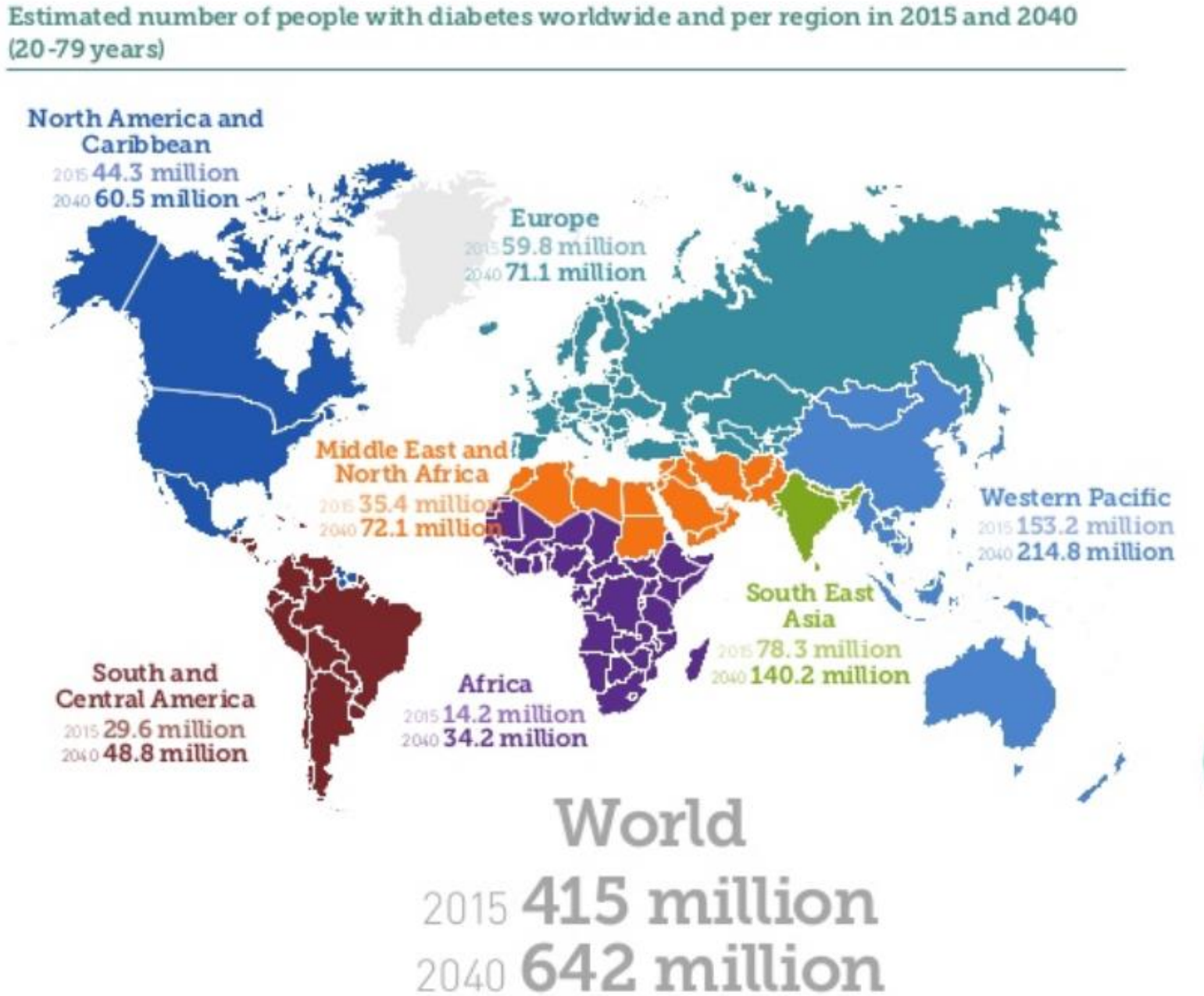
Wpływ zanieczyszczenia powietrza na ryzyko wystąpienia zaburzeń węglowodanowych



