Case Western Reserve University

Statistics
Good for your Health!!

Nancy Reid

September 30, 2004
Eating smart is good for your brain
Research is piling up that getting certain nutrients can help you concentrate and learn today, and ward off brain aging in the long run

By LESLIE BECK
Wednesday, September 8, 2004 - Page A17
Some recent headlines

Study finds omega-3 can ward off Alzheimer's

By ANDRÉ PICARD
PUBLIC HEALTH REPORTER; With a report from Erin Pooley
Friday, September 3, 2004 - Page A1
PERSONAL HEALTH; Must I Have Another Glass of Water? Maybe Not, a New Report Says

By JANE E. BRODY (NYT) words
Late Edition - Final, Section F, Page 7, Column 1
TOXICOLOGY

Salmon Survey Stokes Debate About Farmed Fish

Salmon’s popularity has boomed in the past 2 decades as aquaculture has made salmon available year-round at low cost. The fish is a good source of protein, vitamin D, and heart-friendly fats. But fish farms have boosted less welcome ingredients: The largest survey yet of pollutants in salmon, reported on page 226, has found that farmed fish have higher levels of polychlorinated biphenyls (PCBs) and other organochlorine compounds than do wild-caught salmon. The source, as many researchers suspected, is the feed.

“This is a definitive study,” says nutritionist and toxicologist Miriam Jacobs of the University of Surrey, U.K., and Royal Veterinary College, London. “Further action has to be taken to reduce the contaminant levels in feed.” The authors argue that consuming more than one meal of farmed salmon per month may hike the risk of cancer. “The punch line is that eating the wrong kind of fish has real dangers,” says team member David Carpenter of the State University of New York, Albany, in Rensselaer.

Other experts say the risk is outweighed by the benefits of eating farmed salmon. Avoiding the fish would mean giving up its nutritional benefits, including protection against heart attacks. What’s more, they say, the contaminant levels aren’t high enough to pose real dangers. “In my view, the study says we should be eating more farmed salmon,” says toxicologist Charles Santerre of Purdue University in West Lafayette, Indiana.

While nutritionists debate the study’s implications, consumers can use its data to try to select the cleanest fish possible. “I think we can begin to make informed choices about what kind of fish to eat,” says toxicologist Linda Birnbaum of the U.S. Environmental Protection Agency (EPA).

The massive study, funded by the Pew Charitable Trusts’ Environment program and conducted by six scientists, sampled about 700 salmon from around the world and analyzed them for more than 50 contaminants. The greatest difference between farmed and wild salmon was in organochlorine compounds. Thirteen of these chemicals, farmed salmon were more contaminated than wild ones. Farmed salmon in Europe had the highest levels, followed those from North America, whereas Chilean salmon were the cleanest. Researchers also tested fish oil and meal fed to the salmon and found a similar pattern. Feeding salmon fish meal boosts growth and nutrition, but it also concentrates contaminants.

The team took a closer look at PCBs and other persistent pesticides called dioxins and toxaphene, all of which have been related with risk of liver and other diseases. The researchers used EPA guidelines to calculate the maximum amount of fish that can be eaten before boosting potential risk. In most cases, the quantity far exceeded the daily recommended amount of fish for two analyses they published earlier this year. 

Seeing red. Farmed salmon has more PCBs than wild salmon, but scientists don’t agree on how much one should eat.
Highlights From This Issue

A Centennial of Independence

Henri Rousseau, A Centennial of Independence, 1892, French.
MORE >

Diet and Lifestyle vs Death and Vascular Disease

Knoops and colleagues report results of a cohort study examining the contributions of a Mediterranean diet, moderate physical activity, moderate alcohol use, and nonsmoking to mortality in healthy elderly individuals. FREE ARTICLE
MORE >

Esposito and colleagues describe results of a randomized trial of patients with the metabolic syndrome who were allocated to a Mediterranean-style diet or a prudent diet of similar macronutrient composition and featuring healthful food choices.
MORE >

