will probably continue to have less than healthy lung function for the remainder of his or her life.

We previously reported an association between community-average pollutant concentrations and 8-year lung-function growth.[15] That result relied on comparisons in communities that had different concentrations of regional air pollution, and implicated many pollutants such as nitrogen dioxide, acid vapour, particulate matter with aerodynamic diameter less than 10 μm and 2·5 μm, and elemental carbon. Our present study builds on that result, and shows that in addition to regional pollution, local exposure to large roadways is associated with diminished lung-function development

in children. We did not find any evidence that traffic effects varied depending on background air quality, which suggests that even in an area with low regional pollution, children living near a major roadway are at increased risk of health effects. Our results also suggest that children who live close to a freeway in a high pollution area experience a combination of adverse developmental effects because of both local and regional pollution.

We noted a larger freeway effect in boys than in girls, although the difference between sexes was not significant. By contrast, a cross-sectional European study[29] reported larger traffic effects on lung function in girls than in boys.[29] Several factors could explain this discrepancy in sex-specific effects between studies, from differences in specific air pollution mixtures and underlying population susceptibilities, to the general difficulty of comparisons between longitudinal and cross-sectional study effect estimates. In general, however, both studies show that lung function in children is adversely affected by exposure to traffic.

The concentrations of several pollutants are raised near major freeways. Daytime concentrations of black carbon, ultrafine particulate, and other exhaust pollutants have been reported to be high, but decline exponentially, within 500 m of a freeway,[44,45] although night-time concentrations of ultrafine particulate remain above background concentrations for distances greater than 500 m from a freeway.[45] Some studies have reported increased traffic pollution, particularly nitrogen dioxide, at distances over 1000 m from a freeway.[15,47–49] Elemental carbon, an indicator of pollution from diesel exhaust, varies with nearby high-traffic roads[47,50,51] but can also be transported across large distances.[52] Diesel exhaust is one of the primary contributors to particulate-matter concentrations in those communities most affected by traffic.[53] A pollutant such as elemental carbon could explain our reported health effects both locally and regionally.

Both regional ambient and ultrafine particulate matter present in high concentration in close proximity to roadways can elicit oxidative and nitrosative stress in the airways, which results in inflammation.[54,55] Kulkarni and co-workers[32] reported that traffic-related particulate matter was correlated with the amount of carbon in the airway macrophages of children, which in turn was associated with reductions in FEV_1, MMEF, and FVC. Chronic airway inflammation could produce our reported deficits in MMEF and FEV_1. Additional research is needed to identify the specific traffic pollutants that bring about health effects, and to elucidate the contribution of each pollutant to regional and local associations.

A strength of this study was the long-term, prospective follow-up of two large cohorts of children, with exposure and outcome data obtained consistently. However, as in any epidemiological study, our results could be confounded by one or more other factors related to both traffic and lung-function growth. Our results were robust

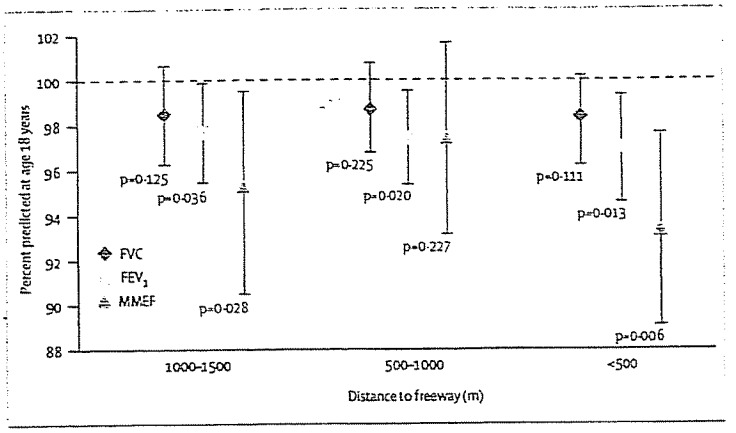

Figure: Percent-predicted lung function at age 18 years versus residential distance from a freeway
The horizontal line at 100% corresponds to the referent group, children living >1500 m from a freeway.

to adjustment for several factors, including socioeconomic status and indoor sources of air pollution, but the possibility of confounding by other factors still exists. Throughout the 8-year follow-up, we noted around an 11% loss of study participants per year. Participant attrition is a potential source of bias in cohort studies. We analysed the subset of children who were followed up for the full 8-year duration of the study and also noted significant traffic-effect estimates, which make participant loss an unlikely explanation for our results. We did not note a significant association between growth and model-based pollution from a freeway, despite large estimated deficits in the highest-exposure quartiles (table 1). However, we were restricted in detection of an association with model-based pollution from freeways because there was little variation in this measure within most of our study communities (webtable 2).

We have shown that residential distance from a freeway is associated with significant deficits in 8-year respiratory growth, which result in important deficits in lung function at age 18 years. This study adds to evidence that the present regulatory emphasis on regional air quality might need to be modified to include consideration of local variation in air pollution. In many urban areas, population growth is forcing the construction of housing tracts and schools near to busy roadways, with the result that many children live and attend school in close proximity to major sources of air pollution. In view of the magnitude of the reported effects and the importance of lung function as a determinant of adult morbidity and mortality, reduction of exposure to traffic-related air pollutants could lead to substantial public-health benefits.

Contributors

W J Gauderman, R McConnell, F Gilliland, E Avol, J Peters, M Jerrett, and N Kunzil participated in the writing of the manuscript. W J Gauderman, H Vora, K Berhane, D Thomas, and F Lurmann participated in the analysis of the data. All named authors took part in the interpretation of results, and approved the final version of the manuscript.

+ 500 meters = 1625 feet.

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Santa Barbara County
Air Pollution Control District

On the Air

Living Near Freeways Harms Children's Lungs

Children who live near busy roads experience slower lung development than children who don't—whether they live in Los Angeles, Santa Maria or Santa Barbara. This is one of the key findings from the Children's Health Study, a 15-year University of Southern California (USC) family of studies of more than 11,000 children from sixteen communities.

Researchers found that children who live within 500 meters, or approximately a third of a mile, of a freeway or busy roadway have substantial deficits in lung function and lung development when compared with children living 1,500 meters, or about a mile away from the roadways.

"The interesting thing about this is that the relationship between busy roadways and slower lung growth holds true regardless of the air quality of the surrounding area, whether it's a community like Los Angeles or Riverside with higher pollution levels, or one like Santa Barbara or Santa Maria where the air is relatively much cleaner," notes Ed Avol, Professor at the Keck School of Medicine at USC. He explains that children who both live near freeways and live in a high-pollution community have a double challenge, because they are affected by both the regional and local pollution.

He comments, "When you think about just how many children in Southern California live near freeways, this is a pretty significant finding." In Santa Barbara and Santa Maria, impacts were primarily related to living near freeways such as Highway 101, he explains, as these communities do not have many high-traffic four-lane roads considered busy roadways.

The Children's Health Study began in 1993 to assess the impacts of air pollution on the growth and development of kids' lungs. Children—primarily fourth graders—were recruited from schools in twelve communities from San Diego to San Luis Obispo counties, ranging from Southern California locations with more polluted air, to Santa Maria and Lompoc. Communities in Santa Barbara and San Luis Obispo counties were selected for their cleaner air, compared to Los Angeles area counties.

The children were studied each year as their lungs developed until high school graduation, and results were compared to air quality measurements in the communities. The research team collected extensive information about each child's home, socioeconomic status and other factors that might affect health.

Then the team visited the schools and measured the children's lungs, assessing how much air could be expelled in one breath and how quickly it could be expelled.

Early findings from the studies showed that the lungs of children in more polluted areas were growing more slowly than the lungs of children in cleaner-air communities. Higher levels of ozone, a key component of smog, were associated with more school absences and more cases of newly diagnosed asthma. The study also showed that when children moved from more polluted to less polluted areas, their lungs began to grow at rates similar to other children in that cleaner community, although they seemed to remain permanently affected by the period of slower growth.

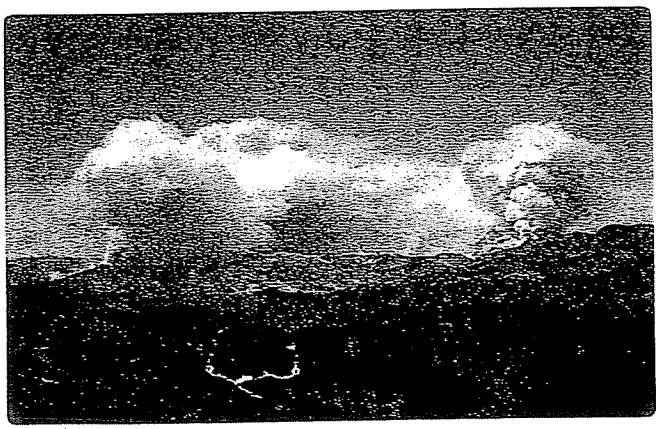
(continued on page two)

Wildfire Smoke

In July and August, APCD and the County Public Health Department issued advisories calling on residents to stay indoors and avoid outdoor exercise when smoke from the Zaca Fire was present in the air. The Zaca Fire started July 4, and burned over 240,000 acres, prompting evacuation orders, and creating ash and plumes of smoke.

APCD Public Information Officer Bobbie Bratz remarked, "The

(continued on page two)



Smoke plumes from the Zaca Fire

Inside...

Children's Lungs (cont'd)

As the studies continued from 2003 to 2007, some of the participating communities have changed—Santa Barbara was added to the study, replacing Lompoc—and researchers began to look at additional issues. They found that children who lived within 500 yards of a freeway had a three percent deficit in how much air they could exhale, and a seven percent deficit in how fast it could be exhaled, compared with children who lived at least 1,500 yards, or nearly a mile, from a freeway—regardless of the air pollution levels in the surrounding community. In addition, with every 20 parts per billion increase in the level of ozone, illness-related school absences increased over 60%—wherever the school was located. To date, 989 children in Santa Maria and 470 children in Santa Barbara have been part of the study.

A University of California at Los Angeles (UCLA) study of ultra-fine particles along freeways found that the airborne levels of these very tiny particles drop off sharply in the first 100 meters from the road. Says Avol: "Our health findings are more gradual. We see the most effects within the first 400 to 500 meters from a freeway, and it goes down gradually after that." One possible explanation for this difference could be that the UCLA measurements were made at a consistent time of day (early afternoon) when the winds were well-established and from a certain direction. As winds shift throughout the day and pollution moves around, the sharp gradients in pollution seen in the UCLA study smooth out and extend out a few 100 meters, more in line with the health signal reported.

As the Children's Health Study results have been published, the authors have been contacted by everyone from school district officials to home buyers with questions about buildings and homes that are close to freeways. Answers are not always possible, notes Avol: "Is it better for a kid to go to school near a freeway, but not to have to spend three to four hours a day on a school bus? Or, people will ask, if they're 800 meters from the freeway, can they go ahead and buy the house? We have to tell them that there's no absolute threshold. And a lot depends on the specific conditions."

He adds, "Many schools are located near freeways, because that's where the land was cheap enough for school districts to build. While we can't do anything about the decisions of the past, we are going to need to think about the implications of these health results for land use decisions in the future."

Researchers have not identified exactly which pollutant in vehicle exhaust is responsible for the effects, or whether road dust, including microscopic particles from tires suspended in the air, plays a role.

Researchers will look more closely at these and other issues as studies continue through 2012. Avol explains that Santa Barbara will play an important role in the next phase of the study, because it has a wide range of variability in traffic density, topography, house distance to the freeway, and other variables of possible interest and health relevance.

Another area of focus as the study continues will be the role of genes in making particular children more susceptible to the effects of air pollution. Researchers will also continue to follow children from the original study phase as they grow older. Says Avol, "We know that reduced lung function can cause health problems later in life. The length of this study allows us to assess the health impacts over time as the children from 1993 mature."

For more information see http://hydra.usc.edu/scehsc/default.asp.

Smoke (cont'd)

main point we communicate is if you smell smoke, then the particles are affecting the air you are breathing. If you see smoke, but don't smell it, then the smoke may be up high. Falling ash particles are mostly too large to stay in our lungs, although they can create dust that is a problem."

She emphasized, "If you have a heart condition, or lung problems—especially asthma—you need to be particularly careful, and call your doctor if your symptoms worsen, or if you start to have new symptoms."

Added Bratz, "We work closely with the County Public Health and Fire Departments during incidents like this one, and appreciate this partnership effort to keep the public informed about the fire and the smoke."

For more information and updates on smoke advisories, see www.OurAir.org.