https://www.slideshare.net/DRVENEREO/versin-previa-del-atlas-idf-2015

The prevalence of diabetes

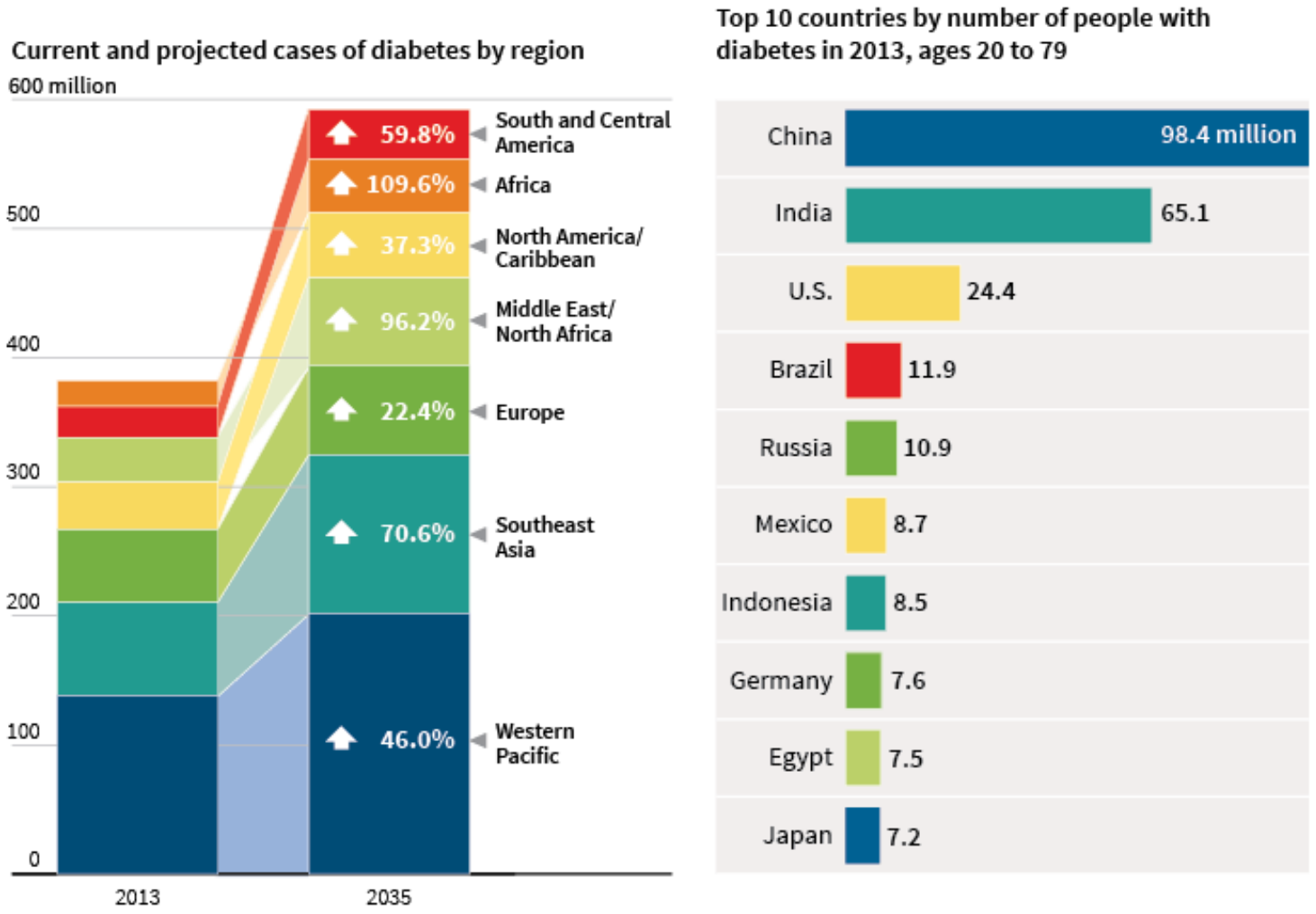
2015

One in **11 adults** has diabetes

2040

One in **10 adults** will have diabetes

World diabetes cases expected to jump 55 percent by 2035

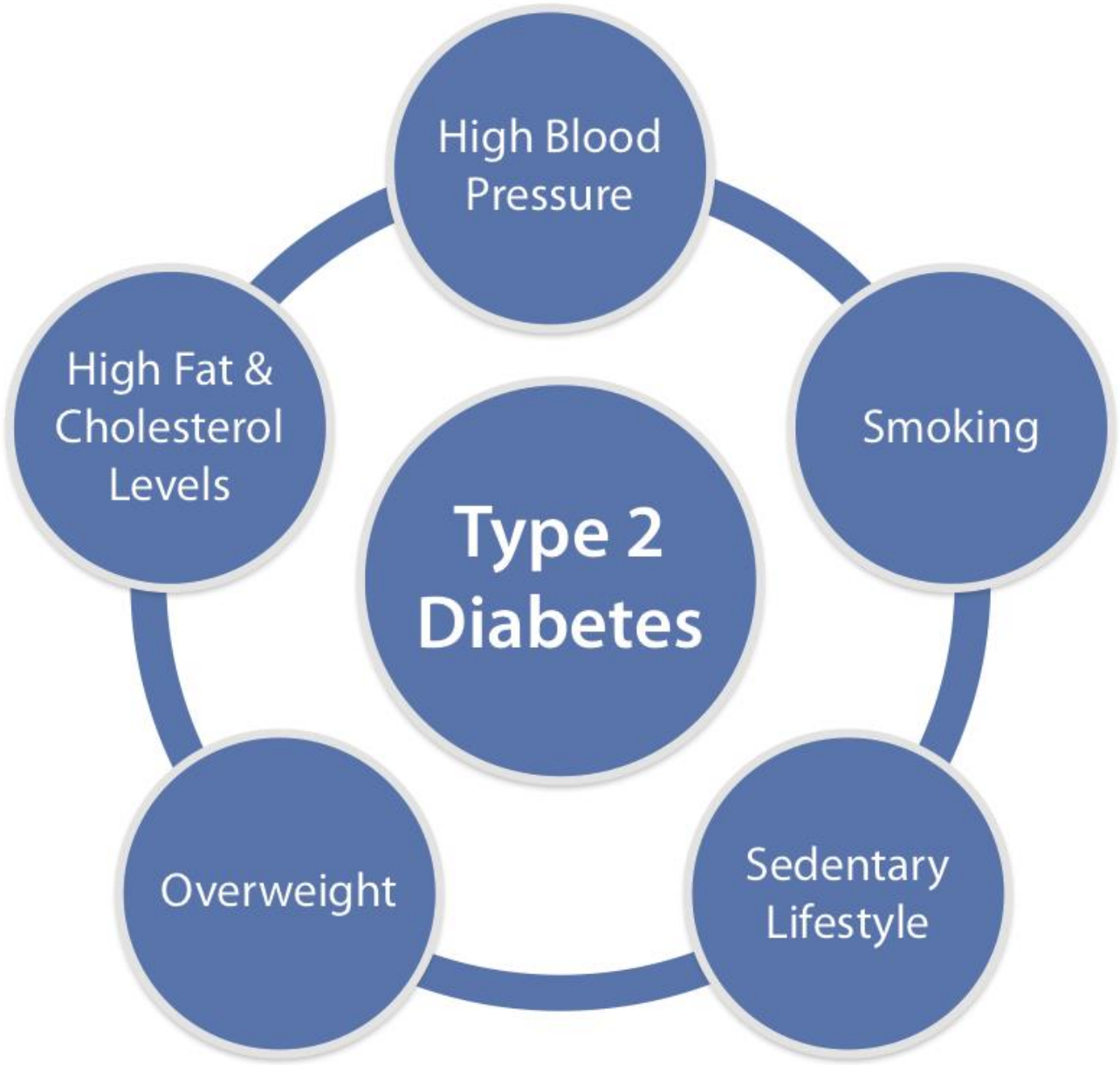


Source: International Diabetes Federation

S. Culp, 12/11/2013

REUTERS

https://www.google.pl/search?biw=1280&bih=588&tbm=isch&sa=1&q=idf+diabetes+atlas&oq=idf+diabetes+atlas&gs_l=psy-ab.3..0i19k1l2j0i30i19k1.70885.75106.0.75309.18.18.0.0.0.0.161.1914.7j11.18.0....0...1.1.64.psy-ab..0.18.1907...0j0i30k1.0.eahFI3OO-y0#imgdii=GwMDHuN7jqx0yM:&imgrc=edCDyzzIAdrGCM:



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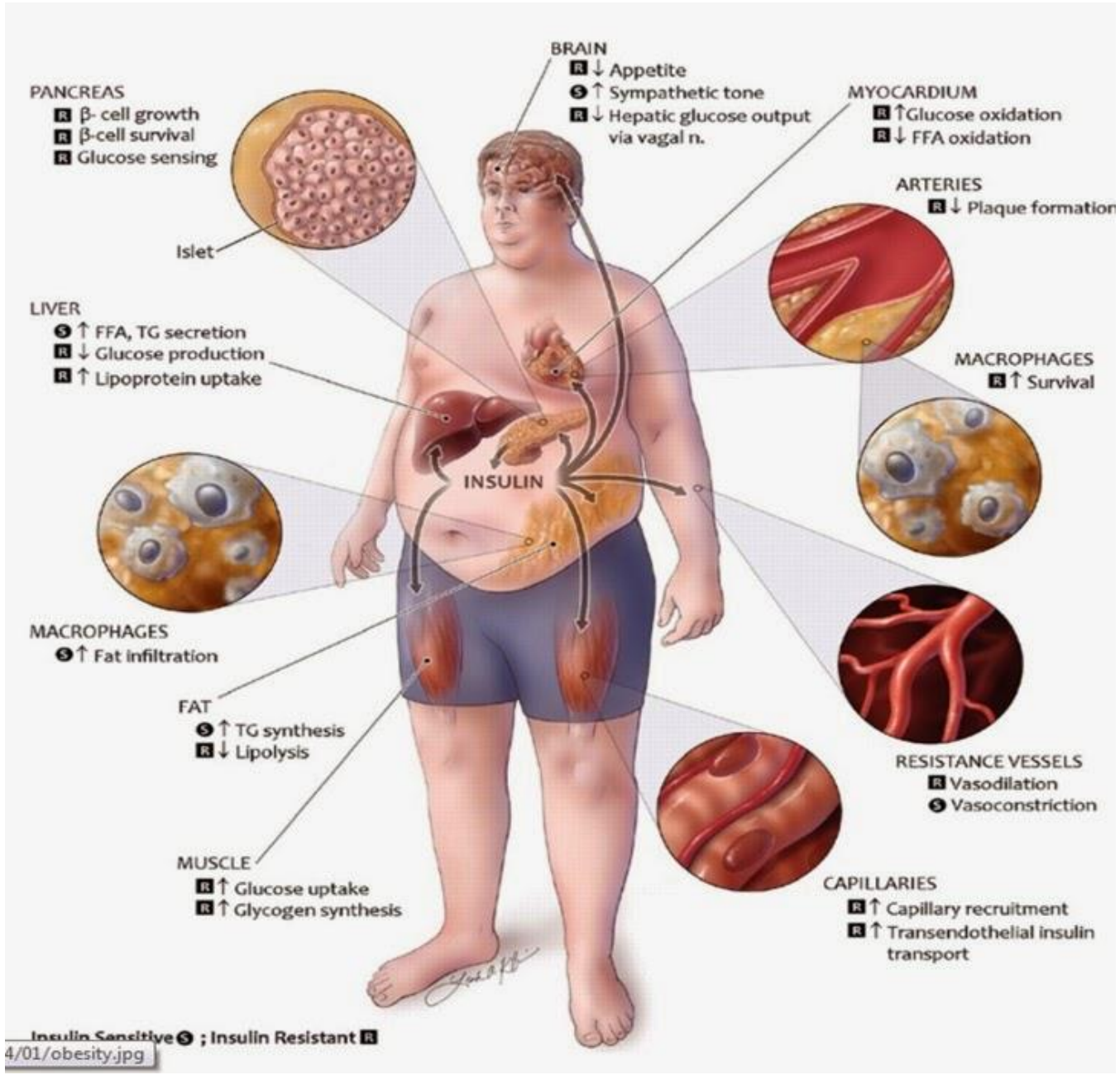
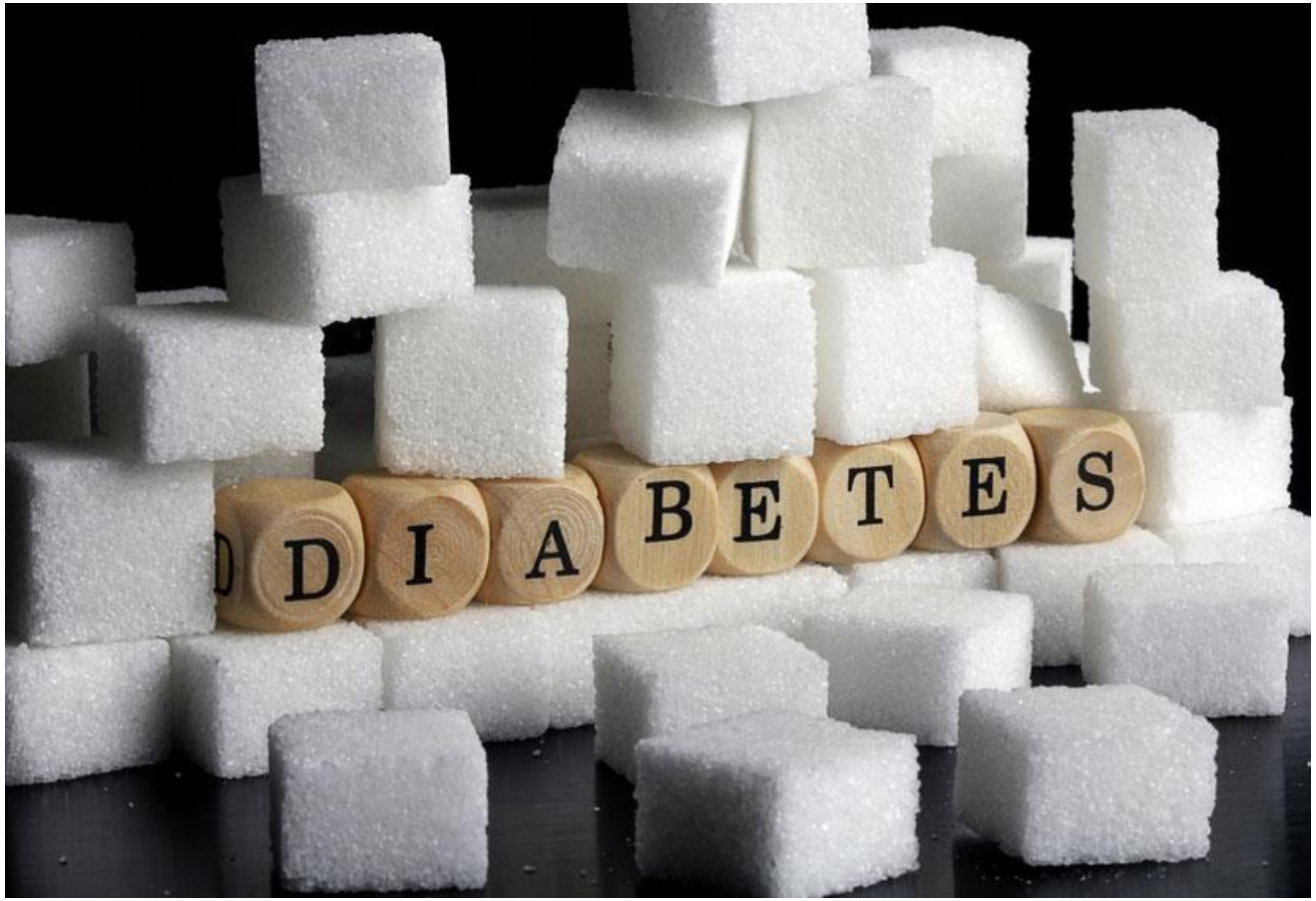


http://4.bp.blogspot.com/-TUs0s6pOvSc/UzbUOBWeMQI/AAAAAAAABLg/ISvO1r9ll9A/s1600/14.jpg

Pandemia schorzeń kardiometabolicznych w krajach rozwiniętych i rozwijających się



- Korelujący z rozwojem cywilizacyjnym wzrost zapadalności na choroby kardiometaboliczne, w tym cukrzycę typu 2, skłania do intensywnych poszukiwań czynnika sprawczego

Klasyczne czynniki ryzyka rozwoju zaburzeń węglowodanowych

- **Zmienność genetyczna** ani **klasyczne czynniki środowiskowe** tj. nieprawidłowa dieta, mała aktywność fizyczna, stosowanie używek, czy mała ilość snu nie wyjaśniają stopnia wzrostu częstości występowania zaburzeń węglowodanowych w społeczeństwach krajów rozwiniętych i rozwijających się.

Zmiana warunków środowiska jako potencjalny czynnik ryzyka rozwoju zaburzeń węglowodanowych

- Wspólnym i narastającym w skali zjawiskiem w tych krajach jest radykalna **zmiana warunków środowiska**: zmniejszenie zasobów środowiska naturalnego i jego zanieczyszczenie.