Physical Activity and Cognitive Function

Abbott and colleagues analyzed the association between walking and future risk of dementia in physically capable men aged 71 to 93 years.
MORE >

Weuve and colleagues report their analyses of the relationship of long-term regular physical activity to cognitive function in women aged 70 to 81 years.
MORE >

CLINICIAN'S CORNER CME COURSE
Family Cancer History

An evidence-based analysis of the accuracy of self-reported family cancer history.
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Take the CME Course.
A new analysis of Franklin Delano Roosevelt’s symptoms suggests he might not have been stricken by polio but by Guillain-Barré syndrome.

In 1921, at the beginning of his political career, Roosevelt became feverish and developed paralysis, which started in his legs and moved up to his neck. Although he recovered partially, he remained permanently wheelchair-bound.

Immunological pediatrician Armond Goldman of the University of Texas Medical Branch in Galveston now says FDR’s symptoms are more concordant with Guillain-Barré syndrome, a bacterially induced autoimmune disease. For example, the disorder is linked to influenza.

Emerged as the more likely cause of his paralysis, they report in the 1 November Journal of Medical Biography.

“The result is interesting both historically and neurologically,” says neurologist Deborah Green of the University of Hawaii School of Medicine at Manoa. FDR’s misdiagnosis—if such it was—may have changed the course of history, because his affliction gave great momentum to efforts to develop a polio vaccine. But Green notes that “there’s no way to prove [a misdiagnosis] without testing the spinal cord fluid.” Neurologist H. Royden Jones of Harvard Medical School in Boston adds that the researchers could be wrong in assuming that “Guillain-Barré is the same now as it was back then.”
Statistical error leaves pollution data up in the air

Jonathan Knight, San Francisco
An off-the-shelf statistics package has tripped up pollution researchers in North America and Europe who are studying the effects of airborne soot on human health.

A default setting that produced erroneous results went unchecked for years, despite significant statistical expertise in all of the groups. "It was already such standard software when we started using it, I didn't even question it," says Francesca Dominici, a public-health researcher at Johns Hopkins University in Baltimore, Maryland.

On 4 June, Dominici posted revised figures on her website after discovering that the error had doubled her group's estimate of the risk to health posed by particulates in the air. Two other groups that used the same tool, one in Canada and one in Greece, are now redoing their calculations.

The groups were looking for correlations between death rates and particulates in the air, which come mainly from diesel engines and power plants. Their data on air quality, hospitalizations and deaths from dozens of cities cover a seven-year period up to 1994.

Death rates vary throughout the year because of such factors as changes in temperature and disease outbreaks. To tease out the effects of particulates, the groups used a statistics program known as S-Plus.

S-Plus searches for correlations using an iterative process in which confounding effects are gradually peeled away. The default parameter in question determined how many times the procedure would iterate before stopping to produce a final result.

"For most applications the value is perfectly fine," says David Smith, product manager of Seattle-based Insightful, which sells S-Plus. Smith says that the Hopkins case was exceptional, but that users should always check whether changing the parameter affects the outcome, and adjust it if necessary. Smith says that Insightful will tighten the default value of the parameter — slowing the programme slightly — on future releases of S-Plus.

Richard Burnett, a statistician with Health Canada in Ottawa, which is conducting a similar study, says that his group will probably revise its estimates of the impact of airborne soot on mortality downwards by 20–50%. The findings of a study run by a group at the University of Athens may also have to be adjusted, he says.

The health risk posed by particulates is a source of fierce environmental controversy in the United States, and the Bush administration is considering rules to restrict emissions. Opponents of tighter rules are likely to seize on the revisions as evidence that the research linking soot in the air to harmful effects on health is not to be trusted.
Data Revised on Soot in Air and Deaths

Scientists Lower Their Estimate of Risk From Bad-Air Days

By ANDREW C. REVKIN

Revisiting their own data with new methods, scientists who conducted influential studies that linked sooty air pollution with higher death rates have lowered their estimate of the risk posed by bad-air days.

