outdoor pollution mixture. Ozone in
England does not seem to play a role in
lung function.

Previous studies do not provide enough
evidence to relate the effects of air
pollution conclusively to one or the other
specific spirometric measures, although
there are some indications of larger effects
on the markers of the small airways.4
Forbes et al provide data only on FEV₁
and the FEV₁/FVC ratio, showing no
association for the ratio after adjusting
for smoking and social class. Larger
misclassification in the measurement of FVC
than FEV₁ in the general population
studies may explain these results in part.

The larger size and improved exposure
assessment in the study of Forbes et al
reinforce the knowledge of a deleterious
effect of current levels of air pollution on
lung health in Europe. However, the
cross-sectional nature of these studies does
not solve important questions regarding
the most relevant age period and exposure
time windows of susceptibility. It is impossible to know if the
effects on the lung function level in
adulthood reflect growth deficits experi-
enced during childhood and whether
these subjects entered the lung function
decline phase with a reduced lung func-
tion. The largest effects observed by
Forbes et al were among the oldest people
(>75 years). Nevertheless, this does not
imply an effect during the decline phase
since it could result from a cohort effect
because of higher historical air pollution
levels for this age group. It is possible that
air pollution behaves like smoking in
adulthood, which accelerates lung func-
tion decline, and that changes in smoking
resulted in changes in the slope of decline.
In any case, there is a need to extend
follow-up studies to children and to per-
form large follow-up studies through to
adolescence in order to quantify the
magnitude of the effect of air pollution in
accelerating lung function decline not
only for a better knowledge of the origins
of COPD, but also to assess the popula-
tion impact of air pollution and the potential
consequences of its reduction.

Competing interests: None declared.


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Can traffic-related air pollution cause asthma?

John R Balmes

Traffic-generated pollution contains par-
ticles and gases (eg, oxides of nitrogen)
that are known to have health effects.1
Concentrations of pollutants emitted by
motor vehicles are highest within 150 m
of roadways and remain raised up to
300 m from roadways, but fall off mark-
edly beyond that range.2 3 Exposure to
the mixture of traffic-generated pollutants
may be more relevant to human health
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Editorial

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pollution can be problematic. Exposure to
traffic can be estimated with complex
dispersion models of pollutants from local
freeway and non-freeway sources, but the
data inputs required for such modelling
are not always available. A frequently
used simpler approach has been to esti-
mate residential distance to roadways.

A number of studies have found an
increased prevalence of asthma or asthma
symptoms in children who live near
roadways with high traffic counts.4 8
One large British study that focused on
traffic within 150 m of children’s homes
found a gradient in risk that increased
markedly with decreasing residential dis-
tance to a main road.4 A large study in
southern California showed an increased
prevalence of asthma and wheeze in
children living within 75 m of a major
roadway.5 Another study by Jerrett et al
that analysed data from the same south-
ern California cohort was able to demon-
strate an association between the
incidence of asthma and exposure to
traffic-related pollution.9 A recent review
summarised the evidence for traffic pollu-
tion as a risk factor for both asthma
exacerbation and onset as strong.10

In contrast to the relatively rich litera-
ture for children, little has been published
on the effects of traffic-related pollution
on asthma in adults. Although several
previous studies in adults with asthma
have found that exposure to traffic—as
measured by distance of residence from
nearest major roadway—was associated
with asthma symptoms, health care uti-
lisation or decreased lung function,11–13
the study by Künzli and colleagues14
reported in this issue of Thorax is the first to show
convincing evidence that exposure to
traffic-related particulate matter increases
the risk of adult-onset asthma (see page
664). When the paper by Künzli et al is
taken together with the study by Jerrett et al,15 we now have evidence in both
children and adults that traffic-related
can cause as well as exacerbate

Given the robust effects observed on
asthma outcomes in other studies of both
children and adults, it is somewhat
surprising that distance to roadway was
not associated with the risk of new-onset
asthma in over 2700 non-smoking Swiss

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Pulmonary infection in Wegener’s granulomatosis and idiopathic pulmonary fibrosis

Nicholas Kim Harrison

Friederich Wegener’s original paper “On generalised septic vessel disease” suggests he thought likely there was an infectious cause for the condition which now bears his eponym.1 The characteristic pathological features of Wegener granulomatosis (WG) are: a necrotising granulomatous inflammation of the respiratory tract with vasculitis affecting both arteries and veins; focal necrotising glomerulonephritis; and a varying degree of systemic vasculitis—the so-called “Wegener’s triad”.2 The granulomatous inflammation is conspicuous for the absence of any obvious microorganism, although granulomatous infections can sometimes be misdiagnosed as WG.3 During the 70 years since Wegener’s description there have been some remarkable advances in both the diagnosis and treatment of this condition and in our understanding of its pathogenesis. However, the precise nature of the initiating factor(s) remains elusive.

What we do know is that there is a strong association between WG and the human leucocyte antigen (HLA)-DPBI*0401 allele, suggesting that there is an inherited predisposition for the condition.4 Interestingly, there is also an association with α-1 antitrypsin deficiency.5 We also know that virtually all patients who subsequently develop the systemic disease have circulating antineutrophil cytoplasmatic antibodies (ANCA)s and these are mainly directed against proteinase-3 (PR3), the so-called “Wegener’s autoantigen”—a serine protease which regulates cell proliferation, differentiation and death.6 This antibody has proved to be a useful biomarker for the diagnosis of WG.7 8 although less so for the likelihood of relapse.9 Furthermore, our increasing knowledge of its biological properties has provided new insights into the pathogenesis of WG. Specifically, whilst PR3 is the target antigen for ANCAs, the observation that elevated levels of PR3 at sites of granulomatous inflammation correlate with increased tumour necrosis factor α (TNFα) has led to the hypothesis that PR3 is directly involved in the modulation of cytokines associated with an aberrant immune response (see Csernok et al10 for a review). Following tissue injury, increased levels of PR3, released from activated or dying neutrophils, might act as a danger/alarm...

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