- Wynika to z dużej aktywności przemysłu oraz **zmiany stylu życia ludzi**, w tym swobodnego dostępu do źródeł energii, środków transportu, przetworzonej żywności itp.

Air Pollution in the World

Real-time Air Quality Index (AQI)

Wskaźniki epidemiologiczne

- Według danych Światowej Organizacji Zdrowia (WHO Environment and Health Information System, ENHIS) z 2009 roku, w 357 miastach Europy (33 kraje) 83% populacji narażonej było na przekraczające normy stężenie PM10.
- Około 40 milionów mieszkańców 115 największych krajów Unii Europejskiej eksponowanych jest na przekraczające normy stężenie co najmniej jednej substancji uznanej za zanieczyszczenie.
- Skala problemu jest znacznie większa w krajach rozwijających się Ameryki Łacińskiej, Azji i Afryki, gdzie nie ma rutynowego monitorowania zanieczyszczeń, a ich normy przekraczane są nawet kilkunastokrotnie.

Wskaźniki epidemiologiczne

- Europejska Agencja Środowiska ulokowała aż 6 polskich miast w pierwszej dziesiątce miast europejskich z największą liczbą dni w roku, w których przekroczono dobowe dopuszczalne stężenie pyłu PM10

Główne źródła zanieczyszczeń powietrza

- spalanie w gospodarstwach domowych węgla i drewna

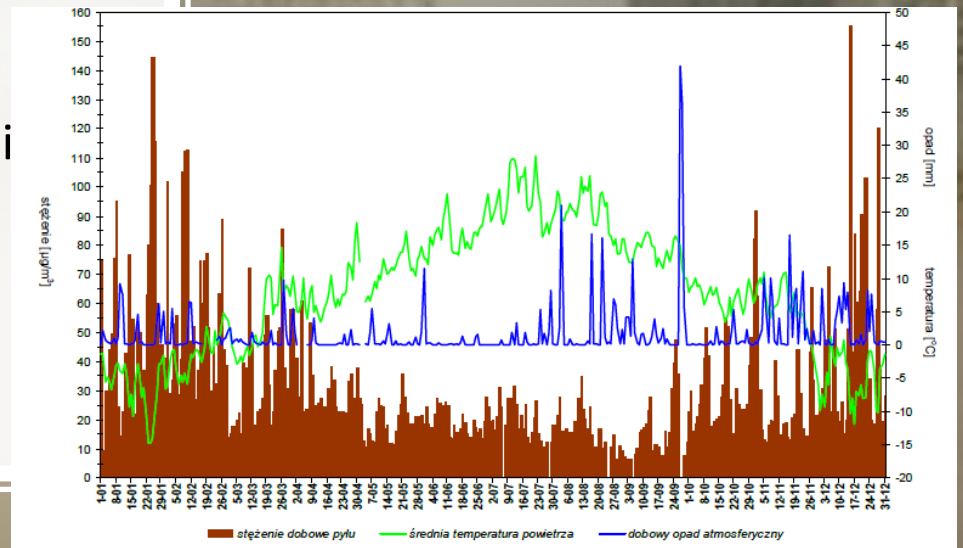
- aktywność przemysłu

- transport drogowy

- Według Naczelnej Izby Kontroli, w Polsce latach 2009-2012 główną przyczyną zanieczyszczenia powietrza pyłem PM10 (od 82 proc. do 92,8 proc.) była tzw. niska emisja, pochodząca z domowych pieców i lokalnych kotłowni węglowych

Wpływ zanieczyszczenie powietrza na organizm człowieka

- Zanieczyszczenie powietrza wykazuje zmienność nasilenia w czasie, zależną m.in. od aktywności przemysłu i aktywności człowieka oraz od pory dnia i warunków klimatycznych tj. zmiany temperatury, nasilenie opadów, ruchy mas powietrza wywołane wiatrami itp.
- Jego wpływ na organizmy żywe, w tym na ludzi, zależny jest od czasu ekspozycji i jej natężenia, współistniejących predyspozycji osobniczych i indywidualnej efektywności mechanizmów ochronnych.



Rys. 4.8. Zależność stężeń pyłu PM10 od temperatury powietrza i opadu atmosferycznego na stacji tła miejskiego w Gdańsku-Wrzeszczu, ul. Leczkowa w 2010 roku.

Wpływ zanieczyszczenia powietrza na organizm człowieka

- POCHP
- choroby nowotworowe
- miażdżyca
- choroba wieńcowa, zawał serca
- nadciśnienie tętnicze
- niewydolność serca
- udar mózgu
- zakrzepica żylna
- bezpłodność
- depresja, większe ryzyko samobójstwa

- większe ryzyko obumarcia płodu
- większe ryzyko wcześniactwa
- mniejsza masa urodzeniowa i wzrost
- gorszy rozwój psychofizyczny
- problemy z pamięcią i koncentracją, niższy IQ
- choroba Alzheimera
- **zaburzenia węglowodanowe**
- …

MECHANISMS IN ENDOCRINOLOGY

Effect of long-term exposure to air pollution on type 2 diabetes mellitus risk: a systemic review and meta-analysis of cohort studies

Bin Wang[1,2], Donghua Xu[3], Zhaohai Jing[1], Dawei Liu[4], Shengli Yan[1] and Yangang Wang[1]

European Journal of Endocrinology
(2014) **171**, R173–R182

Abstract

Objective: To assess the effect of long-term exposure to air pollution on type 2 diabetes risk, a meta-analysis of prospective cohort studies was performed.

Methods: Literature search was conducted with Pubmed, Embase, and Web of Science for prospective cohort studies investigating the association of type 2 diabetes risk with increments in particulate matter (PM, diameter <2.5 μm (PM2.5) or <10 μm (PM10)) or nitrogen dioxide (NO_2). We used a random-effects model to calculate the overall relative risk (RR) with 95% CI.

Results: Of 808 identified articles, ten cohort studies were finally included, which involved a total of 2 371 907 participants and 21 095 incident cases of type 2 diabetes. Elevated risk of type 2 diabetes was significantly associated with long-term exposures to high levels of PM2.5 (RR=1.28, 95% CI 1.06–1.55, $P=0.009$, $I^2=83.5\%$), PM10 (RR=1.15, 95% CI 1.02–1.30, $P=0.022$, $I^2=0\%$), and NO_2 (RR=1.12, 95% CI 1.02–1.23, $P=0.015$, $I^2=63.5\%$). When using standardized risk estimates, the RRs of type 2 diabetes were significant for increments in concentrations of PM2.5 (1.39 per 10 μg/m^3 increment, 95% CI 1.14–1.68, $P=0.001$), PM10 (1.34 per 10 μg/m^3 increment, 95% CI 1.22–1.47, $P<0.001$), and NO_2 (1.11 per 10 μg/m^3 increment, 95% CI 1.07–1.16, $P<0.001$). No obvious evidence of publication bias was observed.

Conclusion: Long-term exposure to high levels of main air pollutants is significantly associated with elevated risk of type 2 diabetes mellitus.

Metaanaliza 10 badań
2 371 907 osób
w tym 21 095 DM2

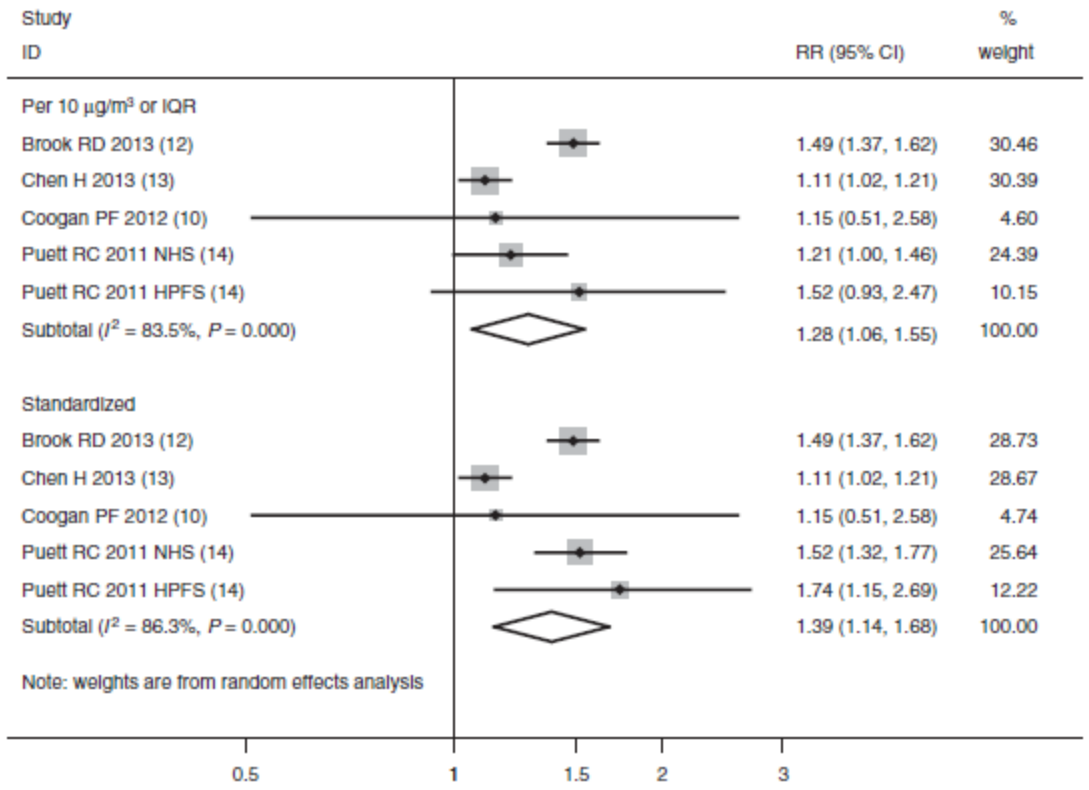
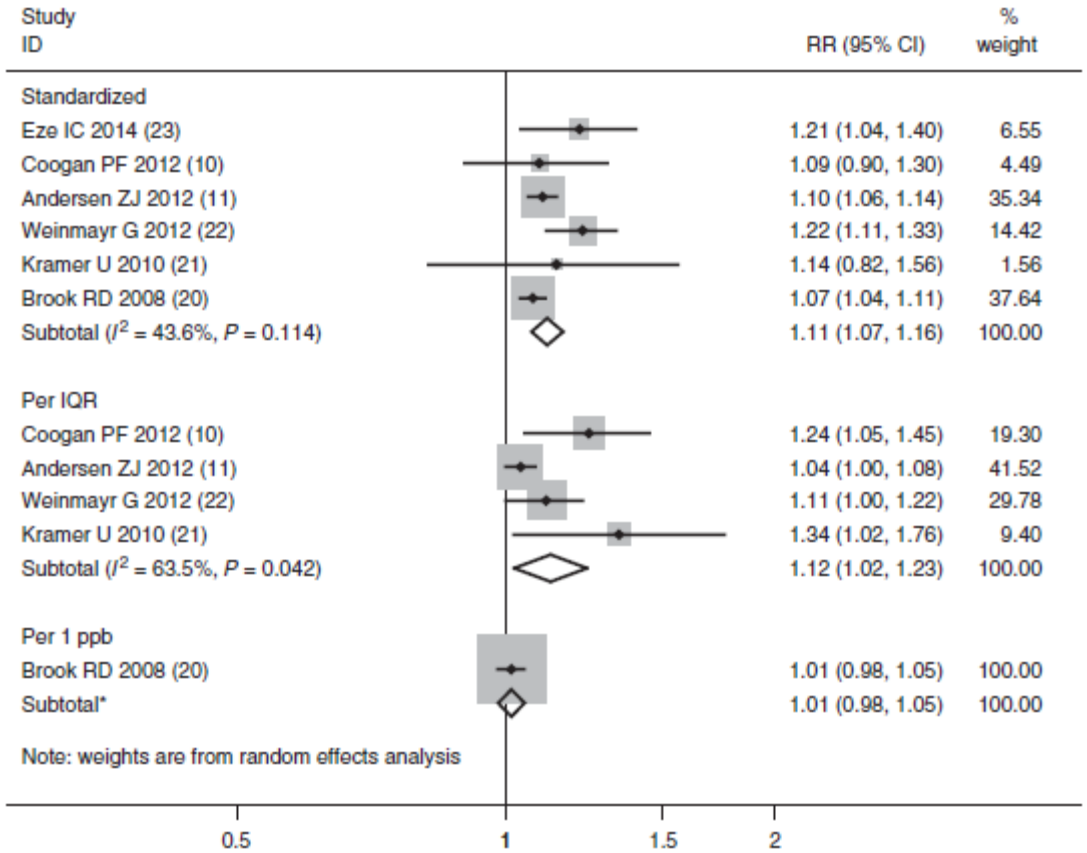