Satellite photo taken August 14 (courtesy NASA) shows smoke over Santa Barbara County reaching in to the San Joaquin Valley.

Traffic-related Air Pollution near Busy Roads
The East Bay Children's Respiratory Health Study

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Recent studies, primarily in Europe, have reported associations between respiratory symptoms and residential proximity to traffic; however, few have measured traffic pollutants or provided information about local air quality. We conducted a school-based, cross-sectional study in the San Francisco Bay Area in 2001. Information on current bronchitis symptoms and asthma, home environment, and demographics was obtained by parental questionnaire (n = 1,109). Concentrations of traffic pollutants (particulate matter, black carbon, total nitrogen oxides [NO_X], and nitrogen dioxide [NO_2]) were measured at 10 school sites during several seasons. Although pollutant concentrations were relatively low, we observed differences in concentrations between schools nearby versus those more distant (or upwind) from major roads. Using a two-stage multiple-logistic regression model, we found associations between respiratory symptoms and traffic-related pollutants. Among those living at their current residence for at least 1 year, the adjusted odds ratio for asthma in relationship to an interquartile difference in NO_X was 1.07 (95% confidence interval, 1.00–1.14). Thus, we found spatial variability in traffic pollutants and associated differences in respiratory symptoms in a region with good air quality. Our findings support the hypothesis that traffic-related pollution is associated with respiratory symptoms in children.

Keywords: air pollution; asthma; bronchitis; epidemiology; vehicle emissions

Numerous epidemiologic studies have documented adverse effects of air pollution on health (1). The majority of these population-based studies have used pollutant concentrations measured at central monitoring sites to estimate exposures and have not, in general, considered local spatial variability in pollutant levels. However, motor vehicle emissions, the principal source of ambient air pollution in most urban areas, are likely to vary substantially within a given community, and researchers have begun to document differences in traffic-related pollutants on a neighborhood scale (2, 3).

Recently, a number of epidemiologic studies have reported associations between residential proximity to busy roads and a variety of adverse respiratory health outcomes in children, including respiratory symptoms, asthma exacerbations, and decrements in lung function (4–12). In some reports, truck traffic has been more strongly associated with these adverse outcomes than total vehicular traffic (6, 7, 10, 11).

Most studies have used metrics of proximity to traffic as surrogates of exposure to traffic pollution (e.g., residential proximity to major roads, traffic volume at the nearest road, or modeled levels of traffic pollution). Few have measured pollutant concentrations as part of the exposure assessment or provided information on local air quality (7, 10–12). The majority of studies have been conducted in Europe and Japan, where fleet composition (diesel versus gasoline), emissions factors, fuel specifications, land use, and population distributions near busy roads differ from those in the United States. Regional and microenvironmental concentrations of particulate matter (PM) may be higher in European cities compared with many parts of the United States (13). Therefore, it is important to evaluate the extent to which proximity to traffic may be associated with health impacts in the United States. Previous studies in the United States were conducted in areas of Southern California and the Northeast with significant local air-quality problems; both used metrics of proximity to traffic, not measured pollutant concentrations (8, 14).

The objective of this study was to explore associations between respiratory symptoms and exposures to traffic-related air pollutants among children living and attending schools near busy roads in an urban area with high traffic density but good regional air quality. Some of the results of this study have been previously reported in the form of abstracts (15).

METHODS

Study Design and Health Assessment

We conducted a school-based, cross-sectional study in the San Francisco metropolitan area (Alameda County, CA) in 2001. The study area was comprised of 10 neighborhoods that span a busy traffic corridor. School sites were selected to represent a range of locations upwind and downwind of major roads (Figure 1).

In spring 2001, we enrolled children (grades 3–5) in participating classes (n = 64) using methods similar to those used in other school-based studies (16–18). We obtained information on health outcomes (bronchitis symptoms in the past 12 months and physician-confirmed asthma in the past 12 months), demographics, home environmental factors, and activity factors using parental questionnaires (English and Spanish) (for additional information on the study design and health assessment, *see* the online supplement). The study protocol was approved by the Committee for the Protection of Human Subjects, California Health and Human Services Agency.

Air Pollution from Traffic

We measured concentrations of traffic pollutants (particulate matter [PM_{10}, $PM_{2.5}$], black carbon [BC], total nitrogen oxides [NO_X], and nitrogen dioxide [NO_2]) at the school sites. PM_{10} and $PM_{2.5}$ mass concentrations were measured using filter-based samples, whereas BC concentrations were determined on the PM_{10} filter samples using an established light attenuation method that we validated for fiberfilm filters (19, 20). NO_X and NO_2 concentrations were determined with passive diffusion samplers (Ogawa, Inc., Pompano Beach, FL). Nitric oxide (NO) concentrations were calculated as the difference between NO_X and NO_2.

Pollutant monitoring was conducted simultaneously at all school sites for 11 1-week intervals in the spring (March–June) and for 8 weeks

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TABLE 3. ODDS RATIOS (95% CONFIDENCE INTERVAL) OF RESPIRATORY ILLNESS BY SCHOOL-BASED AMBIENT AIR POLLUTANT CONCENTRATIONS USING TWO-STAGE MODEL

		All Subjects		LTR Subjects		LTR Females		LTR Males	
		(*n* = 1,109)		(*n* = 871)		(*n* = 462)		(*n* = 403)	
	Exposure	OR	CI	OR	CI	OR	CI	OR	CI
Bronchitis*		n = 93/797		n = 79/635		n = 38/341		n = 41/291	
	NO_x	1.05	(1.01, 1.08)	1.06	(1.03, 1.09)	1.07	(1.03, 1.11)	1.03	(0.98, 1.09)
	NO_2	1.02	(0.99, 1.06)	1.03	(1.00, 1.06)	1.04	(1.01, 1.08)	1.02	(0.98, 1.06)
	NO	1.05	(1.02, 1.09)	1.06	(1.03, 1.09)	1.07	(1.03, 1.11)	1.04	(0.98, 1.10)
	PM_{10}	1.03	(0.99, 1.07)	1.02	(0.98, 1.07)	1.04	(1.01, 1.09)	1.01	(0.95, 1.06)
	$PM_{2.5}$	1.02	(1.00, 1.05)	1.03	(1.01, 1.05)	1.04	(1.02, 1.05)	1.02	(0.99, 1.05)
	BC	1.04	(1.00, 1.08)	1.05	(1.01, 1.08)	1.06	(1.02, 1.10)	1.03	(0.98, 1.08)
Asthma†		n = 101/705		n = 78/562		n = 42/297		n = 36/263	
	NO_x	1.04	(0.97, 1.11)	1.07	(1.00, 1.14)	1.17	(1.06, 1.29)	1.02	(0.93, 1.11)
	NO_2	1.02	(0.97, 1.07)	1.04	(0.98, 1.10)	1.09	(1.03, 1.15)	1.00	(0.94, 1.07)
	NO	1.05	(0.98, 1.12)	1.08	(1.00, 1.15)	1.19	(1.03, 1.36)	1.02	(0.94, 1.12)
	PM_{10}	1.02	(0.96, 1.09)	1.04	(0.97, 1.12)	1.09	(0.92, 1.29)	1.02	(0.94, 1.10)
	$PM_{2.5}$	1.00	(0.97, 1.04)	1.01	(0.97, 1.06)	1.06	(0.99, 1.15)	0.99	(0.95, 1.04)
	BC	1.02	(0.96, 1.09)	1.05	(0.99, 1.13)	1.12	(0.95, 1.33)	1.00	(0.93, 1.09)
Asthma (no outlier, school 5)†		n = 96/641		n = 73/507		n = 38/271		n = 35/233	
	NO_x	1.08	(1.00, 1.17)	1.10	(1.00, 1.20)	1.14	(1.02, 1.28)	1.07	(0.96, 1.19)
	NO_2	1.06	(0.99, 1.13)	1.07	(0.98, 1.17)	1.09	(0.97, 1.22)	1.05	(0.96, 1.16)
	NO	1.08	(1.00, 1.17)	1.09	(1.00, 1.19)	1.14	(1.03, 1.26)	1.07	(0.96, 1.18)
	PM_{10}	1.06	(0.97, 1.16)	1.08	(0.98, 1.19)	1.09	(0.96, 1.24)	1.08	(0.97, 1.19)
	$PM_{2.5}$	1.04	(0.96, 1.12)	1.03	(0.94, 1.13)	1.03	(0.91, 1.17)	1.03	(0.94, 1.14)
	BC	1.07	(0.98, 1.17)	1.09	(0.99, 1.19)	1.14	(1.02, 1.27)	1.06	(0.95, 1.18)

Definition of abbreviations: BC = black carbon; CI = confidence interval; LTR = long-term resident; OR = odds ratio; NO = nitric oxide; NO_x = total nitrogen oxides; NO_2 = nitrogen dioxide; $PM_{2.5}$ = particulate matter of aerodynamic diameter 2.5 μm or less; PM_{10} = particulate matter of aerodynamic diameter 10 μm or less.

Odds ratios are calculated per IQR of average pollutant concentrations as follows: NO_x =14.9 ppb; NO_2 = 3.6 ppb; NO = 11.6 ppb; PM_{10} = 1.4 μg/m³; $PM_{2.5}$ = 0.7 μg/m³; BC = 0.15 μg/m³.

For hierarchical analyses of asthma in long-term residents (current address for 1 year or more), only 9 schools were included in the analysis; one school had no cases (due to low numbers and missing values).

* First stage model adjusted for: child's respiratory illness before age 2; pests, indicator of mold presence.

† First stage model adjusted for: child's respiratory illness before age 2; pests, indicator of mold presence; maternal history of asthma.

least 1 year at current residence) tended to increase the effect estimates slightly in relationship to asthma, especially when the sample was restricted to girls. Stratification by duration of residence or sex did not change the results for bronchitis. Results were similar when nonnormalized pollution values were used (data not shown).

We conducted additional sensitivity analyses, including (*1*) dropping the one school that was an outlier with respect to the proportion of Hispanic students (89% vs. 21–53% at other schools), (*2*) using a different definition for current asthma, and (*3*) stratifying bronchitis by a reported history of asthma. When the "outlier" school was dropped, the magnitude of the ORs for bronchitis did not change much, but the confidence intervals were wider. In the asthma analyses, dropping the outlier school resulted in similar or slightly greater effect estimates. Applying different questionnaire-based asthma definitions showed little change but slightly larger confidence intervals. After stratifying students by whether they also "ever" had asthma, the results suggested that those with a history of asthma were driving the results for bronchitis, but the sample size became too small to make clear inferences. Figures 2 and 3 depict the associations between BC and bronchitis and asthma.

DISCUSSION

To our knowledge, this is the first epidemiologic study in the United States to evaluate relationships between measured traffic-related pollutants and respiratory symptoms. For children residing at their current address for at least 1 year, we found modest but significant increases in the odds of bronchitis symptoms and physician-diagnosed asthma in neighborhoods with higher concentrations of traffic pollutants. These results are consistent with previous reports of positive associations between proximity to traffic and various respiratory outcomes (4–12). Furthermore, our findings were observed in a region with relatively clean air (low concentrations of ozone and PM) (*see* the online supplement for details). Although previous epidemiologic studies in the United States exploring chronic respiratory effects of air pollution in children have shown inconsistent results, this might be due in part to exposure misclassification, as these studies used air quality measurements conducted at single fixed-site monitors in each city (17, 18, 22, 23).

Our findings were robust to multiple sensitivity analyses using different questionnaire-based definitions of current asthma and wheezing in the past 12 months. The slight increase in effect estimates for associations between asthma after restricting the analysis to those with longer duration at current residence may be due to a reduction in exposure measurement error. Our study population was very mobile (23% had moved in the preceding 12 months, and only 32% had lived at the same address since before the age of 2 years).