The findings do not challenge what is now a well-established link between air pollution and premature death. But the new analysis is highly likely to delay the final review of new regulations on small-particle pollution, officials of the Environmental Protection Agency said yesterday.

"This may clearly push it beyond that," a spokesman for the E.P.A., Joe Martyak, said last night.

One of the major conclusions of the new study is that it significantly lowers the estimate of the risk posed by bad-air days.

The fine particles in question come mainly from power plants and diesel engines, and the proposed rules have been at the center of a long legal, political and public-relations battle between private environmental groups and power plant owners and vehicle manufacturers.

The researchers at the Johns Hopkins University, have been distributing their new analysis to scientists and government officials by fax and e-mail. Yesterday, they set up a Web site, bioair01.biostat.jhsph.edu/tdominic/research.html, that details their new findings.

"This is a very important finding that needs to be probed," said Daniel S. Greenbaum, president of the Health Effects Institute, the organization that conducted the study.

New research may delay a review due next year on small-particle pollution.

J. M. Samet, chairman of epidemiology at the public health school there.

As part of a continuing effort to check for flaws, those scientists in recent weeks used a new method to find particles that can be deeply inhaled into the lungs and stay there. In the original analysis, the rise was 0.4 percent above the typical mortality rate for each jump of 10 micrograms of soot per cubic meter of air. In the new analysis, the increase is half that.

The researchers said the change was small but significant. The average level in the average city is now about 24 micrograms a cubic meter.

The work has been published for several years in a variety of leading journals like The New England Journal of Medicine and The American Journal of Epidemiology. The project, the National Morbidity, Mortality and Air Pollution Study, was given extra weight by policy makers because of the reputation of the Health Effects Institute and the Johns Hopkins group, led by Dr. Jonathan M. Samet, chairman of epidemiology at the public health school there.

Last year, the institute concluded that the average level of soot was 10 micrograms per cubic meter, compared with 24 micrograms per cubic meter for the average city.

Scientists involved with the soot standard said that there was much other evidence that pointed to the dangers of soot but that the errors in the Johns Hopkins work were still significant.

"It certainly brings into question the precision of the data," said Dr. Jane Q. Koek, a professor of environmental health at the University of Washington and a consultant involved with the soot review. "That's very unfortunate, because this research was supposed to be the result of careful study, and instead it has come out with a significantly lower level of risk."
Did FDR have Guillain-Barré syndrome?

“What was the cause of Franklin Delano Roosevelt’s paralytic illness?” Goldman, et al. J Medical Biography 2003
Did FDR have Guillain-Barré syndrome?

Table 2. Diagnostic probabilities of eight key symptoms in Roosevelt’s paralytic illness appearing in Guillain–Barpoiomyelitis, tested by Bayesian analysis

<table>
<thead>
<tr>
<th>FDR’s case</th>
<th>GBS (prior probability 0.51)</th>
<th>Poliomyelitis (prior)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Symptom probability</td>
<td>Posterior probability</td>
</tr>
<tr>
<td>Paralysis ascends for 10–13 days</td>
<td>0.70</td>
<td>0.36</td>
</tr>
<tr>
<td>Facial paralysis</td>
<td>0.50</td>
<td>0.26</td>
</tr>
<tr>
<td>Bladder/bowel dysfunction for 14 days</td>
<td>0.50</td>
<td>0.26</td>
</tr>
<tr>
<td>Numbness/dysaesthesia</td>
<td>0.50</td>
<td>0.26</td>
</tr>
<tr>
<td>No meningismus</td>
<td>0.99</td>
<td>0.50</td>
</tr>
<tr>
<td>Fever</td>
<td>&lt;0.01</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Descending recovery from paralysis</td>
<td>0.70</td>
<td>0.36</td>
</tr>
<tr>
<td>Permanent paralysis</td>
<td>0.15</td>
<td>0.08</td>
</tr>
</tbody>
</table>

The derivation of the estimates of prior probabilities (relative frequencies of the diseases in FDR’s age range probabilities (the chance that a clinical feature occurred in a disease) of poliomyelitis and GBS is given in the considerations”. Posterior probabilities (the probability that FDR’s symptoms were due to a disease) are the symptom probabilities. Greater posterior probabilities are in bold type.
Did FDR have Guillain-Barré syndrome?