Figure 2

Association between long-term exposure to high level of PM2.5 and type 2 diabetes risk.

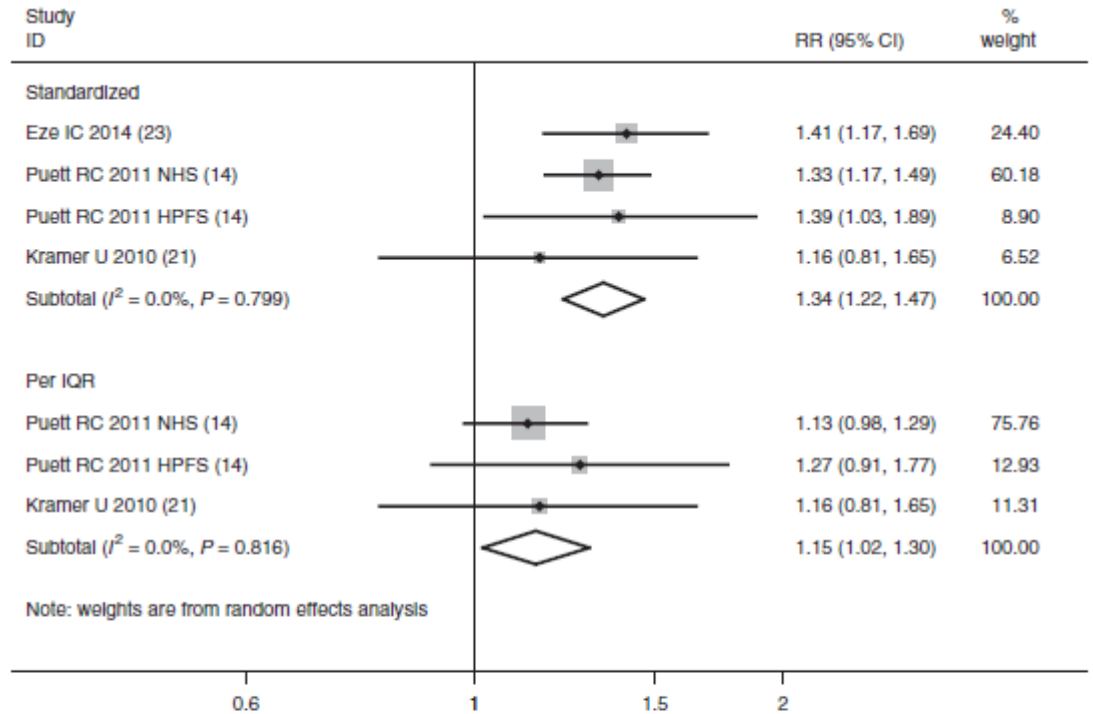
PM2.5 RR 1.28



NO2 RR 1.12

Figure 3

Association between long-term exposure to high level of NO_2 and type 2 diabetes risk. *I^2 and P values are not available as there was only one study and heterogeneity could not be analysed.



PM10 RR 1.15

Figure 4

Association between long-term exposure to high level of PM10 and type 2 diabetes risk.

Association between Ambient Air Pollution and Diabetes Mellitus in Europe and North America: Systematic Review and Meta-Analysis

Ikenna C. Eze,[1,2] Lars G. Hemkens,[3] Heiner C. Bucher,[3] Barbara Hoffmann,[4,5] Christian Schindler,[1,2] Nino Künzli,[1,2] Tamara Schikowski,[1,2,4] and Nicole M. Probst-Hensch[1,2]

[1]Swiss Tropical and Public Health Institute, Basel, Switzerland; [2]University of Basel, Basel, Switzerland; [3]Basel Institute for Clinical Epidemiology and Biostatistics, University Hospital Basel, Basel, Switzerland; [4]IUF-Leibniz Research Institute for Environmental Medicine, Düsseldorf, Germany; [5]Medical Faculty, Heinrich Heine University of Düsseldorf, Düsseldorf, Germany

Environmental Health Perspectives • VOLUME 123 | NUMBER 5 | May 2015

Metaanaliza 13 badań
Europa i Północna
Ameryka

BACKGROUND: Air pollution is hypothesized to be a risk factor for diabetes. Epidemiological evidence is inconsistent and has not been systematically evaluated.

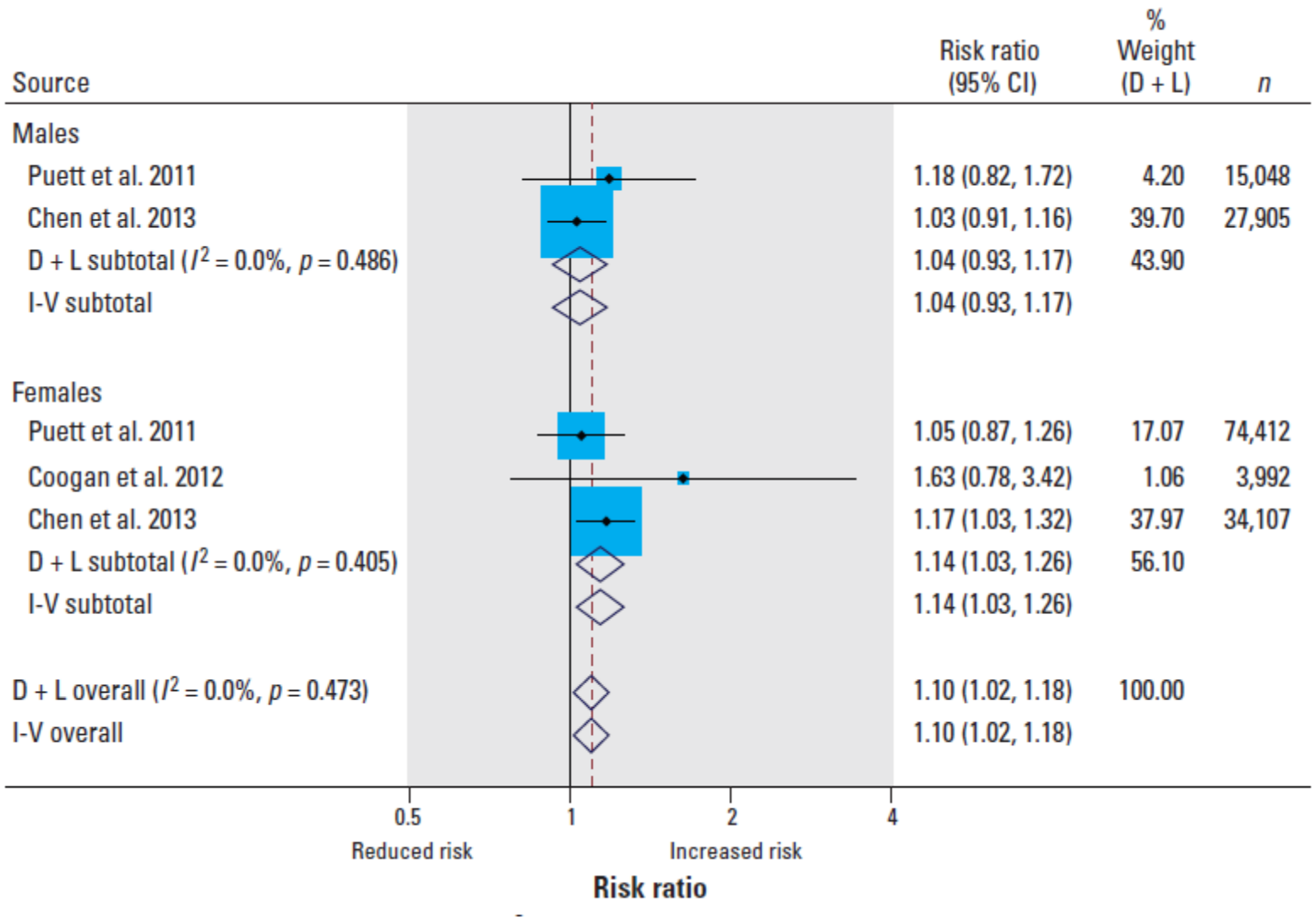
OBJECTIVES: We systematically reviewed epidemiological evidence on the association between air pollution and diabetes, and synthesized results of studies on type 2 diabetes mellitus (T2DM).

METHODS: We systematically searched electronic literature databases (last search, 29 April 2014) for studies reporting the association between air pollution (particle concentration or traffic exposure) and diabetes (type 1, type 2, or gestational). We systematically evaluated risk of bias and role of potential confounders in all studies. We synthesized reported associations with T2DM in meta-analyses using random-effects models and conducted various sensitivity analyses.