We considered whether there might be bias due to nonresponse or self-reporting. We saw no significant difference in proportions of questionnaires returned in Spanish versus English by school, but there was a modest inverse correlation between pollution concentrations measured at each school and response rate. However, the response rate for individual classrooms within each school varied as well and appeared to depend on the willingness of teachers to encourage participation. Dropping the school closest to a freeway (which also had the highest measured pollutant concentrations, a high percentage of Hispanic students, and the lowest response rate) did not change the effect estimates for

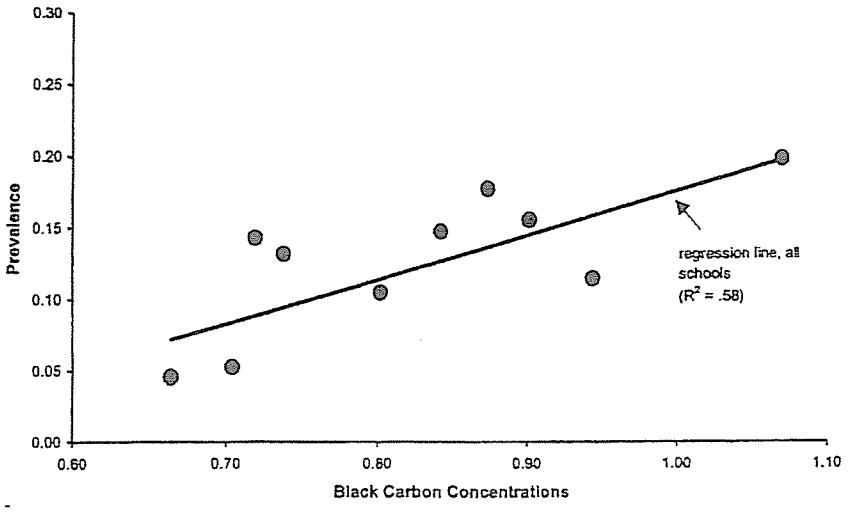

Figure 2. Adjusted school-specific bronchitis prevalence rates versus black carbon, long-term residents.

bronchitis and increased the estimates for asthma. This would suggest that knowledge of potential high traffic exposure probably did not affect parental reporting of the children's respiratory histories. This study was not undertaken in response to public concerns about traffic nor, at the time the study was conducted, was there much local interest in potential health hazards of proximity to traffic. Therefore, reporting and nonresponse biases were unlikely to have unduly influenced our results.

We found increased association with asthma (but not bronchitis) with exposure to traffic air pollutants for girls who had lived at their current addresses at least 1 year compared with boys (Table 3). Several investigators have also reported greater traffic-associated effect estimates for girls versus boys (7, 8, 10, 24, 25). Previous air pollution studies examining the sex-specific effects of air pollution on lung function and lung function growth have been mixed (26, 27). The reasons for the observations in our study are unclear and deserve attention in future studies.

Exposures

We found spatial variability in exposure due specifically to roads with heavy traffic within a relatively small geographic area for BC, NO_X, NO, and to a lesser extent NO_2. There was less variation in $PM_{2.5}$ across schools; this is consistent with previous observations that $PM_{2.5}$ is more likely to reflect regional air quality (2). The higher effect estimates with BC, NO_X, and NO compared with NO_2 and $PM_{2.5}$ suggest that primary or fresh traffic emissions may play an etiologic role in these relationships. Although NO_X, NO, and BC may serve as indicators of exposure to traffic-related pollutant mixtures, they may also act as etiologic agents themselves (28).

We found that downwind direction was an important determinant of increased exposure to traffic pollutants and that a simple traffic indicator (school location downwind and < 300 m from a major road) gave estimates of ORs similar to or greater than pollutant measurements in preliminary analyses using a one-stage model (data not shown). Within a geographic area with flat terrain and low-rise buildings, the direction of wind in relationship to the traffic source is the most important weather parameter. Other parameters important in air dispersion of traffic pollutants (e.g., atmospheric stability, wind speed, and surface topography) would be relatively similar at the different school sites.

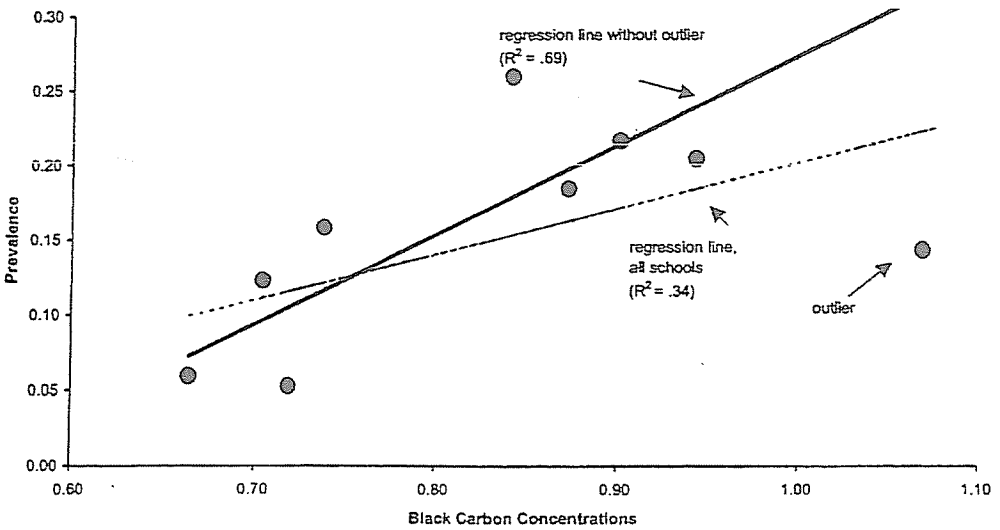

Figure 3. Adjusted school-specific asthma prevalence rates versus black carbon, long-term residents.

A simple single-stage logistic model using pollutant measurements also yielded positive associations between pollutants and symptoms with a much larger effect estimate and smaller confidence intervals.

We assumed that traffic-related pollutants measured at the neighborhood schools would be a good proxy for the children's overall exposure to such pollutants. Children attending the schools in this study generally lived within walking distance and did not use school buses. Therefore, pollutant concentrations in the children's neighborhoods probably tracked those at their schools. The most plausible exposure error in an urban setting would be that subjects who attend schools with very high traffic exposures from a nearby freeway would tend to have similar or lower home exposures, whereas children with low school exposures would tend to live in homes with similar or only slightly higher traffic exposures. This pattern of measurement error would tend to underestimate the association between exposure and outcome (29).

Alternatively, repeated daily exposures for 6–8 hours during the school year may themselves represent biologically important influences on some children's respiratory health, analogous to occupational exposures for susceptible adults. In a recent study of proximity to traffic and respiratory health, Janssen and colleagues found that effect estimates based on the school-to-highway distance were comparable or greater than those based on residence-to-highway distance (11).

The average measurements at each school were used to estimate long-term average traffic air pollutant concentrations. We measured pollutants at each of the 10 sites concurrently (to avoid concerns of week-to-week variability) in two different periods that reflect the major seasonal wind patterns for the area. We found that the rank order (relative values) of the schools did not vary from week to week or season to season, supporting the validity of this approach. Additionally, the NO_x and NO_2 concentrations at schools upwind or further from high traffic roads were similar to NO_X and NO_2 concentrations measured at the closest fixed-site monitor (21). Although there may have been some changes in the absolute traffic volume on major roads in recent years, the principal traffic patterns in the area have not changed. Thus, the relative values (rank order) of the site-specific pollutant concentrations measured in our study are likely to be representative of those in recent years.

The cross-sectional nature of our study design is a further limitation on causal inference, but we observed the same or modest increase in effect estimates for current asthma and bronchitis when we restricted our analysis to those who had lived at their present address for at least a year. Most studies on proximity to traffic and respiratory symptoms have been cross-sectional, and further longitudinal studies are needed to elucidate the role of traffic-related air pollution in the development and exacerbation of asthma and other respiratory symptoms.

Another limitation was that the exposures were assigned at the group level (n = 10); however, the multilevel analysis allows adjustment for individual confounders in the first stage of analysis. Moreover, in this respect, this study is comparable with other epidemiologic investigations (e.g., the Harvard Six Cities Study and the Children's Health Study in Southern California) (n = 12 communities). Another recent cross-sectional study of traffic-related air pollution and respiratory symptoms included 13 schools (18, 22, 23).

We also lacked information on indoor measurements of traffic-related pollutants. However, recent studies have found high correlations between personal exposures to NO_2 and traffic parameters (30). Others have found that indoor concentrations and exposure to soot (PM from diesel exhaust) is highly correlated with outdoor levels (2).

Other Covariates

Maternal asthma, household mold/moisture, pests, and chest illness before the age of 2 years were important explanatory variables in the final model for current asthma, consistent with previous studies (31–33). We explored whether current levels of traffic pollution could modify the risk of current asthma symptoms depending on past history of chest illness; however, there was not sufficient power to explore interactions based on early medical history. Race/ethnicity and indicators of socioeconomic status were not important predictors of health outcomes in our study. This may be due, in part, to our study design (i.e., the schools were selected to have relatively similar measures of socioeconomic status).

We did not find associations between exposure to environmental tobacco smoke and current asthma; the results of previous cross-sectional studies in school-aged children have been mixed (34). The prevalence of current household smokers in our study was small, however, limiting study power. It is possible that there is some underreporting of household smoking (7% in our study vs. 19% statewide). (35) Alternatively, a substantial portion of our study population was less acculturated Hispanics (30% of parents responded in Spanish), and only 3.6% of Hispanic households reported a history of maternal smoking. Other investigators have also observed very low smoking rates (less than 5%) among less acculturated Hispanics (B. Eskenazi, personal communication) (36). If underreporting does exist, it is possible that residual confounding might have affected our estimates of pollutant/respiratory health outcome relationships. However, the addition to the regression model of variables correlated with exposure to environmental tobacco smoke (e.g., socioeconomic status and race–ethnicity) did not change the pollutant effect estimates, suggesting that significant confounding by environmental tobacco smoke was not likely.

In summary, we found associations between traffic-related pollutants and asthma and bronchitis symptoms in the past 12 months in a highly urbanized region of the United States with good regional air quality, where local air pollution is dominated by vehicular sources. Although the cross-sectional study design, exposure assignment at the group level, small geographic area, and possible unmeasured covariates may limit the generalizability of the study, our findings are consistent with previous investigations in Europe and the United States (11, 14, 37). In addition, our results underscore the limitations of using central air monitoring stations for assigning population exposures. Concentrations of air toxics such as diesel exhaust particles or surrogates such as BC or soot should be more widely monitored. Measurement of personal exposures to traffic pollutants is not feasible in large population-based studies; the use of geographic modeling approaches to estimate exposures for individuals may be a good alternative (38). Future studies that can better characterize exposures to traffic pollutants, and their sources (i.e., diesel versus gasoline engines) will be important to understand better the public health impacts of motor vehicle emissions.

Conflict of Interest Statement: J.J.K. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; S.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; M.L. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; B.C.S. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; A.T.H. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript; B.O. does not have a financial relationship with a commercial entity that has an interest in the subject of this manuscript.

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Review

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Cardiovascular implications of exposure to traffic air pollution during exercise

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Summary

Regular aerobic exercise is recommended by physicians to improve health and longevity. However, individuals exercising in urban regions are often in contact with air pollution, which includes particles and gases associated with respiratory disease and cancer. We describe the recent evidence on the cardiovascular effects of air pollution, and the implications of exercising in polluted environments, with a view to informing clinicians and other health professionals. There is now strong evidence that fine and ultra fine particulate matter present in air pollution increases cardiovascular morbidity and mortality. The main mechanisms of disease appear to be related to an increase in the pathogenic processes associated with atherosclerosis. People exercising in environments pervaded by air contaminants are probably at increased risk, due to an exercise-induced amplification in respiratory uptake, lung deposition and toxicity of inhaled pollutants. We make evidence-based recommendations for minimizing exposure to air-borne toxins while exercising, and suggest that this advice be passed on to patients where appropriate.

Introduction

Regular aerobic exercise promotes a range of physiological changes that correlate with decreased morbidity and increased longevity.[1] Accordingly, health professionals recommend regular endurance training in order to maintain health, and also as a therapeutic intervention in certain diseases (e.g. hypertension, diabetes mellitus, coronary heart disease). Many exercise programs involve training in outdoor areas, which in urban environments may be near roadways, in close proximity to motor vehicles. Automobile emissions are estimated to be the greatest single contributor to urban air pollution, and their toxic constituents contribute to respiratory disease and cancer.[2] We discuss recent findings on the lesser-known cardiovascular effects of acute and chronic exposure to air pollution, with particular emphasis on the potential health implications of exercising in environments permeated by automotive pollution.