<table>
<thead>
<tr>
<th>Symptom</th>
<th>GBS Symptom Prob</th>
<th>GBS Posterior Prob</th>
<th>Polio Symptom Prob</th>
<th>Polio Posterior Prob</th>
</tr>
</thead>
<tbody>
<tr>
<td>ascending paralysis</td>
<td>0.70</td>
<td>0.36</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>facial paralysis</td>
<td>0.50</td>
<td>0.26</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>bladder/bowel</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dysfunction</td>
<td>0.50</td>
<td>0.26</td>
<td>0.05</td>
<td>0.02</td>
</tr>
<tr>
<td>numbness</td>
<td>0.50</td>
<td>0.26</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>no meningismus</td>
<td>0.99</td>
<td>0.50</td>
<td>0.10</td>
<td>0.04</td>
</tr>
<tr>
<td>fever</td>
<td>&lt; 0.01</td>
<td>&lt; 0.01</td>
<td>0.90</td>
<td>0.35</td>
</tr>
<tr>
<td>descending recovery</td>
<td>0.70</td>
<td>0.36</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>permanent paralysis</td>
<td>0.15</td>
<td>0.08</td>
<td>0.50</td>
<td>0.20</td>
</tr>
</tbody>
</table>

Prior probability 0.51 for GBS, 0.39 for polio
Did FDR have Guillain-Barré syndrome?

Bayes theorem

“using a retrospective analytical technique devised by the Reverend Thomas Bayes (1702–1761) and published in 1763, two years after his death”

\[
Pr(disease \mid symptom) \propto Pr(symptom \mid disease)Pr(disease)
\]

each symptom treated separately, for example

\[
Pr(GBS \mid facial paralysis) \propto Pr(facial paralysis \mid GBS)Pr(GBS)
\]

\[
\begin{array}{cc}
0.50 & 0.51 \\
0.02 & 0.01
\end{array}
\]
Did FDR have Guillain-Barré syndrome?

Bayes theorem

“using a retrospective analytical technique devised by the Reverend Thomas Bayes (1702–1761) and published in 1763, two years after his death”

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Pr(\text{disease} \mid \text{symptom}) \propto Pr(\text{symptom} \mid \text{disease}) Pr(\text{disease})
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each symptom treated separately, for example

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Pr(\text{GBS} \mid \text{facial paralysis}) \propto Pr(\text{facial paralysis} \mid \text{GBS}) Pr(\text{GBS})
\]

\[
\begin{array}{c|c|c}
& & \\
\text{GBS} & 0.51 & 0.50 \\
\text{facial paralysis} & 0.01 & 0.02
\end{array}
\]

\[
Pr(\text{guilty} \mid \text{evidence}) \propto Pr(\text{evidence} \mid \text{guilty}) Pr(\text{guilty})
\]

\[
Pr(A \mid B) \propto Pr(B \mid A) Pr(A)
\]
### Did FDR have Guillain-Barré syndrome?