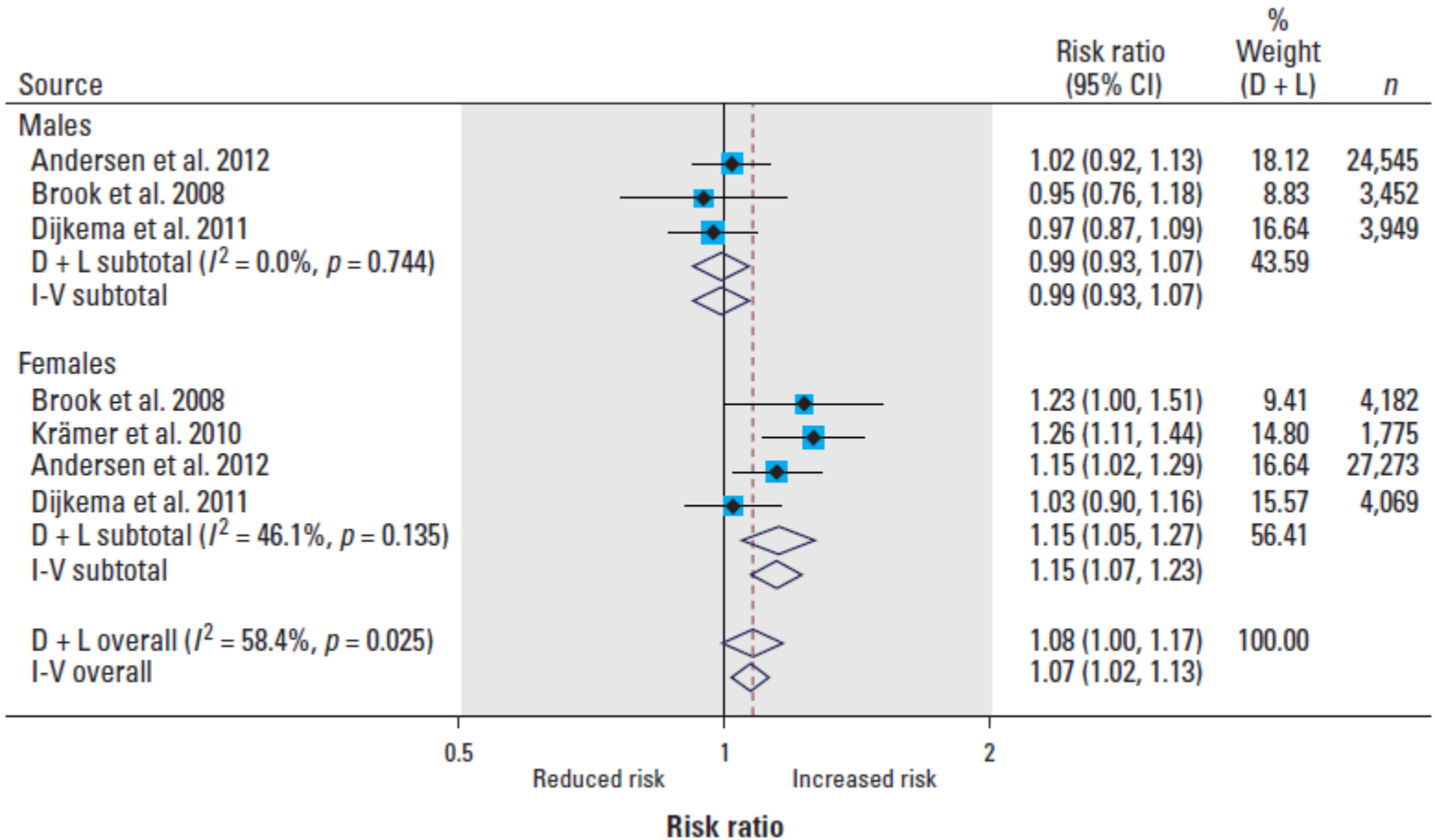
RESULTS: We included 13 studies (8 on T2DM, 2 on type 1, 3 on gestational diabetes), all conducted in Europe or North America. Five studies were longitudinal, 5 cross-sectional, 2 case–control, and 1 ecologic. Risk of bias, air pollution assessment, and confounder control varied across studies. Dose–response effects were not reported. Meta-analyses of 3 studies on $PM_{2.5}$ (particulate matter ≤ 2.5 μm in diameter) and 4 studies on NO_2 (nitrogen dioxide) showed increased risk of T2DM by 8–10% per 10-μg/m^3 increase in exposure [$PM_{2.5}$: 1.10 (95% CI: 1.02, 1.18); NO_2: 1.08 (95% CI: 1.00, 1.17)]. Associations were stronger in females. Sensitivity analyses showed similar results.

CONCLUSION: Existing evidence indicates a positive association of air pollution and T2DM risk, albeit there is high risk of bias. High-quality studies assessing dose–response effects are needed. Research should be expanded to developing countries where outdoor and indoor air pollution are high.

CITATION: Eze IC, Hemkens LG, Bucher HC, Hoffmann B, Schindler C, Künzli N, Schilowski T, Probst-Hensch NM. 2015. Association between ambient air pollution and diabetes mellitus in Europe and North America: systematic review and meta-analysis. Environ Health Perspect 123:381–389; http://dx.doi.org/10.1289/ehp.1307823



PM2.5 and risk of DM2
RR 1.1



NO2 and risk of DM2
RR 1.08

JDI Journal of Diabetes Investigation Open access
Official Journal of the Asian Association for the Study of Diabetes
ORIGINAL ARTICLE

Association between particulate matter 2.5 and diabetes mellitus: A meta-analysis of cohort studies

Dian He[1,2*†], Shaowen Wu[3†], Haiping Zhao[1,2], Hongyan Qiu[4], Yang Fu[1,2], Xingming Li[5], Yan He[1,2]

[1]Department of Epidemiology and Health Statistics, School of Public Health, Capital Medical University, [2]Beijing Municipal Key Laboratory of Clinical Epidemiology, [3]Beijing Obstetrics and Gynecology Hospital, Capital Medical University, Beijing, [4]Department of Epidemiology and Health Statistics, School of Public Health, Ningxia Medical University, Yinchuan, and [5]School of Health Administration and Education, Capital Medical University, Beijing, China

J Diabetes Investig 2017; 8: 687–696

Metaanaliza z 2017r.
10 badań
Ocena wpływu PM 2.5 na ryzyko DM2 i DM w ciąży

ABSTRACT

Aims/Introduction: The present meta-analysis was carried out to assess the association between exposure to the level of atmospheric particulate matter 2.5 (PM2.5; fine particulate matter with aerodynamic diameter less than 2.5 μm) and type 2 diabetes mellitus or gestational diabetes mellitus (GDM).

Materials and Methods: We searched the Medline, EMBASE, Cochrane and Web of Science databases to obtain articles according to the responding literature search strategies. Among a total of 279 identified articles, 55 were reviewed in depth, of which 10 articles (11 cohort studies) satisfied the inclusion criteria. Only cohort studies that disclosed the association between PM2.5 and type 2 diabetes mellitus or GDM were included in this article. A fixed-effects model was selected if $P > 0.1$ and $I^2 < 50\%$; otherwise, a random-effects model would be used to calculate the total effect value. Subgroup analysis was further carried out according to the types of diabetes mellitus (type 2 diabetes mellitus and GDM). The relative risk was used to estimate the association between PM2.5 and diabetes mellitus.

Results: The positive associations between PM2.5 and the incidence of type 2 diabetes mellitus were found in the long-term exposure period (relative risk 1.25, 95% confidence interval 1.10–1.43), which showed that with every 10-μg/m^3 increase in PM2.5, the risk of type 2 diabetes mellitus would increase by 25% in the long-term exposure. Although the significant associations were not identified between maternal exposure to PM2.5 and GDM in the first trimester, the second trimester and the entire pregnancy periods, we could conclude that maternal exposure to PM2.5 in the entire pregnancy period would be more likely to lead to developing GDM (relative risk 1.162, 95% confidence interval 0.806–1.675) than the other two periods.

Conclusions: Long-term exposure to PM2.5 would be more likely to lead to developing type 2 diabetes mellitus, but more studies would be required to confirm the association between PM2.5 and GDM. It might be a wise to take effective measures to reduce PM2.5 exposure in vulnerable populations, especially for pregnant women.

wzrost PM2.5 o 10ug/m3 – wzrost ryzyka DM2 o 25%

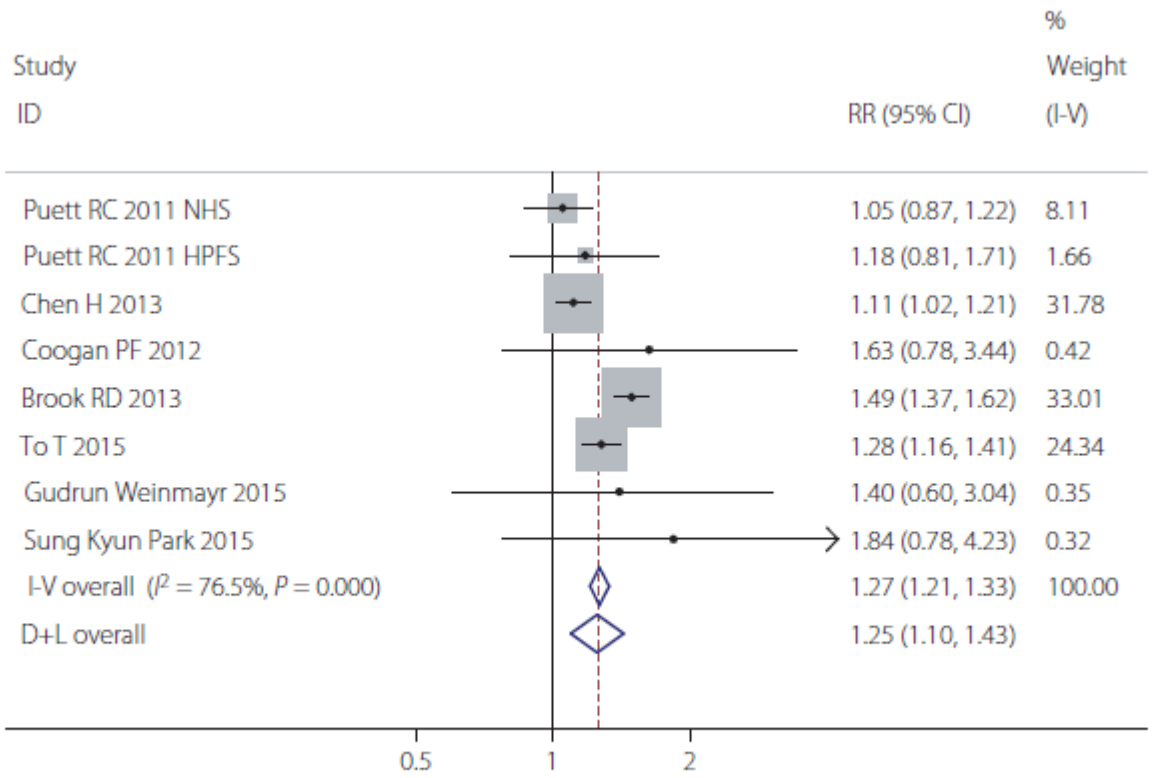


Figure 2 | Forest plot of meta-analysis in the associations between long-term exposure to particulate matter 2.5 and type 2 diabetes mellitus. 95% CI, 95% confidence interval; D+L, DerSimonian-Laird; I-V, inverse variance; RR, relative risk.