Components of automotive pollution

A diverse mixture of suspended particles and gases containing reactive free radical species is released into the atmosphere as a consequence of fuel

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combustion. The ambient air pollution is mostly (~99.4%) invisible to the naked eye, unless present in very high concentrations, in which case it may be seen as the haze commonly associated with large cities. Particulate matter air pollution is a heterogeneous mixture of solids and liquids that are generally classified according to aerodynamic diameter, as coarse (<10 μm; PM_{10}), fine (<2.5 μm; $PM_{2.5}$) or ultrafine (<0.1 μm; $PM_{0.1}$) particles. Individual particles may be complex chemical mixtures comprising different biochemically active components on the outside compared to the inside. Presently, the air quality standards for PM_{10} and $PM_{2.5}$ in the US are 50 $\mu g/m^3$ and 15 $\mu g/m^3$ respectively, which represent the annual maximal allowable arithmetic means for particulate matter. In the UK, the recommended limit of PM_{10} over a 24-h period is 50 $\mu g \cdot m^3$, which is regularly exceeded. Some contend that these standards may be too low, considering that deleterious health effects may be elicited after regular exposure to concentrations lower than these in outdoor air.[3]

The major gaseous components of automotive pollution include sulphur dioxide (SO_2), carbon monoxide (CO), nitrogen oxides (NO_x) and ozone (O_3). A variety of readily evaporable toxic volatile organic compounds (e.g. benzene, ethylene) are also emitted from unburnt fuel, as well as during the fuel combustion process. Indeed, the interaction of these compounds with NO_x forms additional O_3 in the presence of heat and sunlight. Another class of over 100 chemicals formed during incomplete burning of fuel and other organic substances (e.g. wood and tobacco) is that of the polycyclic aromatic hydrocarbons (e.g. benzo(a)pyrene, fluorine), many of which are known carcinogens.[4] Ultimately, air pollution comprises a complex mixture of thousands of chemicals and chemical interactions.

Cardiovascular epidemiological evidence

Early epidemiological studies examining the relationship between chronic exposure to automotive pollution and disease found an unexpectedly increased incidence of cardiovascular mortality in workers exposed to high levels of pollution.[5,6] Initially, these findings were inconclusive, because investigators were unable to account for the potential confounding variable of cigarette smoking. However, more recent and well-controlled epidemiological studies have supported these original findings by consistently observing an association between ambient concentrations of particulate matter and cardiovascular morbidity[7–9] and mortality.[2,3,10–12] Indeed, these relationships remain, despite air pollution levels being lower than the recommended national standards of each country. $PM_{<2.5}$ appears to be the most studied and may be of more risk to health, possibly due to these very small particles penetrating the lower regions of the lung and entering the circulation.[13]

Studies have demonstrated a significant elevation in the incidence of life-threatening myocardial infarctions[8] and cardiac arrhythmias[7] in the immediate period (hours to days) following exposure to high levels of atmospheric fine particulate matter. Similarly, the presence of fine and ultrafine particulate pollution has predicted the risk for exercise-induced ST-segment depression (indicative of myocardial ischaemia) in subjects with coronary heart disease.[9] Other epidemiological data link air pollution to an augmentation of systemic inflammation, as measured by C-reactive protein,[14] an acute-phase protein associated with adverse outcome in patients with unstable ischaemic syndromes. Interestingly, compared to controls, patients with diabetes mellitus were more than twice as likely to be admitted to hospital with heart-disease-related pathology when atmospheric PM_{10} increased by 10 $\mu g/m^3$,[3,15] suggesting that certain populations may be more susceptible to particulate pollution. Indeed, frail individuals with existing heart and lung disease have also been shown to be more susceptible to the effects of air pollution, as this population had an increased risk of death after exposure to PM_{10} levels that were within the national standard for the US.[3] All these large-scale studies show a persistent concentration-dependent association between air pollution and cardiovascular risk.

Cardiovascular mechanisms of action

The main pathway by which particulate air pollution contributes to increased risk is probably by direct augmentation of atherosclerosis,[16–18] the underlying cause of most cardiovascular disease. The aetiology of atherosclerosis is multifactorial, but the process may be initiated and promoted by toxic insult to the endothelium, and is associated with chronic inflammation as well as high circulating concentrations of metabolites indicative of oxidative stress.[19] Table 1 summarizes some of the experimental work on exposure to automotive pollution relevant to atherosclerosis.

It has only recently been established that ultrafine particles translocate to the circulation directly after inhalation,[13] which provides an explanation

for the route by which toxic pollutants may systemically affect the blood, vasculature and organs such as the heart. Several studies have reported diminished antioxidant defences and increased markers of oxidative damage (e.g. malondialdehyde) in plasma and endothelial cells after exposure to components of automotive pollution such as CO and nitrogen dioxide (NO_2).[20–23] Workers chronically exposed to high pollutant levels have plasma that is more susceptible to oxidation, possibly due to depletion of water-soluble antioxidants.[24] These detrimental blood and cellular changes may increase the oxidation of circulating low-density lipoproteins, thus making these particles more susceptible to increased uptake into the arterial wall and accelerating atherogenesis.[25] This hypothesis is supported by the demonstration that lipid uptake into the arterial wall is increased in human coronary arteries perfused with blood containing low levels of carboxyhaemoglobin.[26] Additional cardiovascular risk may also arise from thrombotic complications as a consequence of increased platelet activation[27] and an elevated inflammatory response[28] caused by systemic infiltration of air pollution.

In humans, environmentally relevant concentrations of $PM_{2.5}$ and O_3 acutely augment brachial artery vasoconstriction.[29] Rats exposed to the same pollutants also show elevated concentrations of plasma endothelin-1, a powerful vasoconstrictor.[30] These data are of particular interest to cardiovascular risk in humans, because augmented vasomotor tone (vasoconstriction) has the potential to increase myocardial afterload and ischaemia. Each time the heart contracts, a pressure wave travels through conduit arteries of low resistance to peripheral arteries of higher resistance. A portion of the pressure wave is reflected back to the heart (wave reflection) and the intensity of this reflection is dependent on the tone of the large conduit arteries. Increased large artery vasoconstriction causes increased wave reflection, such that there is an early return to the heart of the arterial pressure waveform. This early returning waveform boosts central (aortic) systolic blood pressure (afterload), in addition to diminishing the time and pressure of coronary artery perfusion, thus promoting ischaemia.[31] If regular exercise in a polluted environment exacerbates this effect, risk would be enhanced, because *central*, and not peripheral, blood pressure correlates with left ventricular hypertrophy,[32] carotid intima media thickness[33] and all-cause mortality.[34]

Abnormal function of the endothelial cells lining the arteries has been implicated in early atherogenesis, possibly due to decreased bioavailability of nitric oxide, leading to vasoconstriction, smooth muscle cell proliferation and thrombotic processes.[35] Benzo(a)pyrene, a cytotoxic component of air pollution, can alter gene expression and enhance the proliferation of vascular smooth muscle cells,[36] a structural change that accompanies atherosclerosis. Further, phenanthraquinone, a constituent of diesel exhaust, inhibits nitric oxide production in bovine endothelial cells, increases blood pressure and suppresses nitric-oxide-mediated vasodilatation in rats.[37] Inhibition of endothelial-dependent vasorelaxation has also been demonstrated in isolated ring preparations of rat thoracic aortas exposed to diesel[38] and motorcycle exhaust particles.[39] Taken together, these data imply that normal vascular homeostasis is interrupted by exposure to auto pollution, and this would help to explain excessive cardiovascular morbidity and mortality from air pollution.

Exercise and automotive pollution

A primary reason why exercising individuals may be at heightened risk for disease is because, even at low intensities, a significant rise in pulmonary ventilation and diffusion capacity occurs,[40] meaning that the concentration of inspired particles will increase. Indeed, the total amount of particulate matter deposited in the lungs of exercising humans has been shown to be directly related to minute ventilation, and greater deposition has been demonstrated during slower, deeper breathing rather than rapid and shallow breathing.[41] Daigle *et al.*[42] recently determined that the total amount of $PM_{0.1}$ deposited in the respiratory tract of humans during moderate exercise was approximately five times that at rest. Additionally, the fractional penetration of pollutants to the lung is greater when breathing by mouth compared to the nose. This effect is attributed to increased absorption of gases and deposition of particles in the nasopharyngeal region ('scrubbing' action), which is bypassed with oral breathing.[43] During exercise, particularly at higher intensities, a greater portion of air is taken in via the mouth,[44] thus again increasing the load of inhaled toxicants.

As expected, the total lung burden of inhaled particles increases as the concentration of ambient pollutants increases.[43] Blood levels of toxins may rapidly reach harmful levels, as was shown in New York City runners after 30 min of exercise near busy roadways. This activity evoked an acute rise in blood carboxyhaemoglobin levels from 1.7% to 5.1%,[45] which is similar to those found in regular cigarette smokers. Of particular interest was the

finding that during exercise, only low concentrations of pollutants (O_3 and NO_2) were required to cause similar lung damage to that achieved by high concentrations of the same compounds at rest.[46] Also, animals exercising during exposure to O_3 had more severe lung parenchymal lesions than at rest with the same O_3 concentration.[47] Finally, uptake of pollutants into the lower respiratory tract[43] and lung tissue damage[46] appears to be higher when inspired compounds are in a mixture (i.e. formaldehyde and ammonium nitrate) rather than in isolation. These findings imply firstly, that some chemicals may act in a synergistic manner to adversely effect health and secondly, exercise may exacerbate toxic effects. A summary of the interaction between air pollution, exercise and cardiovascular risk is shown in Figure 1.

Recommendations

Clinicians should not be dissuaded from encouraging people to exercise, since epidemiological studies indicate that the benefits of regular exercise outweigh potential harm.[1,48] However, given the evidence linking air pollution to disease, together with the possibility that exercising near road traffic may intensify harmful effects, it is advisable to avoid or minimize exposure to air-borne contaminants. Accordingly, we recommend that physicians

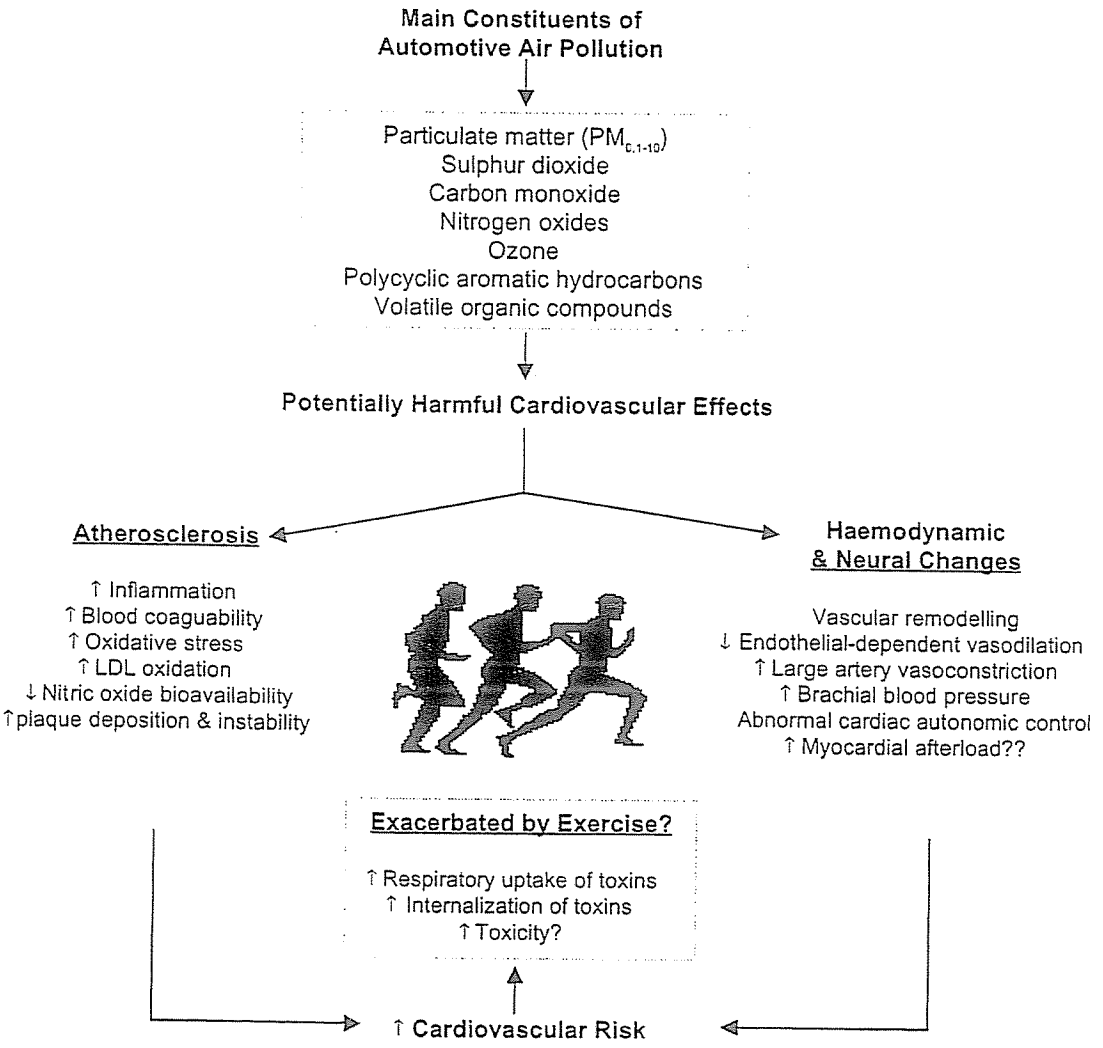

Figure 1. Summary of the potentially adverse effects of automotive pollution on the cardiovascular system.

and other health professionals advise patients undertaking an exercise program to *exercise outdoors in parks and recreation areas away from busy roadways or industrial sites*. This advice does not take into consideration regional differences in ambient pollutant levels that vary with the time of day. Therefore, in large cities where the ambient atmospheric levels of particulate matter regularly exceed national air safety standards (i.e. London), it may be useful to limit exercise sessions to the hours of the day when air pollution is likely to be less concentrated (i.e. early hours of the morning). Importantly, some populations may be especially sensitive to air pollution (i.e. children, elderly, diabetics or those with existing heart or lung disease) and care should be given to offer prescriptive advice to these people in particular.