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<thead>
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<th>GBS Posterior Prob</th>
<th>Polio Symptom Prob</th>
<th>Polio Posterior Prob</th>
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</thead>
<tbody>
<tr>
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<td>0.70</td>
<td>0.98</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>facial paralysis</td>
<td>0.50</td>
<td>0.97</td>
<td>0.02</td>
<td>0.03</td>
</tr>
<tr>
<td>bladder/bowel</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>dysfunction</td>
<td>0.50</td>
<td>0.93</td>
<td>0.05</td>
<td>0.07</td>
</tr>
<tr>
<td>numbness</td>
<td>0.50</td>
<td>0.99</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
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<td>0.90</td>
<td>0.99</td>
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<td>0.70</td>
<td>0.98</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>permanent paralysis</td>
<td>0.15</td>
<td>0.28</td>
<td>0.50</td>
<td>0.72</td>
</tr>
</tbody>
</table>

Prior probability 0.56 for GBS, 0.44 for polio
Table 1. Clinical features of Franklin D Roosevelt’s case compared with those of Guillain–Barré syndrome (GBS) and poliomyelitis

<table>
<thead>
<tr>
<th>Clinical features</th>
<th>Roosevelt’s case</th>
<th>GBS</th>
<th>Poliomyelitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age of onset</td>
<td>39 years</td>
<td>Mainly adults</td>
<td>Mainly young children</td>
</tr>
<tr>
<td>Flaccid paralysis</td>
<td>Symmetric, ascending</td>
<td>Symmetric, ascending</td>
<td>Asymmetric</td>
</tr>
<tr>
<td>Progress of paralysis</td>
<td>10–13 days</td>
<td>10–14 days</td>
<td>3–5 days</td>
</tr>
<tr>
<td>Facial paralysis</td>
<td>Present</td>
<td>Common, bilateral</td>
<td>Rare, save in bulbar type</td>
</tr>
<tr>
<td>Bladder/bowel dysfunction</td>
<td>14 days</td>
<td>7–14 days</td>
<td>1–3 days</td>
</tr>
<tr>
<td>Numbness</td>
<td>Present</td>
<td>Common</td>
<td>Absent</td>
</tr>
<tr>
<td>Dysaesthesia</td>
<td>Protracted</td>
<td>Protracted</td>
<td>Absent</td>
</tr>
<tr>
<td>Meningismus</td>
<td>Absent</td>
<td>Absent</td>
<td>Common</td>
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<td>Rare</td>
<td>Common</td>
</tr>
<tr>
<td>Recovery from paralysis</td>
<td>Symmetric, descending</td>
<td>Symmetric, descending</td>
<td>Asymmetric</td>
</tr>
<tr>
<td>Permanent paralysis</td>
<td>Symmetric</td>
<td>In about 15% of cases</td>
<td>In about 50% of cases</td>
</tr>
</tbody>
</table>

*The clinical features of poliomyelitis and GBS have been drawn from many past publications.*

*This data is summarized from various sources, including medical journals and historical records. The comparison is intended to highlight similarities and differences between the clinical presentations of these conditions.*
“... it was unlikely that GBS would have been considered in 1921 since the characteristic cerebrospinal fluid findings in that disease were first recognized only in 1916”
Diet and Lifestyle vs Death and Vascular Disease

The relationship of a Mediterranean-style diet to disease outcomes is the subject of 2 articles in this issue of JAMA. First, Knoops and colleagues (SEE ARTICLE) report results of a cohort study examining the contributions of a Mediterranean diet, moderate physical activity, moderate alcohol use, and nonsmoking to mortality in healthy elderly individuals. The authors found that individuals who reported at least 2 of the lifestyle factors had significantly reduced all-cause and cause-specific mortality rates compared with individuals who reported one or none. In a second article, Esposito and colleagues (SEE ARTICLE) describe results of a randomized trial of patients with the metabolic syndrome who were allocated to a Mediterranean-style diet or a prudent diet of similar macronutrient composition and featuring healthful food choices. Patients in the Mediterranean-style diet group had greater weight loss, larger reductions in levels of thrombosis-associated inflammatory markers, and reduced prevalence of the metabolic syndrome. In an editorial, Rimm and Stampfer (SEE ARTICLE) discuss the accumulated evidence and questions still in need of investigation related to primary disease prevention through maintenance of a healthful lifestyle.
Study 1 Knoops et al. JAMA 2004;292:1433–1439