ekspozycja matek na PM2.5 w początkowym okresie ciąży zwiększa ryzyko wystąpienia cukrzycy (RR 1.162)

Association of Atmospheric Particulate Matter and Ozone with Gestational Diabetes Mellitus

Hui Hu,[1] Sandie Ha,[1] Barron H. Henderson,[2] Tamara D. Warner,[3] Jeffrey Roth,[3] Haidong Kan,[4] and Xiaohui Xu[1]

[1]Department of Epidemiology, College of Public Health and Health Professions and College of Medicine, [2]Department of Environmental Engineering Sciences, Engineering School of Sustainable Infrastructure and Environment, and [3]Department of Pediatrics, College of Medicine, University of Florida, Gainesville, Florida, USA; [4]Department of Environmental Health, School of Public Health, Fudan University, Shanghai, China

Environmental Health Perspectives • VOLUME 123 | NUMBER 9 | September 2015

410 267 kobiet ciężarnych
Floryda 2014-2015
Ekspozycja na PM2.5 i ozon w kolejnych trymestrach ciąży

BACKGROUND: Ambient air pollution has been linked to the development of gestational diabetes mellitus (GDM). However, evidence of the association is very limited, and no study has estimated the effects of ozone.

OBJECTIVE: Our aim was to determine the association of prenatal exposures to particulate matter ≤ 2.5 μm ($PM_{2.5}$) and ozone (O_3) with GDM.

METHODS: We used Florida birth vital statistics records to investigate the association between the risk of GDM and two air pollutants ($PM_{2.5}$ and O_3) among 410,267 women who gave birth in Florida between 2004 and 2005. Individual air pollution exposure was assessed at the woman's home address at time of delivery using the hierarchical Bayesian space–time statistical model. We further estimated associations between air pollution exposures during different trimesters and GDM.

RESULTS: After controlling for nine covariates, we observed increased odds of GDM with per 5-μg/m^3 increase in $PM_{2.5}$ ($OR_{Trimester1}$ = 1.16; 95% CI: 1.11, 1.21; $OR_{Trimester2}$ = 1.15; 95% CI: 1.10, 1.20; $OR_{Pregnancy}$ = 1.20; 95% CI: 1.13, 1.26) and per 5-ppb increase in O_3 ($OR_{Trimester1}$ = 1.09; 95% CI: 1.07, 1.11; $OR_{Trimester2}$ = 1.12; 95% CI: 1.10, 1.14; $OR_{Pregnancy}$ = 1.18; 95% CI: 1.15, 1.21) during both the first trimester and second trimester as well as the full pregnancy in single-pollutant models. Compared with the single-pollutant model, the ORs for O_3 were almost identical in the co-pollutant model. However, the ORs for $PM_{2.5}$ during the first trimester and the full pregnancy were attenuated, and no association was observed for $PM_{2.5}$ during the second trimester in the co-pollutant model (OR = 1.02; 95% CI: 0.98, 1.07).

CONCLUSION: This population-based study suggests that exposure to air pollution during pregnancy is associated with increased risk of GDM in Florida, USA.

CITATION: Hu H, Ha S, Henderson BH, Warner TD, Roth J, Kan H, Xu X. 2015. Association of atmospheric particulate matter and ozone with gestational diabetes mellitus. Environ Health Perspect 123:853–859; http://dx.doi.org/10.1289/ehp.1408456

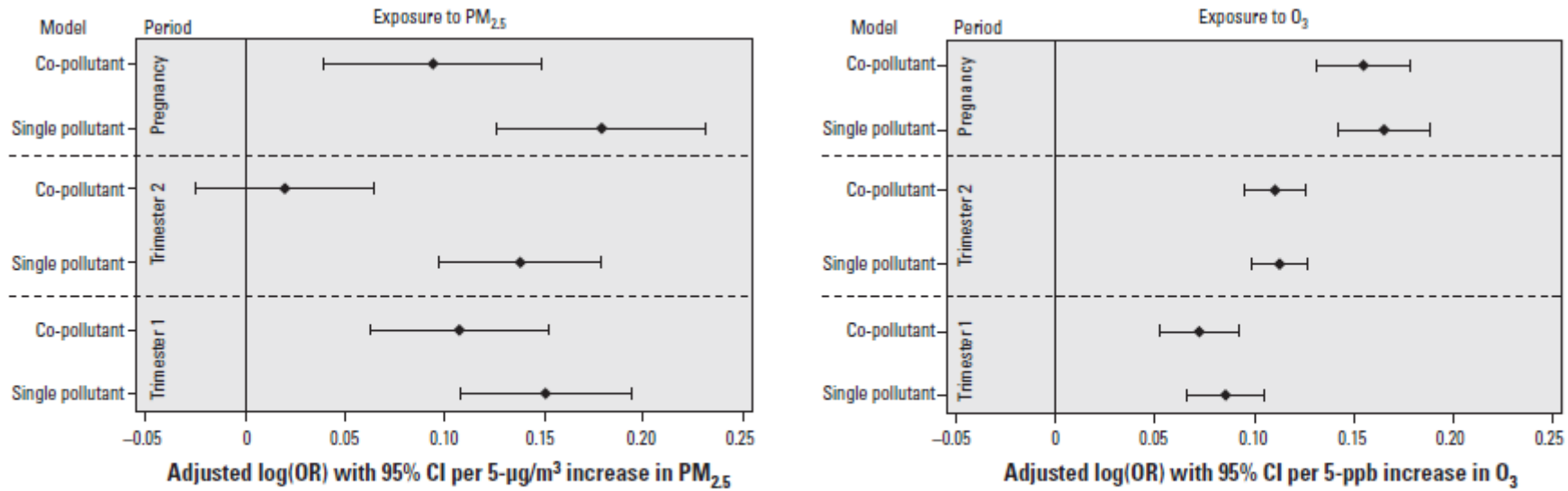


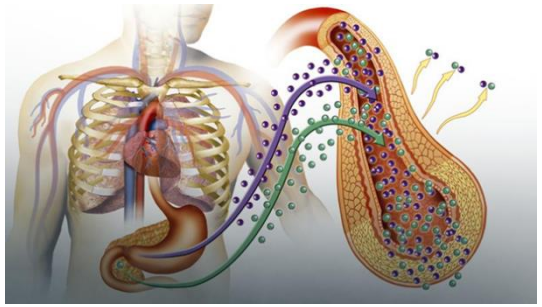
Figure 1. Adjusted log(OR) for risk of GDM with per 5 units increase in gestational exposure to pollutant for single- and co-pollutant models among women who gave birth in 2004–2005 in Florida, USA. Diamonds reflect the central estimate; whiskers represent the 95% CIs.

Ryzyko wystąpienia zaburzeń węglowodanowych

jest niezależne od:

- wieku badanych (stwierdza się nawet u dzieci!)
- predyspozycji osobniczej (np. otyłości)
- czasu ekspozycji (zmiany obserwowane są już po 5-ciodniowym narażeniu!)

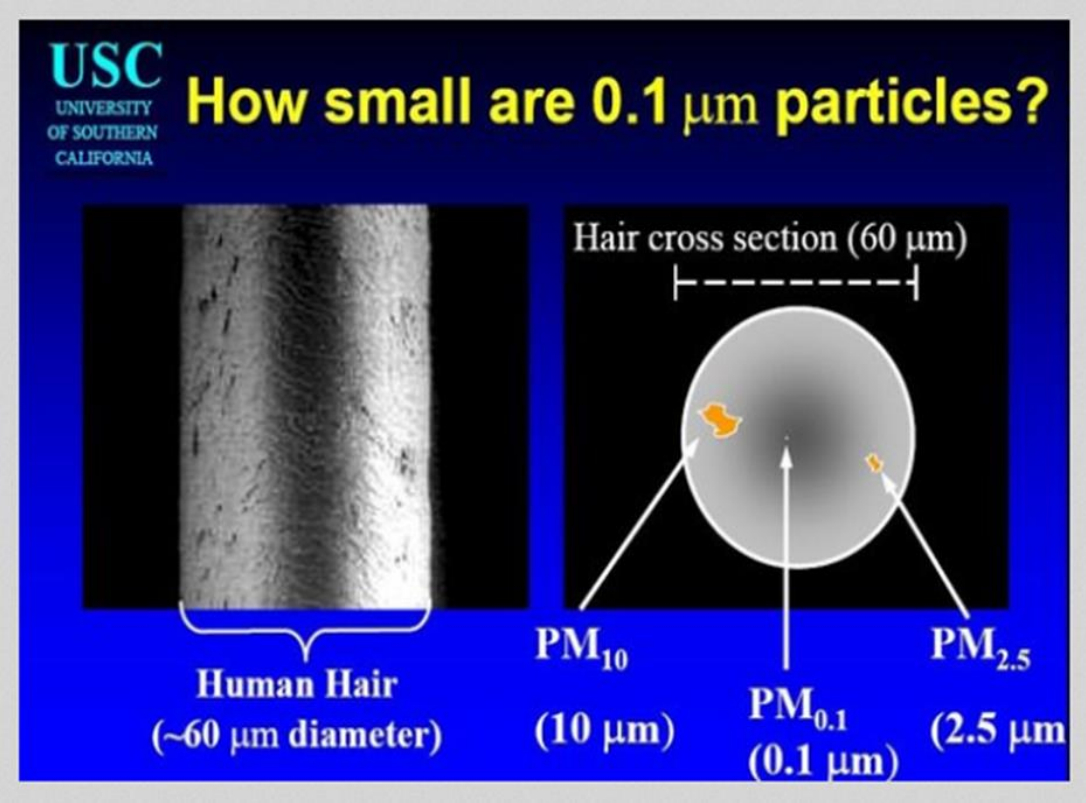
jest większe nawet w warunkach jeszcze dopuszczalnych zakresów stężeń składowych pyłów zawieszonych w powietrzu!