Conclusions

There is now strong evidence that traffic air pollution aggravates the risk for cardiovascular disease in a concentration-dependent manner. While there is no doubt that regular physical activity is of great benefit to health and longevity, both in healthy individuals and those with pre-existing cardiovascular conditions, exercising in urban environments in close contact with motor vehicles and high ambient levels of particulate matter will increase the total amount of inspired toxins, and most likely increase the risk of disease. Despite the evidence, it is not uncommon to see individuals exercising in highly polluted areas such as alongside busy suburban roadways, and through inner cities congested with motor vehicles, which may be more harmful than beneficial. The purpose of this review was to inform clinicians and other health professionals of the hazards of exercising in environments pervaded by automotive pollution, so that they may appropriately advise patients on avoidance.

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ENVIRONMENTAL EXPOSURE

Traffic exposure and lung function in adults: the Atherosclerosis Risk in Communities study

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Background: Traffic exposure is a major contributor to ambient air pollution for people living close to busy roads. The relationship between traffic exposure and lung function remains inconclusive in adults.
Methods: A cross-sectional study was conducted to investigate the association between traffic exposure and lung function in the Atherosclerosis Risk in Communities (ARIC) study, a community based cohort of 15 792 middle aged men and women. Traffic density and distance to major roads were used as measures of traffic exposure.
Results: After controlling for potential confounders including demographic factors, personal and neighbourhood level socioeconomic characteristics, cigarette smoking and background air pollution, higher traffic density was significantly associated with lower forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) in women. Relative to the lowest quartile of traffic density, the adjusted differences across increasing quartiles were 5.1, −15.4 and −21.5 ml for FEV_1 (p value of linear trend across the quartiles = 0.041) and 1.2, −23.4 and −34.8 ml for FVC (p trend = 0.010). Using distance from major roads as a simpler index of traffic related air pollution exposure, the FEV_1 was −15.7 ml (95% CI −34.4 to 2.9) lower and the FVC was −24.2 ml (95% CI −46.2 to −2.3) lower for women living within 150 m compared with subjects living further away. There was no significant effect of traffic density or distance to major roads on lung function in men. The FEV_1/FVC ratio was not significantly associated with traffic exposure in either men or women.
Conclusions: This is the largest published study of traffic exposure and pulmonary function in adults to date. These results add to growing evidence that chronic exposure to traffic related air pollution may adversely affect respiratory health.

Road traffic is a major factor in ambient air pollution in industrialised countries, contributing pollutants including fine particulate matter, carbon monoxide and oxides of nitrogen. An expanding body of epidemiological research suggests that traffic related exposure is associated with acute and chronic respiratory effects.[1–7] For example, residential proximity to busy roads is associated with a variety of adverse respiratory health outcomes including symptoms[1–3] and asthma exacerbation.[4–7] The effect of traffic air pollution on adult lung function remains inconclusive; exposure to automobile exhaust was associated with lower lung function in adults in some studies[8–12] but not others.[13 14]

Traffic emissions result in small scale spatial variations and higher concentrations within short distances from major roads.[15 16] Air pollution data from fixed monitoring stations may be inadequate to study traffic related air pollution and health outcomes, especially for those living near busy roads. For example, Hoek and colleagues identified a consistent association between cardiopulmonary mortality and living near a major road, but not with estimated ambient background concentration of the traffic indicator pollutants black smoke and nitrogen dioxide.[17] Assessment of traffic exposure can enhance studies of health effects of ambient air pollution because local sources are important, and because few people live close to the monitoring stations which are purposefully located away from local sources like busy roads. For people living close to busy roads, air pollution from traffic sources may be more important than the area background measured at the closest monitoring station.

We examined the relation between traffic exposure and lung function in a population based cohort of 15 792 middle-aged men and women, the Atherosclerosis Risk in Communities (ARIC) study.

METHODS

Study population

Participants were from the ARIC study which is designed to investigate the natural history and aetiology of atherosclerosis and its sequelae. The design, objectives and quality control activities of the ARIC study have been reported in detail elsewhere.[18 19] Participants were sampled from four US communities: Forsyth County, North Carolina; Jackson, Mississippi; northwest suburbs of Minneapolis, Minnesota; and Washington County, Maryland. The lung function variables collected during visit 1 (1987–9) were used with the traffic and background air pollution data in a cross-sectional analysis. Participants of an ethnicity other than African American or white were excluded from the current analysis (n = 48). Also, African-Americans from Minnesota and Maryland field centres were excluded (n = 55) because of their small number.

We also repeated this analysis using visit 2 (1990–2) and conducted a longitudinal analysis on the change in lung function between visit 1 and visit 2.

Pulmonary function measurements

The main measurements of lung function were forced expiratory volume in 1 s (FEV_1), volume of gas (in litres) exhaled in the first second of expiration; forced vital capacity (FVC), total volume of gas exhaled; and the ratio of FEV_1/FVC.

Abbreviations: ARIC, Atherosclerosis Risk in Communities; BMI, body mass index; ETS, environmental tobacco smoke; FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity; GIS, geographical information system; PM_{10}, particulate matter <10 μm

Table 2 Distributions of distance to different classes of roads at ARIC participant residences (in metres, n=13 972)*

	Mean	SD	Minimum	P25	Median	P75	Maximum
Distance to class 1 roads	2207	2008	30	896	1795	2774	17053
Distance to class 2 roads	2755	2573	20	923	2254	3593	22382
Distance to class 3 roads	363	502	10	103	230	427	5389
Distance to class 4 roads	29	42	10	10	18	31	833

P25 and P75, 25th and 75th percentile.
*Interstate highways (class 1), state highways (class 2), major arterials (class 3) and local roads (class 4).

Traffic exposure and lung function

Because we observed a significant interaction between sex and traffic density for FVC ($p = 0.041$), the relationships between measures of traffic exposure and lung function are presented using sex-specific multivariate regression.

Both before and after adjustment for confounders, an inverse relation was found between lung function and traffic density among women (table 3). Relative to the lowest quartile of traffic density, the adjusted differences across increasing quartiles were 5.1, −15.4 and −21.5 ml for FEV_1, and 1.2, −23.4 and −34.8 ml for FVC. This linear trend was significant for both FEV_1 ($p = 0.041$) and FVC ($p = 0.010$). We did not find a clear association between traffic density and FEV_1 or FVC in men (table 4).

A similar pattern, although of lower statistical significance, was seen for living near major roads. Among women living within 150 m of a major road, the FEV_1 was −15.7 ml (95% CI −34.4 to 2.9) lower and the FVC was −24.2 ml (95% CI −46.2 to −2.3) lower in multivariate analyses compared with women living further away (table 5). In a sensitivity analysis using different cut off points (≥100 m and <100 m), similar but weaker patterns were found in women (table 5). We did not observe significant effects of distance in men.

The ratio of FEV_1/FVC was not significantly associated with traffic density or distance to major roads in either sex (tables 3–5).

We did not observe significant effect modification of the association between traffic density and lung function by smoking ($p = 0.989$ for FEV_1 and 0.867 for FVC) or ethnicity ($p = 0.371$ for FEV_1 and 0.147 for FVC).

When the analyses were repeated using traffic exposure and lung function data obtained at visit 2, the associations were generally similar to those described for visit 1 (see tables A7 and A8 in the online appendix available at http://thorax.bmj.com/supplemental).

Given the small effect of traffic in our study and earlier publications[8–12] and because we had only two pulmonary function measurements spaced 3 years apart, we did not anticipate being able to detect an effect of traffic on change in pulmonary function. Nonetheless, we performed supplementary longitudinal analyses. No significant associations were found (see tables A9 and A10 in the online appendix available at http://thorax.bmj.com/supplemental).

DISCUSSION

This study provides evidence that lung function, as measured by FEV_1 and FVC, is reduced in adults exposed to higher levels of traffic, especially among women. To our knowledge, this is the first population-based study in the USA and the largest one to date in the world to investigate the relation between measured traffic exposure and lung function in adults. The magnitude of the observed association between traffic exposure and lung function was similar to reported effects of outdoor air pollution[29] and smaller than effects of personal smoking[30] or ETS exposure.[31]

As in some other studies,[32–36] we did not find a significant association between the FEV_1/FVC ratio and measures of traffic exposure. This suggests that the traffic related reduction in FEV_1 and FVC was probably due to loss of lung volume (restriction) rather than airflow obstruction. Of course, a reduced FVC in the presence of a normal FEV_1/FVC can be used to suggest—but not to diagnose—the presence of a restrictive abnormality.[37]

There are relatively few published studies of traffic related air pollution and lung function in either children or adults. In the Netherlands, investigators found that exposure to traffic related air pollution, especially diesel exhaust particles, was associated with reduced lung function in children living near major motorways.[38] A German study suggested that exposure to a pollution profile of heavy traffic and domestic heating was related to markedly lower FVC and FEV_1 in children.[39] Several recent studies on traffic air pollution provide some support for our finding.[10–12 14] An 8-year longitudinal study of 5682 women in Tokyo showed a larger decrease in FEV_1 for participants with

Table 3 Associations between traffic density and FEV_1, FVC and FEV_1/FVC ratio in female ARIC participants (1987–9)

	Quartiles of traffic density				
	1 (lowest)	2	3	4	p Value†
FEV_1 (ml)					
Age adjusted model	0	2.8 (−28.6 to 34.2)	−15.9 (−47.1 to 15.4)	−34.7 (−66.2 to −3.1)	0.011
Multivariate model*	0	5.1 (−21.7 to 31.9)	−15.4 (−42.3 to 11.5)	−21.5 (−48.5 to 5.5)	0.041
FVC (ml)					
Age adjusted model	0	−5.9 (−43.8 to 32.1)	−26.0 (−63.8 to 11.8)	−47.1 (−85.3 to −8.9)	0.016
Multivariate model*	0	1.2 (−30.4 to 32.7)	−23.4 (−55.0 to 8.2)	−34.8 (−66.5 to −3.1)	0.010
FEV_1/FVC (%)					
Age adjusted model	0	0.1 (−0.4 to 0.5)	−0.1 (−0.6 to 0.4)	−0.1 (−0.6 to 0.3)	0.276
Multivariate model*	0	0.1 (−0.4 to 0.6)	0.0 (−0.5 to 0.5)	0.1 (−0.4 to 0.5)	0.911

FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity.
*Adjusted for centre, ethnicity, age, smoking status, pack-years of cigarette smoking, environmental tobacco smoke, body mass index, occupation, educational level, census tract-based income, height, square of height and background air pollution level.
†p Values for trend based on quartiles scaled by quartile medians.