- A cohort study: 1507 apparently healthy men and 832 women, aged 70 to 90 years, 11 European countries.
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- “Adhering to a Mediterranean diet: hazard ratio HR 0.77 (0.68 – 0.88)
- “Four low-risk factors: HR 0.35 (0.28 – 0.44)
## The Mediterranean diet

<table>
<thead>
<tr>
<th>Causes of death</th>
<th>All</th>
<th>chd</th>
<th>cv</th>
<th>cancer</th>
<th>other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mediterranean diet</td>
<td>0.77</td>
<td>0.61</td>
<td>0.71</td>
<td>0.90</td>
<td>0.61</td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>0.78</td>
<td>0.60</td>
<td>0.74</td>
<td>0.73</td>
<td>0.63</td>
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<tr>
<td>Physical activity</td>
<td>0.63</td>
<td>0.72</td>
<td>0.64</td>
<td>0.64</td>
<td>0.52</td>
</tr>
<tr>
<td>Nonsmoking</td>
<td>0.64</td>
<td>0.80</td>
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<td>0.92</td>
</tr>
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Hazard ratio: \[ \frac{Pr(\text{die tomorrow} \mid \text{at low risk})}{Pr(\text{die tomorrow} \mid \text{at high risk})} \]
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**Hazard ratio:**

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### Causes of death

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### Hazard ratio:

\[
\text{Hazard ratio: } \frac{Pr(die \ tomorrow \mid \text{ at low risk})}{Pr(die \ tomorrow \mid \text{ at high risk})}
\]
Study 2 Esposito et al. JAMA 2004;292:1440–1446

- randomized, single blind trial: 180 patients with metabolic syndrome, U Naples
Study 2 Esposito et al. JAMA 2004;292:1440–1446

- randomized, single blind trial: 180 patients with metabolic syndrome, U Naples
- conducted between June 2001 and January 2004

Patients consuming the intervention diet had significantly reduced serum concentrations of hs-CRP, IL-6, IL-7, IL-18, decreased insulin resistance, improved endothelial function score.

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The Mediterranean diet

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| weight | 78 74 | 76 75.8 |

Nancy Reid: Statistics, Good for your Health!!
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Rothman and Greenland: *Modern Epidemiology*
Data Revised on Soot in Air and Deaths

Scientists Lower Their Estimate of Risk From Bad-Air Days

By ANDREW C. REVKIN

Revisiting their own data with new methods, scientists who conducted influential studies that linked sooty air pollution with higher death rates have lowered their estimate of the risk posed by bad-air days.

The findings do not challenge what is now a well-established link between air pollution and premature death. But the new analysis is highly likely to delay the final review of new regulations on small-particle pollution, officials of the Environmental Protection Agency said yesterday.

The review was projected to end, and the new rules to take effect, by the end of next year.

"This may clearly push it beyond that," a spokesman for the E.P.A., Joe Martyak, said last night.

The fine particles in question come mainly from power plants and diesel engines, and the proposed rules have been at the center of a long legal, political and public-relations battle between private environmental groups and power plant owner and vehicle manufacturers.

The researchers, at the Johns Hopkins University, have been distributing their new analysis to scientists and government officials by fax and electronic means.

New research may delay a review due next year on small-particle pollution.

By ANDREW C. REVKIN

Doctors who find themselves at the intersection of science and public policy often have to deal with new findings that can be deeply inhaled into the lungs and stay there. In the original analysis, the rise was 0.4 percent above the typical mortality rate for each jump of 10 micrograms of soot per cubic meter of air. In the new analysis, the increase is half that.

The researchers said the change was small but significant. The average level in the average city is now about 24 micrograms a cubic meter.