Spektrum zaburzeń gospodarki węglowodanowej jako efekt narażenia na zanieczyszczenie powietrza

- ↑ insulinooporności
- nieprawidłowa glikemia na czczo
- nieprawidłowa tolerancja węglowodanów
- cukrzyca
- gorsza kontrola metaboliczna cukrzycy

Pył zawieszony w powietrzu



Analiza chemiczna pyłu PM2.5 wykazuje m.in.

metale (arsen, nikiel, kadm, ołów, glin, tytan, żelazo)

wielopierścieniowe węglowodory aromatyczne [benzo(a)piren, benzo(a)antracen, benzo(b)fluoranten, benzo(j)fluoranten, benzo(k)fluoranten]

aniony (sód, potas, wapń, magnez, jon amonowy NH4+)

kationy (siarczany SO4 2-, azotany NO3-, chlorki Cl-)

formy związków węgla: węgiel organiczny (OC), węgiel elementarny (EC) *

*Inspekcja Ochrony Środowiska. Analiza stanu zanieczyszczenia powietrza pyłem PM10 i PM2,5 z uwzględnieniem składu chemicznego pyłu oraz wpływu źródeł naturalnych. Raport syntetyczny. Zabrze, kwiecień 2011

PLOS | ONE

RESEARCH ARTICLE

Association of Urinary Metal Profiles with Altered Glucose Levels and Diabetes Risk: A Population-Based Study in China

Wei Feng, Xiuqing Cui, Bing Liu, Chuanyao Liu, Yang Xiao, Wei Lu, Huan Guo, Meian He, Xiaomin Zhang, Jing Yuan, Weihong Chen, Tangchun Wu*

PLOS ONE | DOI:10.1371/journal.pone.0123742 April 13, 2015

Methods

We conducted a cross-sectional study to investigate the associations of urinary concentrations of 23 metals with FPG, impaired fasting glucose (IFG) and diabetes among 2242 community-based Chinese adults in Wuhan. We used the false discovery rate (FDR) method to correct for multiple hypothesis tests.

Results

After adjusting for potential confounders, urinary aluminum, titanium, cobalt, nickel, copper, zinc, selenium, rubidium, strontium, molybdenum, cadmium, antimony, barium, tungsten and lead were associated with altered FPG, IFG or diabetes risk (all $P < 0.05$); arsenic was only dose-dependently related to diabetes ($P < 0.05$). After additional adjustment for multiple testing, titanium, copper, zinc, selenium, rubidium, tungsten and lead were still significantly associated with one or more outcomes (all FDR-adjusted $P < 0.05$).

Conclusions

Our results suggest that multiple metals in urine are associated with FPG, IFG or diabetes risk. Because the cross-sectional design precludes inferences about causality, further prospective studies are warranted to validate our findings.

Polski indeks jakości powietrza	SO_2 [µg/m^3]	NO_2 [µg/m^3]	CO [µg/m^3]	PM_{10} [µg/m^3]	$PM_{2,5}$ [µg/m^3]	O_3 [µg/m^3]	C_6H_6 [µg/m^3]
Bardzo dobry	0–50	0–40	0–2 000	0–20	0–12	0–24	0–5
Dobry	50–100	40–100	2 000–6 000	20–60	12–36	24–70	5–10
Umiarkowany	100–200	100–150	6 000–10 000	60–100	36–60	70–120	10–15
Dostateczny	201–350	150–200	10 000–14 000	100–140	60–84	120–160	15–20
Zły	351–500	200–400	14 000–20 000	140–200	84–120	160–240	20–50
Bardzo zły	>500	>400	>20 000	>200	>120	>240	>50

Normy stężeń pyłu zawieszonego zalecane przez Światową Organizację Zdrowia (WHO)[4]:

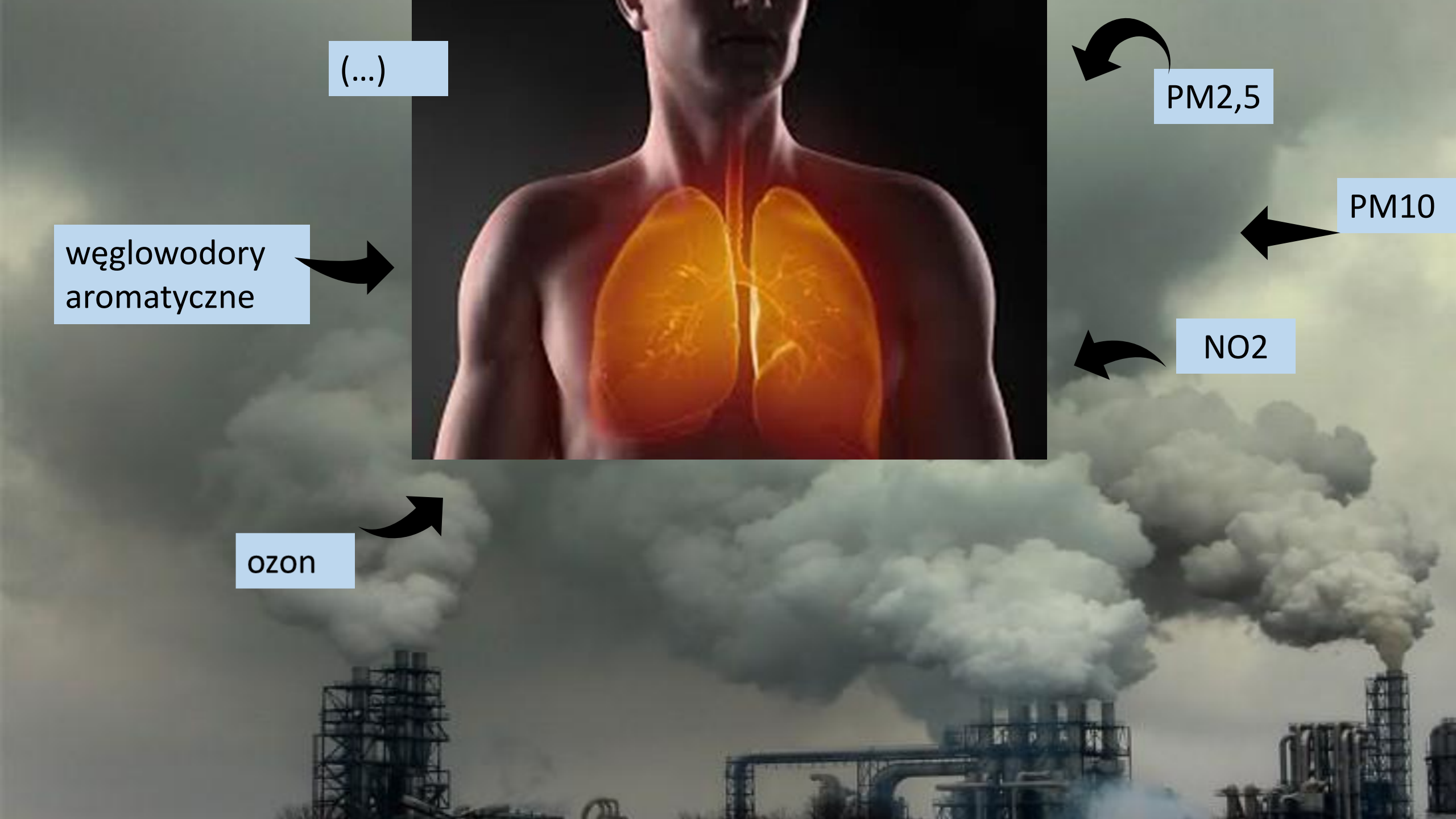
- norma średniego 24-godz. stężenia pyłu PM10: **50 µg/m^3**
- norma średniego rocznego stężenia pyłu PM10: **20 µg/m^3**
- norma średniego 24-godz. stężenia pyłu PM2,5: **25 µg/m^3**
- norma średniego rocznego stężenia pyłu PM2,5: **10 µg/m^3**

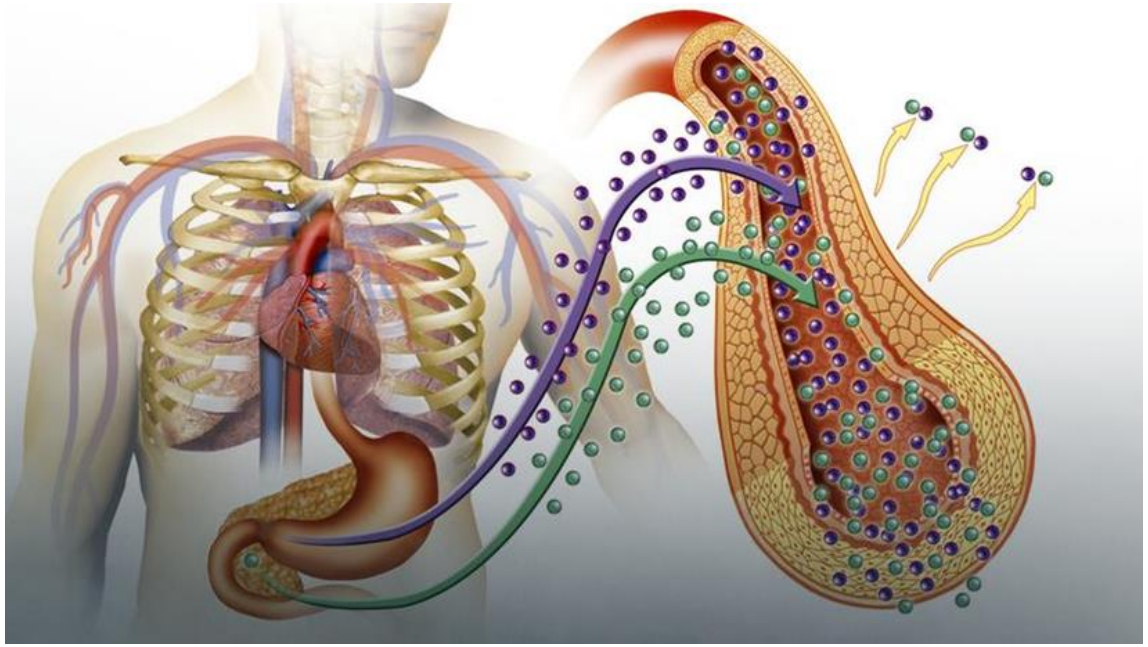
Normy stężeń dwutlenku azotu zalecane przez Światową Organizację Zdrowia (WHO)[4]:

- norma średniego 1-godz. stężenia NO_2: **200 µg/m^3**
- norma średniego rocznego stężenia NO_2: **40 µg/m^3**