Table 4 Associations between traffic density and FEV_1, FVC and FEV_1/FVC ratio in male ARIC participants (1987–9)

	Quartiles of traffic density				
	1 (lowest)	2	3	4	p Value†
FEV_1 (ml)					
Age adjusted model	0	58.9 (5.9 to 111.8)	19.2 (−34.1 to 72.5)	19.6 (−34.9 to 74.1)	0.664
Multivariate model*	0	21.2 (−23.7 to 66.0)	−11.2 (−56.7 to 34.2)	10.2 (−35.9 to 56.3)	0.994
FVC (ml)					
Age adjusted model	0	55.2 (−6.6 to 117.0)	13.2 (−49.0 to 75.5)	24.8 (−38.9 to 88.5)	0.956
Multivariate model*	0	16.4 (−34.0 to 66.7)	−21.9 (−73.0 to 29.2)	11.7 (−40.2 to 63.5)	0.856
FEV_1/FVC (%)					
Age adjusted model	0	0.3 (−0.3 to 1.0)	0.4 (−0.3 to 1.0)	0.3 (−0.4 to 0.9)	0.892
Multivariate model*	0	0.2 (−0.4 to 0.8)	0.2 (−0.5 to 0.8)	0.1 (−0.5 to 0.7)	0.936

FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity.
*Adjusted for centre, ethnicity, age, smoking status, pack-years of cigarette smoking, environmental tobacco smoke, body mass index, occupation, educational level, census tract-based income, height, square of height and background air pollution level.
†p Values for trend based on quartiles scaled by the quartile medians.

higher traffic density.[10] A cross-sectional Thai study reported that the FEV_1 and partial expiratory flow volume in 78 police in Bangkok (with high traffic exposure) was much lower than in 68 police in a rural area.[11] In a cross-sectional study of 4757 German women, living near a major road had a detrimental effect on lung function,[12] and in a cross-sectional study of 1986 Tokyo residents, pulmonary function did not vary with distance to roads but automobile exhaust was associated with respiratory symptoms.[14]

We found clearer associations in women than in men. Our measures of traffic exposure, traffic density and distance to major roads were inversely associated with lung function only in women. Few studies have reported stratified analyses of air pollution health effects by sex, and the patterns are not conclusive. In children, several studies[38 40 41] found stronger effects of air pollution on respiratory symptoms and lung function in girls than in boys. Conversely, in the Children's Health Study (CHS), the effect of traffic air pollution on lung development was stronger in boys than in girls.[42] Gehring *et al* also found a stronger effect of traffic air pollution on respiratory symptoms in boys than in girls.[43] In adults, studies in the USA and Switzerland found similar effects in both sexes,[35 44 45] whereas Abbey *et al* observed an association between lower lung function and long term exposure to particles only in men.[46]

Our finding of a stronger association of reduced lung function with greater traffic exposure in women is consistent with some studies[47–49] showing that women experience a greater smoking related decline in lung function than men, but not others.[50] Compared with men, women have slightly greater airway reactivity[51] so dose-response relations may be detected more easily. In addition, the validity of the exposure may vary by sex and could partly explain the observed sex difference in our data. At visit 1, 35% of women but only 17% of men reported being home makers, unemployed or retired. Thus, women may, on average, have spent more time at home than men. While we cannot confirm this with our data, this has been reported in other studies.[52] Our exposure assessment based on home address might therefore better reflect the true exposure level for women than for men.

We did not observe an effect of background air pollution on lung function. This is not surprising given that the ARIC study was not designed to examine air pollution and was limited to four communities. Furthermore, as these communities were not well supplied with air pollution monitors during the study period, little variation in air pollution within communities was captured by the available measurements.

Motor vehicle emissions, the principal source of ambient air pollution in most urban areas, are likely to vary substantially even within a given community. Research has documented differences in traffic related pollutants on a neighbourhood scale.[53] The traditional exposure assessment relying on a small number of monitors might not therefore estimate individual

Table 5 Associations between distance to major roads and FEV_1, FVC and FEV_1/FVC ratio in ARIC participants (1987–9)

		Dichotomised at 150 m			Dichotomised at 100 m		
		≥150 m	<150 m	p Value	≥100 m	<100 m	p Value
Women							
FEV_1 (ml)	Age adjusted model	0	−29.5 (−52.2 to −6.9)	0.011	0	−17.4 (−41.9 to 7.1)	0.165
	Multivariate model*	0	−15.7 (−34.4 to 2.9)	0.099	0	−17.2 (−37.5 to 3.2)	0.098
FVC (ml)	Age adjusted model	0	−33.2 (−60.4 to −5.9)	0.017	0	−10.2 (−39.8 to 19.3)	0.497
	Multivariate model*	0	−24.2 (−46.2 to −2.3)	0.030	0	−16.4 (−40.3 to 7.5)	0.178
FEV_1/FVC (%)	Age adjusted model	0	−0.1 (−0.5 to 0.2)	0.505	0	−0.3 (−0.7 to 0.0)	0.084
	Multivariate model*	0	0.1 (−0.3 to 0.4)	0.731	0	−0.2 (−0.5 to 0.2)	0.268
Men							
FEV_1 (ml)	Age adjusted model	0	−38.1 (−76.7 to 0.6)	0.054	0	−43.9 (−85.9 to −2.0)	0.040
	Multivariate model*	0	−6.4 (−38.1 to 25.3)	0.693	0	−18.5 (−53.2 to 16.2)	0.295
FVC (ml)	Age adjusted model	0	−17.0 (−62.0 to 28.0)	0.460	0	−27.7 (−76.5 to 21.1)	0.266
	Multivariate model*	0	10.9 (−24.7 to 46.5)	0.548	0	−7.9 (−46.8 to 31.1)	0.692
FEV_1/FVC (%)	Age adjusted model	0	−0.5 (−0.9 to 0.0)	0.058	0	−0.5 (−1.0 to 0.0)	0.052
	Multivariate model*	0	−0.3 (−0.7 to 0.2)	0.214	0	−0.3 (−0.7 to 0.2)	0.249

FEV_1, forced expiratory volume in 1 s; FVC, forced vital capacity.
*Adjusted for centre, ethnicity, age, smoking status, pack-years of cigarette smoking, environmental tobacco smoke, body mass index, occupation, educational level, census tract-based income, height, square of height and background air pollution level.

exposure levels adequately. In early epidemiological studies of the health effects of traffic exposure, the self-reported proximity of the home address to major roads[54] and self-reported road type and traffic density[55 56] were used. In contrast, we used objective measures of traffic related air pollution at residential addresses such as GIS based assessment of traffic density and distance to major roads.

The limitations of our analysis should be noted. Because the geocodes for participants' addresses at visits 1 and 2 (1987–92) were obtained from the TIGER file by Mapping Analytics, error could result from the use of older road network data. To assess this we randomly selected 100 participants from each of the ARIC communities and re-geocoded their residential addresses using the GDT software which incorporates a more recent road network database. Using these new geocodes, we re-calculated the traffic densities and distances to major roads and compared them with the original results. The two geocoding methods resulted in similar estimates for the distance to nearest major roads (data not shown). For traffic density, the two methods yielded quite concordant values for the Forsyth, Jackson and Minneapolis communities, but concordance was lower for Washington County. This might reflect a renaming of streets that occurred there. We therefore repeated our analyses excluding Washington County from our analysis. With the less precise exposure assessment in Washington County excluded, the associations of traffic exposure with FEV_1 and FVC became modestly stronger despite the reduced sample size (tables A11 and A12 in the online appendix available at http://thorax.bmj.com/supplemental). These additional analyses suggest that the association we observed is relatively robust to geocoding error.

We used a relatively simple proxy for traffic related air pollution (traffic density and distance to major roads) and did not attempt to model the concentrations of traffic related air pollutants or to validate our exposure assessment with measurements. Although recent data suggest stronger associations with stop-and-go traffic than moving traffic and with truck traffic compared with car traffic,[1] like most studies we could not classify traffic by type. We did not consider the acute effect of traffic exposure on lung function; however, given evidence that the association between the daily ambient level of air pollution and daily means of lung function is smaller in magnitude than the association between the long-term level of ambient air pollution and average lung function,[45 57] we suspect that any bias introduced by not controlling for the acute effect of traffic exposure would be minimal.

We lacked assessment of traffic related air pollution on approximately 11.6% of subjects (10.0% of women and 13.5% of men) whose addresses could not be geocoded. This raises concern about potential selection bias. However, when we compared pulmonary function measurements and demographic characteristics in ARIC participants with and without geocoded addresses (table A1 in the online appendix available at http://thorax.bmj.com/supplemental), we did not find significant differences. It is therefore unlikely that the data missing for 10.0% of women would have created the observed associations. Our exposure assessment was limited to residential address and we did not have information on duration of residence or on home exposures to other sources of pollutants such as cooking or heating. However, it is reassuring that an earlier study of ARIC participants reported very high concordance between county and state of residence in past decades to that at visit 1,[58] and in a study based on a sub-cohort of ARIC participants,[59] more than 60% of subjects had addresses at ages between 30 and 50 years that were assigned latitudes and longitudes almost identical to those associated with their visit 1 address.

As in any epidemiological study, residual confounding is possible. However, we adjusted for known and potential confounders including demographic characteristics, personal and neighbourhood level socioeconomic characteristics, cigarette smoking and background air pollution. The cross-sectional nature of our data, as well as initial non-response, is also a limitation of our data.

A major strength of our analysis is that it was based on a large community based cohort from four US communities. We also had an objective and quantitative respiratory outcome—namely, lung function. In addition, both exposure and outcome data were collected at the individual level together with extensive data on potential confounders. There is evidence that, in addition to individual level factors, the residential area or neighbourhood may have an additional effect on health.[60] Therefore, in the current analysis we also adjusted for a community level measure of socioeconomic status to help account for confounding.

In summary, in the ARIC study, higher exposure to traffic—as measured by traffic density and distance to roadways—was related to modestly reduced lung function in women. To our knowledge, this is the largest study of traffic exposure and pulmonary function to date. Our results add to growing evidence that chronic exposure to traffic related air pollution may adversely affect respiratory health.

ACKNOWLEDGEMENTS

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Further data are available in the appendix available online at http://thorax.bmj.com/supplemental.

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Competing interests: None.

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ORIGINAL ARTICLE

Road traffic noise and hypertension

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Background: It has been suggested that noise exposure increases the risk of hypertension. Road traffic is the dominant source of community noise exposure.
Objective: To study the association between exposure to residential road traffic noise and hypertension in an urban municipality.
Methods: The study population comprised randomly selected subjects aged 19–80 years. A postal questionnaire provided information on individual characteristics, including diagnosis of hypertension. The response rate was 77%, resulting in a study population of 667 subjects. The outdoor equivalent traffic noise level (Leq 24 h) at the residence of each individual was determined using noise-dispersion models and manual noise assessments. The individual noise exposure was classified in units of 5 dB(A), from <45 dB(A) to >65 dB(A).
Results: The odds ratio (OR) for hypertension adjusted for age, smoking, occupational status and house type was 1.38 (95% confidence interval (CI) 1.06 to 1.80) per 5 dB(A) increase in noise exposure. The association seemed stronger among women (OR 1.71; 95% CI 1.17 to 2.50) and among those who had lived at the address for >10 years (OR 1.93; 95% CI 1.29 to 2.83). Analyses of categorical exposure variables suggested an exposure–response relationship. The strongest association between exposure to traffic noise and hypertension was found among those with the least expected misclassification of true individual exposure, as indicated by not having triple-glazed windows, living in an old house and having the bedroom window facing a street (OR 2.47; 95% CI 1.38 to 4.43).
Conclusion: The results of our study suggest an association between exposure to residential road traffic noise and hypertension.

Noise acts as a ubiquitous stress-mediating factor in the physical environment. General annoyance, disturbances in psychosocial well-being and reduction in sleep quality are commonly reported effects of noise exposure.[1 2] An increased risk of non-auditory physiological effects due to noise, such as hypertension and ischaemic heart disease, have also been suggested.[3–8] Most previous studies have been performed in occupational settings with high noise levels.[5 6 8–10] Community noise is less well studied.

Road traffic is the dominating source of community noise in the urban environment. Few studies have investigated an association between exposure to road traffic noise and hypertension, and the results are conflicting.[3 4 11] Studies in this field have low precision and validity problems, including crude exposure assessments, selection bias and limited control of important confounding factors. Exposure has usually been assessed either from subjective reports or without consideration of important factors that may influence the individual exposure level—for example, window type, bedroom window orientation and type of residence.

The suggested biological mechanism for an association between exposure to community noise and hypertension is that noise induces stress by disturbing sleep and interfering with relaxation and concentration and many other cognitive effects that activate the sympathetic nervous system and the endocrine system.[12] The primary physiological effects of noise exposure are vegetative reactions such as increase in blood pressure, heart rate and finger pulse amplitude, cardiac arrhythmia, and changes in respiration and body movements.[13] Therefore, a hypothesis has emerged that stress due to persistent exposure to environmental noise could result in permanent vascular changes, with increased blood pressure and ischaemic heart disease as potential outcomes.[14–16]

Our objective was to study a possible association between exposure to residential road traffic noise and hypertension among adults in an urban municipality. To better characterise individual noise exposure, we aimed at investigating factors that may influence the true exposure level, such as window type, bedroom window orientation and type of residence.