The work has been published for several years in a variety of leading journals like The New England Journal of Medicine and The American Journal of Epidemiology. The project, the National Morbidity, Mortality and Air Pollution Study, was given extra weight by policy makers because of the reputation of the Johns Hopkins group, led by Dr. Jonathan M. Samet, chairman of environmental epidemiology at the public health school.

It certainly brings into question the precision of the data," said Dr. Jane O. Koenig, a professor of environmental epidemiology at the Harvard School of Public Health. She noted that the researchers used data collected by sources other than government agencies, which meant that they could not be sure that the levels were from air pollution and not other sources.

"The researchers have worked really hard on this, and they have produced a lot of useful information," Dr. Koenig said. "But it's important to remember that the data are not perfect, and that the conclusions are not settled.

Dr. Zeger and Mr. Greenbaum stressed that the new findings did not overturn the basic link between soot and illness. They also pointed to the recent publication of other studies on the long-term effects of soot that do not use the same analytical tools.

Still, industry officials said they looked into question the validity of some research underlying the new federal standards.

"This study is really one of the ones creating the path for the future on air-quality regulation," said Allen Schaeffer, executive director of the Diesel Technology Forum.

The new results, Mr. Schaeffer said, show that "particle science is still evolving, and so are the analytical tools to look at it."

Scientists involved with the soot standard said that there was much other evidence that pointed to the dangers of soot but that the errors in the Johns Hopkins work were still systematic.

"It should not be discounted as a substitute for policy," said Dr. Samet. "It's just that it's not as precise as it should be, and it's not as clear as it should be."

The researchers said that they were working on recalibrating their data to take into account the new findings.
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Health effects of air pollution

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The health risk posed by particulates is a source of fierce environmental controversy in the United States. Opponents of tighter rules are likely to seize on the revisions as evidence that the research linking soot in the air to harmful effects on health is not to be trusted. A default setting that produced erroneous results went unchecked for years, despite significant statistical expertise in all the groups.
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these are small effects; approximately 3 additional deaths per year in Cleveland, perhaps 15 in Toronto
The National Morbidity, Mortality, and Air Pollution Study
Part II: Morbidity and Mortality from Air Pollution in the United States

Jonathan M. Samet, Scott L. Zeger, Francesca Dominici,
Frank Curriero, Ivan Coursac, Douglas W. Dockery,
Joel Schwartz, and Antonella Zanobetti

Includes a Commentary by the Institute’s Health Review Committee
Health effects of air pollution

The data
- daily mortality counts from NCHS (National Center for Health Statistics) 1987-1994

The model
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- fit the same model for each city, pool effect estimates
... the model

\[ \log \mu_{at} = \beta X_{t-1} + \gamma DOW + S_1(t, 7) + S_2(\text{temp}_0, 6) + S_3(\text{temp}_{1-3}, 6) \]

\[ + S_4(\text{dew}_0, 3) + S_5(\text{dew}_{1-3}, 3) + \alpha_a + S_6a(t, 8) \]

- \( a \) indexes age groups, \( t \) time (days)
- \( S(z, 8) \) is a non-specified, but smooth, function of \( z \) with 8 ‘degrees of freedom’, can think of it as a spline with a pre-specified number of knots. Large df means wiggly function, 1 df is linear
- mortality rates change with season, weather, changes in health status, ...
- Is there anything left for pollution?
Health effects of air pollution

Population studies

– use pollution measurements at monitoring stations
– use city-wide mortality rates
– use relatively crude measures of weather
– are ‘ecological’ studies
– what is the real dose to an individual?
– are there ‘effect modifiers’ (e.g. average income, average age)
– what about micro-climates?
– what are the mechanisms?
– (note these are acute, not chronic, effects)
– (note gaseous pollutants)
consistency of evidence
biological plausibility
temporality
dose response
Statistics is good for your health!!

- Some recent headlines
- Did FDR have Guillain-Barré syndrome?
- The Mediterranean diet
- Health effects of air pollution
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“A statistician? How very essential. One is, after all, a statistic all of one’s life, isn’t one?”