METHODS

Study population

The study was performed in a municipality with 55 000 inhabitants located 15 km north of Stockholm City. A questionnaire designed for a countywide investigation of health effects related to various environmental factors was distributed in April 1997 to 1000 individuals aged 19–80 years living in the municipality.[17] A stratified random sampling procedure was applied to ensure a sufficient number of subjects exposed to traffic noise, consisting of two strata with 500 residents in each. The noise-exposed group was drawn from those living within 100 m on each side of the highway, main roads or the railway. The other sample was drawn from the remaining parts of the municipality. Statistics Sweden performed the sampling by combining the National Population Register (containing background information for the study population) with the Real Estate Register (containing geographical coordinates for the residence of each individual). The response rate was 77% in both samples. This study focused on exposure to road traffic noise; thus, subjects who were residing close to the railway (n = 91) were not included. One subject who had removed the identification sticker from the returned questionnaire was excluded, as we did not have the address of that subject. In total, the study comprised 667 subjects.

Questionnaire

The survey included 87 questions and was mainly focused on prevalence of allergic diseases and environmental risk factors of regional importance. Information on educational level, employment status, general living conditions, and smoking habits was

Cindy Moore

From: Dan and Karen Lovelace [danandkaren@verizon.net]
Sent: Tuesday, January 22, 2008 12:32 AM
To: Cindy Moore
Subject: Citrus Village Draft Neg Dec

Attachments: ROW Relinquish.pdf; Noncompliance issues Padre Shopping Center.doc; CVDMND01-08.doc


ROW Relinquish.pdf (906 KB)


Noncompliance issues Padre Sho...


CVDMND01-08.doc (51 KB)

Hi Cindy

Please accept the 3 attachments concerning the Citrus Village project.

Thank you,

Karen Lovelace

EXHIBIT B

Consideration for this conveyance lies in Grantee's promise to use and manage the real property herein (the "**PROPERTY**") as part of an affordable housing project operated in compliance with that certain document entitled Regulatory Agreement and Declaration of Restrictive Covenants made by and between the Redevelopment Agency of the County of Santa Barbara, and **GRANTEE,** recorded on February 1 2001 in the Official Records of Santa Barbara County as Instrument Number 2001-0007569 (the "**AGREEMENT**"). The **AGREEMENT** is hereby expressly incorporated herein by reference and this deed shall be interpreted in accordance with the terms and conditions herein and of the **AGREEMENT**. It is the express intent of **COUNTY** and **GRANTEE** that should the **PROPERTY** be used and/or managed in ways inconsistent with the **AGREEMENT**, that such **PROPERTY** shall be subject upon the option of **COUNTY,** its successors and assigns, to forfeiture by **GRANTEE,** its successors and assigns, and returned to **COUNTY**, or its successors and assigns. The following limitations shall operate as express conditions subsequent and shall be binding upon **GRANTEE,** its successors and assigns.

Should **GRANTEE** materially violate the provisions of the **AGREEMENT** or use the **PROPERTY** for purposes inconsistent with those specified herein, **COUNTY,** its successors or assigns shall have the right to re-enter said land and retake possession thereof and the estate granted herein shall be terminated and forfeited. Upon re-entry by **COUNTY,** its successors or assigns, all right, title, and interest to the **PROPERTY** shall vesting **COUNTY,** its successors or assigns. Such right of re-entry may be exercised by recording a notice of the re-entry in the office of the County Recorder of Santa Barbara.

Waiver of any breach of covenant, or the absence of action upon the failure of condition, shall not be deemed a waiver of any subsequent breach nor prevent the enforcement of **COUNTY'S** right of re-entry, and **COUNTY,** its successors or assigns may avail itself of all remedies in equity and law to protect the provisions of this deed and those of **AGREEMENT**.

This right of re-entry shall terminate upon the termination or expiration of the **AGREEMENT**.

Noncompliance issues related to adjacent Shopping Center:

Sec. 35-231.8. Setbacks for Buildings and Structures

Sec. 35-231.8. Side and Rear: 10 feet except:

2 a. 20 feet when a Convenience Shopping Center abuts a residential district

Sec. 35-231.12. Landscaping/Screening

3. Along each side or rear boundary abutting a residential district there shall be provided an ornamental masonry wall not less than six feet in height extending to within 20 feet of the street right-of-way line of existing or proposed streets, plus a row of trees which will provide continuous screening to an approximate height of not less than 20 feet nor more than 40 feet when mature.

5. Such landscaping shall be installed and maintained in accordance with the approved Final Development Plan.

Sec. 35-231.13. General District Regulations

2. Areas for trash shall be enclosed and architecturally screened in such a manner as to conceal all trash or stored material from public view.

Earl D. and Karen Lovelace
7372 Padova Drive
Goleta CA 93117
January 18, 2008

City of Goleta
130 Cremona Drive Suite G
Goleta CA 93117

RE: Citrus Village Draft Mitigated Negative Declaration, December 22, 2007

To Whom it May Concern:

We offer the following comments regarding the proposed project known as "Citrus Village". Firstly, as this project possesses a similar site plan, more building square footage and is not dissimilar in amenities other than the provision and siting of covered parking, all comments submitted by the public for the El Encanto Apartment Negative Declaration are pertinent to Citrus Village.

Commencing with the wording on page 3 under "Project Description" of the Citrus Village DMND, the applicant and staff take the potentially precedent setting position of presuming passage of amendments to the Goleta General Plan; amendments which are **proposed**, not approved.

Citrus Village, previously an approved 100% affordable housing project, now violates the following Housing Element provisions of the Goleta General Plan:

Policy HE 8: Preservation of Existing Housing and Neighborhoods [GP]
Objective: *Protect, conserve, and enhance the existing stock of housing and ensure that existing affordable housing at risk of conversion to market rates will remain affordable.*

HE 8.2 Condominium Conversions. [GP] Except when the effective vacancy rate for rental units averages 5 percent or more over the preceding 3 years, the City will, to the extent feasible under state law, conserve its existing multifamily rental housing by prohibiting conversions of rental developments to condominium ownership. Exceptions may be considered for limited-equity cooperatives and other innovative housing proposals that will be affordable to lower-income households.
HE 8.3 Protection of Existing Affordable Housing. [GP] The City will strive to ensure that all affordable housing, whether provided through government subsidy programs or incentives granted by the City or County in approving projects, deed restrictions, or City or County inclusionary requirements, will remain affordable for the longest term allowed by law. In its expenditures from the Affordable Housing Trust Fund and other actions, the City will give priority to preservation of existing affordable units where the County's affordability covenants or other regulatory agreements will be reaching the end of the term specified in those documents (see Appendix Table 10A-31, Approved and Existing Affordable Housing Units in Goleta, 2005)
Policy HE 8: Preservation of Existing Housing and Neighborhoods [GP]
Objective: *Protect, conserve, and enhance the existing stock of housing and ensure that existing affordable housing at risk of conversion to market rates will remain affordable.*
HE 8.1 Conversion of Conforming Residential Units. [GP] The City will discourage the

conversion of conforming residential units to nonresidential uses and regulate, to the extent permitted by law, conversion of rental housing developments to nonresidential uses to protect and conserve the rental housing stock.

HE 8.2 Condominium Conversions. [GP] Except when the effective vacancy rate for rental units averages 5 percent or more over the preceding 3 years, the City will, to the extent feasible under state law, conserve its existing multifamily rental housing by prohibiting conversions of rental developments to condominium ownership. Exceptions may be considered for limited-equity cooperatives and other innovative housing proposals that will be affordable to lower-income households.

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Policy HE 9: Excellence in New Housing Design [GP]

Objective: *Ensure that new housing is well designed to be compatible with and enhance Goleta's neighborhoods and the community as a whole.*

HE 9.1 Housing Design Process. [GP] The City will review proposed new housing to achieve excellence in site and building design and conduct design review in an efficient process consistent with the design principles articulated in the City's design review requirements.

HE 9.2 Design and Neighborhood Context. [GP] It is the City's intent that neighborhood identity and sense of community should be enhanced by designing all new housing to have a sensitive transition of scale and compatibility in form to the surrounding area. Buffers will be provided in mixed-use areas between residential and nonresidential uses.

HE 9.3 Housing Design Principles for Multifamily and Affordable Housing. [GP] The intent in the design of new multifamily and affordable housing is to provide stable, safe, and attractive neighborhoods through high-quality architecture, site planning, and amenities that address the following principles (see related Policy VH 4):

a. **Reduce the Appearance of Building Bulk.** Require designs that break up the perceived bulk and minimize the apparent height and size of new buildings, including the use of upper-story step-backs, variations in wall and roof planes, and landscaping. Application of exterior finish materials and trim, and windows and doors, for example, are important elements of building design and an indicator of overall building quality.

b. **Recognize Existing Street Patterns.** Incorporate transitions in height and setbacks from adjacent properties to respect adjacent development character and privacy. Design new housing so that it relates to the existing street pattern, creates a sense of neighborliness with surrounding buildings, and integrates pedestrian and bicycle systems.

c. **Enhance the "Sense of Place" by Incorporating Focal Areas.** Design new housing around natural and/or designed focal points that are emphasized through direct pedestrian and bicycle pathway connections. Site design and placement of structures shall include the maximum amount of usable, contiguous open space.

d. **Minimize the Visual Impact of Parking and Garages.** Discourage residential designs in which garages dominate the public façade of the residential building.

e. **Provide Buffers between Housing and Nonresidential Uses.** Ensure compatibility of residential and nonresidential uses by addressing parking and driveway patterns, transitions between uses, entries, site planning, and the provision of appropriate buffers to minimize noise, lighting, or use impacts.
f. **Maximize Privacy for Individual Units.** Site design, including placement of structures, pedestrian circulation, and common areas, as well as elements of architectural design such as, but not limited to, placement of windows, shall achieve a maximum degree of privacy for individual dwelling units within multifamily projects, including privacy for individual exterior spaces.
g. **Maximize Security and Safety.** Site and architectural design of multifamily residential projects shall emphasize principles of "defensible space," security for residents, and public safety and shall facilitate policing and observation by the City's police department from public streets and rights-of-way to the extent feasible.

HE 9.4 Resource Conservation. [GP] The City will promote development and construction standards that provide resource conservation by encouraging housing types and designs that use renewable and/or sustainable materials, cost-effective energy conservation measures, and fewer resources (water, electricity, etc.) and therefore cost less to operate over time. The City shall require individual residential units within multifamily housing projects to be separately metered for all utilities, including, but not limited to, water, natural gas, and electricity (see related Policy CE 13).

HE 9.5 Renewable Energy Technologies. [GP] Promote the use of sustainable and/or renewable materials and energy technologies, such as solar, in new and rehabilitated housing when possible (see related Policy CE 13).
Implementation Programs [GP]
IP-9A Prepare Residential Design Guidelines. Implement the Design Review process and prepare design guidelines/criteria that will establish effective, consistent development review factors for use by applicants, the community, staff, and decision makers in the review of housing proposals.
IP-9B Promote Solar Design. Develop design standards adapted to Goleta's climate relating to solar orientation, including lot layout for subdivisions, location and orientation of new structures, landscaping, fences, and impervious surfaces to conserve energy.
IP-9C Establish "Green" Building Standards and Processes. Adopt a "Green Building Program" to encourage the use of green building materials and energy conservation measures in new construction.

Note:
The approved El Encanto Apartments project was 16 very low-income affordable rental units. In terms of size, bulk and scale, it had far less impact on the surrounding neighborhood than Citrus Village (see plans).
Citrus Village offers little in comparison. The surrounding community consists of the adjacent Brookside condos at 8 units/acre. The vast majority of the units there are 2 bedroom 1.5 baths and approx 750 square feet. The few corner units are 3 bedroom 2 bath at 1050 square feet. The average square footage of the surrounding El Encanto Heights neighborhood single family residences are around 1300 square feet. It is a predominately single story community.
Citrus Village violates all design standards set forth, including second story setbacks. It must be reworked to meet health, safety and design standards:
Reduce ridge height (2 story can be easily accommodated well under 25 feet). **The photos provided by the applicant do not accurately portray visual impact.** The

applicant is asking for modifications. Modifications have potential added negative impact. It is the applicant's responsibility to prove otherwise. Allowing the developer to superimpose a drawing of the structures onto a photo is unacceptable. Story poles must be required for this project.

Eliminate chimneys-fireplaces consume additional space and energy unnecessarily. They draw attention to height and are unsightly in this context.

The architecture is incompatible with the surrounding community. Reduce the number and square footage of the units and the mass and scale of the structures to fit into the surrounding neighborhood.

Redesign the site plan to accommodate parking at the west side of the property. This allows use of existing Brookside landscape easement for the new development's open space and moves living quarters away from noxious, incompatible shopping center land use (gas station noises and fumes, 7-11).

Also, CA State law regulates minimum distance between bar/tavern establishments and residential. Padre Shopping Center has a bar in the NE corner. Bar patrons are frequently outside drinking and smoking. There will UNDOUBTEDLY be conflicts if these incompatible uses are sited too closely. The shopping center and its businesses should not be hampered because of lack of foresight and bad decisions-making on the part of the County Board of Supervisors. Make the best out of it though redesign and moving the living areas to the east..

Adhere to landscape buffer requirements where shopping centers border new residential - Inland Zoning Sec. 35-231.8, 35-231.12 and 35-231.13 (see attached)

Comply with emergency services access standards. Many exceptions and modifications were approved for El Encanto Apartments only because of extreme pressure to provide for the affordable units. The Citrus Village project obviously no longer meets those objectives.

IT IS NOT APPROPRIATE for an ingress/egress the width of a driveway to satisfy access to 11 large houses. Residents should not risk getting hit by a car exiting their front doors! Do not make this obvious, precedent-setting mistake, not here, not there, not anywhere!

Re-measure the plans: all components have been crammed in place with fewer than inches to spare. **Ensure that Calle Real roadway easement and encroachment into setbacks does not become the "Oops" factor.**

Correct the project description: that the project is 11 units on **40,877.55 square feet**, not 1.03 acres. The former applicant was granted a re-zone with the Calle Real ROW abandonment for the El Encanto Apartment project. The current applicant lost the easement (see attached) and therefore no longer has an acre. The ROW reverted to the citizens of Goleta. Where does this legally leave the DR zoning?

The history of this parcel requires that staff handle it with care.

It could easily become the next poster child for what went very wrong in the City of Goleta's new planning process.

Cindy Moore

From: Dan and Karen Lovelace [danandkaren@verizon.net]
Sent: Tuesday, January 22, 2008 5:28 PM
To: Cindy Moore
Subject: RE: Citrus Village Draft Neg Dec

Subject: Citrus Village Draft Neg Dec Comments due 01/22/08

Hi Cindy

Concerning the Citrus Village project:

Reclaimed water is available in the street @ Ellwood Station Road and Calle Real, ie next door to this project and should be required to tap into that supply.

Thank you,

Karen Lovelace

January 20, 2008

Cindy Moore
City of Goleta
130 Cremona Drive, Suite B
Goleta, CA 93117

Dear Cindy,

These are my comments on the Citrus Village Mitigated Negative Declaration.

This project was submitted under the current General Plan and should not be reviewed by any other standards. The discussion of any potential changes to the General Plan in the future is not appropriate for use on this project. The General Plan shouldn't be changed to fit projects, this and all projects should comply with the General Plan.

LU 1.10 still requires common open space and the project shouldn't be approved with lower standards. The project still needs to comply with building intensity standards. CE 10.3 is designed to keep runoff volumes from increasing. The proposed changes will increase runoff volumes and pollutants.

Final Development Plan
Page 5. It should be pointed out that a 28 foot wide right of way along Calle Real belongs to the City of Goleta. The developer has no authority to dedicate this property. The mention of the dedication of the right of way on page 9 should be deleted. This improperly appears to give legitimacy to the applicant's ownership of the right of way.

Page 9. The 5' 6" wall and 2' wide bioswale along the western property boundary are in the sideyard setback. The bioswale and drainage should not go through individual residences private patios. They should be placed in common open space. The size and number of units are not appropriate for this parcel. The units need to be scaled down because they are not compatible with the neighborhood.

Page 12. The request for seven modifications for only two affordable units isn't a reasonable request. The driveway, parking, setback, and landscaping requirements are needed. These provisions that protect the public safety should not be eliminated. It is obvious that there are too many units to fit onto the lot without causing numerous health and safety problems.

Aesthetics
This project will place an elevated row of very large, two story townhouses next to the Brookside condo complex. This will obscure any view to the west from the Brookside complex.

Page 29. When was the project site raised and leveled? Did the grading have the required permits?

Air Quality

Page 39. Health Risk Assessment. The project isn't far enough from the freeway to protect the residents health. There is no mention that, Calle Real, a two lane road is right at the front property line with its increased pollution.

Objectionable Odors. It is stated that "retail uses, which are not known to produce objectionable odors." There will be odors from the restaurant and the bar and grill, which could be objectionable. The bar and grill is located on the property line next to the tot lot and could pose health concerns due to patrons going outside to smoke. The gas station odors would be objectionable and unhealthy.

Page 40. AQ 1-1. There should be an additional sentence added to the second bullet on gravel pads. It has been used in recent EIRs. "If visible track-out results on any public roadway despite the use of such pads, the contractor shall cause the material to be removed by street cleaning within one hour of its occurrence and again at the end of the work-day." The following sentence should be added to the sixth bullet. "The name and phone number of the responsible individual shall also be posted on a sign with letter heights of at least 4.5 inches near the primary site access point." It is important that there is public access to the information when City and County staff is not available. This has also been incorporated into recently approved environmental documents.

Page 42. AQ 2-1. This mitigation should include a "Prohibition against the installation and use of wood burning fireplaces." This has been incorporated in recently approved environmental documents.

Page 43. It would seem there would be health concerns about using outdoor areas such as the tot lot due to the unmitigatable pollution from Highway 101, Calle Real, and the gas station next door.

Hazards and Hazardous Materials

Page 61. There is no discussion of the hazard posed by the existing gas station adjacent to the project. There are the risks of fuel, oil, and chemical spills as well as fires. Some of these units are only 10 feet from the property line.

Hydrology and Water Quality

Page 63. Has the site been graded since March 2005?

Page 64. The large amount of lot coverage with hardscape will increase the rate of runoff. The bioswales and drainage going through private backyards eliminates any certainty that they will be protected. Homeowners are likely to view the bioswales as just part of their yard that they can use for any purpose they want. There is no guarantee that they won't place storage, furniture, or other items in the bioswale.

Page 66. In the first sentence under Inundation the word "seiche" is used. This word should be changed so the meaning is understandable to the average person.

Page 67. HYDRO/WQ 1-2 Plan Requirements. The mitigation that must be done by the HOA needs to also be placed in the CC&Rs.

Page 68. HYDRO/WQ 1-3 The use of labels at stormdrains, indicating dumping waste is prohibited, will be of little value because the dumping is not done at the stormdrains. This is totally inadequate mitigation and not in compliance with the General Plan.

Public Services

Page 81. Schools. The elementary school that serves the project site is not El Rancho. El Rancho School has been closed for several years.

Table PS-1 should be updated.

Page 82. Fire Protection. This project further worsens public safety by adding additional population in western Goleta. This places western Goleta at increased danger due to the already seriously inadequate ratio of service. The residents of western Goleta have suffered for many years with inferior service. Simply contributing money does nothing to alleviate the problem. There is not even a viable site for a new fire station. The area shown in the General Plan has the Bluffs development, Santa Barbara Shores Park, and Sperling Preserve on one parcel and the other parcel, "Haskell's Landing", is currently processing a development plan for homes.
The design of the project with a 24' wide street with only one way in and out is not adequate. In the past the Fire Department has claimed they would not approve such unsafe design. The Fire Department continued the practice of caving in to developer pressure will put the residents of this project at increased danger.
The "no parking" on the 24' wide streets needs to be enforced through strong provisions and penalties established in the CC&Rs. HOA's all have a constant problem with people parking on 24'wide streets despite the "no parking" signs.
It is stated that the project "includes features that prohibit parking within the fire lane access-way." A red painted curb and "No parking" signs are not serious deterrents. It is a private street therefore the police will not enforce the signage.

Page 83. Table PS-2 needs to be corrected using the appropriate school.

Transportation/Traffic

Page 88. The trip generation numbers seem to be seriously under estimated. These units are larger than both the average condo/townhouse and than the homes in the immediate vicinity. They should be considered as single family residential units for trip generation numbers.

Page 90. Long Term Parking. The current parking shortage in this area is well known. Inadequate parking in an area already seriously impacted by lack of sufficient parking should not be permitted.

Page 91. TR 1-1. The painted curbs and "no parking" signs will not prevent parking along the main drive aisle. HOAs are limited in their ability to enforce parking regulations. It would help to have them in the CC&Rs. The police will not enforce parking regulations on private streets.

TR 2-1. Construction parking should not be allowed on the adjacent residential streets. The developer should provide private parking for construction workers and equipment. The public streets are not for construction site parking. The streets in the vicinity of the project are already seriously impacted by insufficient residential parking.

Page 98. SW 1-1. The Solid Waste Management Plan should include all listed measures. A location should be required on-site for composting. The local waste hauler already has a curbside recycling program.

Page 99. SW 1-3. Trash blowing around the construction site is always a problem and trash should be collected daily. The second sentence should read, "Waste shall be picked up on a daily basis and receptacles emptied weekly or more frequently as directed by City staff."

Nowhere in the document did I see a discussion of the safety issue created by lack of sidewalks. The only place to walk is in the street. Considering the lack of open space, a sidewalk should be added to the project.

This project has too many units of too large a size to fit on this parcel. The majority of units are larger than any of the single family homes in the neighborhood. The project should be scaled down so that the issues of aesthetics, air quality, public safety, stormwater runoff, parking, and neighborhood compatibility can be adequately addressed.

This document does not adequately discuss and mitigate all the environmental impacts of this project.

Barbara S. Massey
7912 Winchester Circle
Goleta, CA 93117

STATE OF CALIFORNIA—BUSINESS, TRANSPORTATION AND HOUSING AGENCY ARNOLD SCHWARZENEGGER, Governor

DEPARTMENT OF TRANSPORTATION
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Flex your power!
Be energy efficient!

January 10, 2008

Ms. Cindy Moore
City of Goleta Planning and Environmental Services
130 Cremona Dr #B
Goleta, CA 93117

05-SB-101- PM24.79

Subject: Citrus Village Draft Mitigated Negative Declaration

Dear Ms. Moore:

Thank you for the opportunity to review the mitigated negative declaration for the proposed Citrus Village project. Caltrans staff offers the following comments:

1. Page 88, Project specific impacts. The text indicates that a traffic analysis was submitted March 2005. This infers that data collection and level of service analysis occurred prior to that date. Traffic data and/or analysis should be no older than two years. Beyond that time, data should be refreshed otherwise ambient or existing conditions from which project impacts are judged, may not be accurately depicted.

2. In August 2007 the County of Santa Barbara released revisions to the Final EIR for the Isla Vista Master Plan. On page 10 (table 6.7-8) of that revision the baseline at Storke Road/Hollister Ave is presented as v/c 0.96 and LOS E. Although that report's "baseline" definition appears to be more in line with a cumulative condition, the transportation discussion for Citrus Village includes one reference to existing conditions at the Storke / Hollister intersection, but there is no substantive or quantitative discussion about project or cumulative impacts with respect build scenarios. The discussion concludes that payment of traffic impact mitigation fees reduces impacts to something less than significant. And while that may in fact be accurate, the narrative should discuss the intersections or road segments relevant to the project in terms of existing, existing plus project, cumulative conditions, and then if the data presents impacts, what the projects are that will mitigate the impacts, and how the subject project undergoing analysis fits into that scheme. At this time, that narrative does not capture entirely that discussion. The discussion about the US 101 / Glen Annie and Storke Road ramp intersection are similarly spartan.

3. Hydrology, Page 65, surface drainage post construction and page 68, HYDRO/WQ 2-1. As discussed in the text, without hydrologic calculations potential impacts related to alteration of on-site drainage patterns it is not known or clear what the impacts to neighboring properties will be, and that it may potentially be significant. US 101 is located directly south of Calle Real. The project proposes at least three drainage pipe systems from the property to outlet into the Calle Real gutter. Mitigation measure HYDRO/WQ 2-1 requires the execution of a drainage / stormwater management plan post-project approval. It is not known at this time if any of the flows will be directed to Caltrans hydraulic facilities or if the project's new flows

will be directed to El Encanto Creek. Caltrans requires that no net gain from development be introduced into existing facilities. Please ensure that the project flows do not impact these facilities.

I hope these comments provide your agency a better understanding of Caltrans' concerns with respect to this proposed project. If you have questions about this letter please contact me at (805) 549.3632.

Sincerely,

Chris Shaeffer
D5 Development Review

c: D. Murray (D5)
A. Orfila (SBCAG)