4. *WHO Air quality guidelines for particulate matter, ozone, nitrogen dioxide and sulfur dioxide. Global update 2005.* WHO, 2006. Online: http://whqlibdoc.who.int/hq/2006/WHO_SDE_PHE_OEH_06.02_eng.pdf

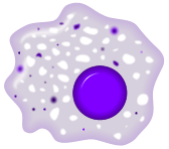
http://smog.imgw.pl/content/health


(...)
PM2,5
PM10
węglowodory aromatyczne
NO2
ozon

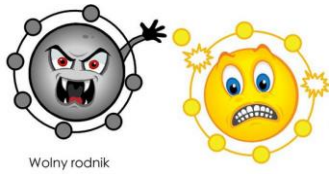


PŁUCA

lokalny stan zapalny

↓ 

subkliniczny systemowy stan zapalny



+

ogólnoustrojowy stres oksydacyjny

↓

TKANKI OBWODOWE

↓

insulinooporność
zaburzenie produkcji insuliny

Szlak patogenetyczny prowadzący od patologii płuc tj. miejsca aspiracji zanieczyszczeń, do rozwoju zaburzeń metabolizmu węglowodanów

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TOXICOLOGICAL SCIENCES, 143(2), 2015, 231–241

doi: 10.1093/toxsci/kfu250
CONTEMPORARY REVIEW

Air Pollution as a Risk Factor for Type 2 Diabetes

Xiaoquan Rao*, Priti Patel†, Robin Puett†, and Sanjay Rajagopalan*[1]

*Division of Cardiovascular Medicine, University of Maryland, Baltimore and †Maryland Institute for Applied Environmental Health, Department of Epidemiology and Biostatistics, School of Public Health, University of Maryland, College Park

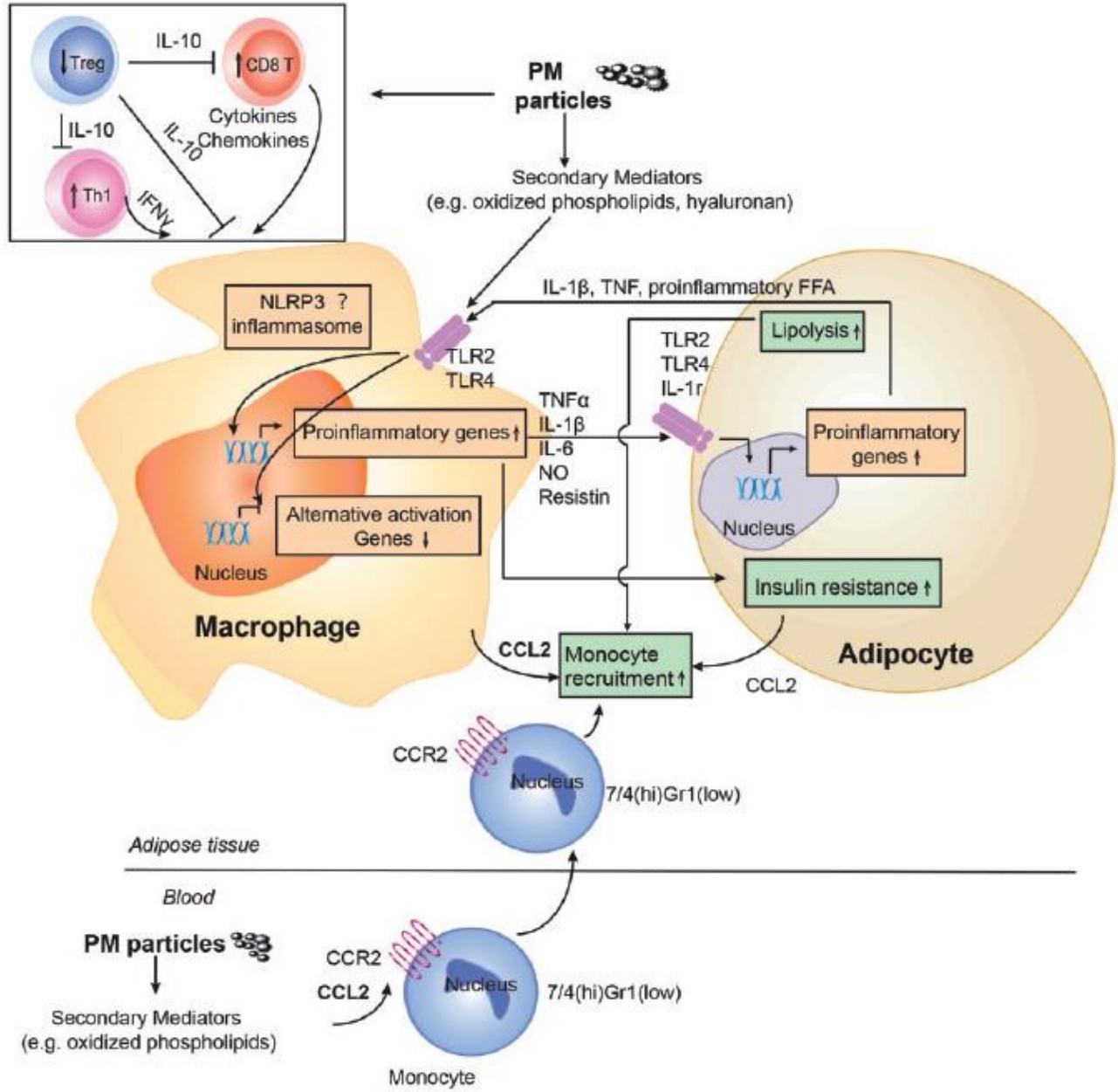

FIG. 2. Mechanisms underlying air pollution-mediated immune activation. PM, particulate matter; TLR, Toll-like receptor; Treg, regulatory T cells; CCL2, C-C motif ligand 2; CCR2, C-C chemokine receptor type 2; NLRP3, NOD-like receptor family, pyrin domain containing 3; NO, nitric oxide; TNF-α, tumor necrosis factor-α.

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CONTEMPORARY REVIEW

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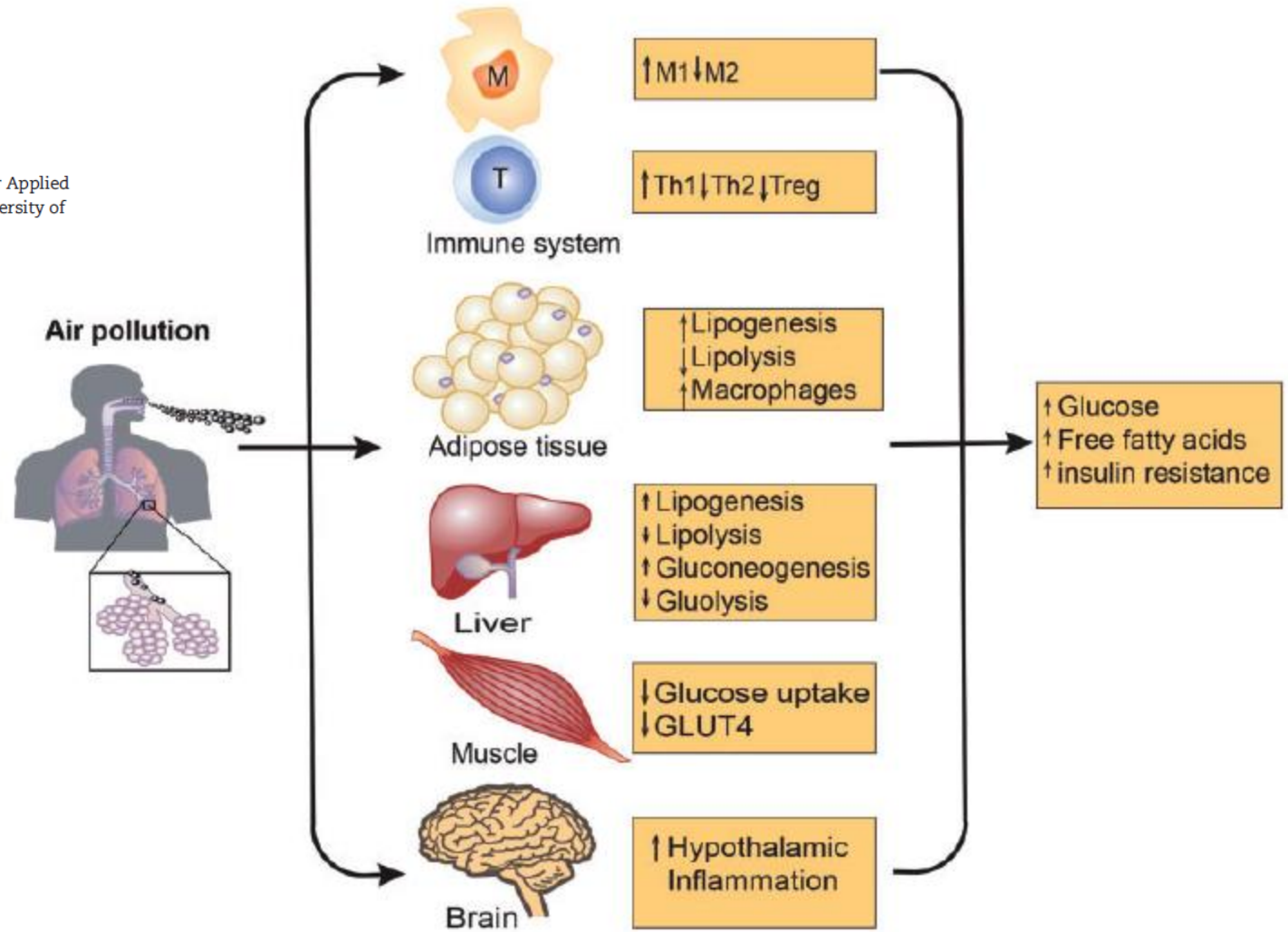

FIG. 1. Effect of air pollution on immune system, adipose tissue, muscle, liver, and brain. M1, classically activated macrophages; M2, alternatively activated macrophages; Th1, T helper type 1; Th2, T helper type 2; GLUT4, glucose transporter type 4.

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Toxicol Pathol. 2013 February ; 41(2): 361–373. doi:10.1177/0192623312464531.

Epidemiological and Experimental Links between Air Pollution and Type 2 Diabetes

Cuiqing Liu[1,2], **Zhekang Ying**[1], **Jack Harkema**[3], **Qinghua Sun**[1], and **Sanjay Rajagopalan**[1,*]

[1]Davis Heart & Lung Research Institute, The Ohio State University, Columbus, Ohio, USA

[2]Department of Physiology, Hangzhou Normal University, Hangzhou, China

[3]Department of Pathobiology and Diagnostic Investigation, Michigan State University, East Lansing, Michigan, USA

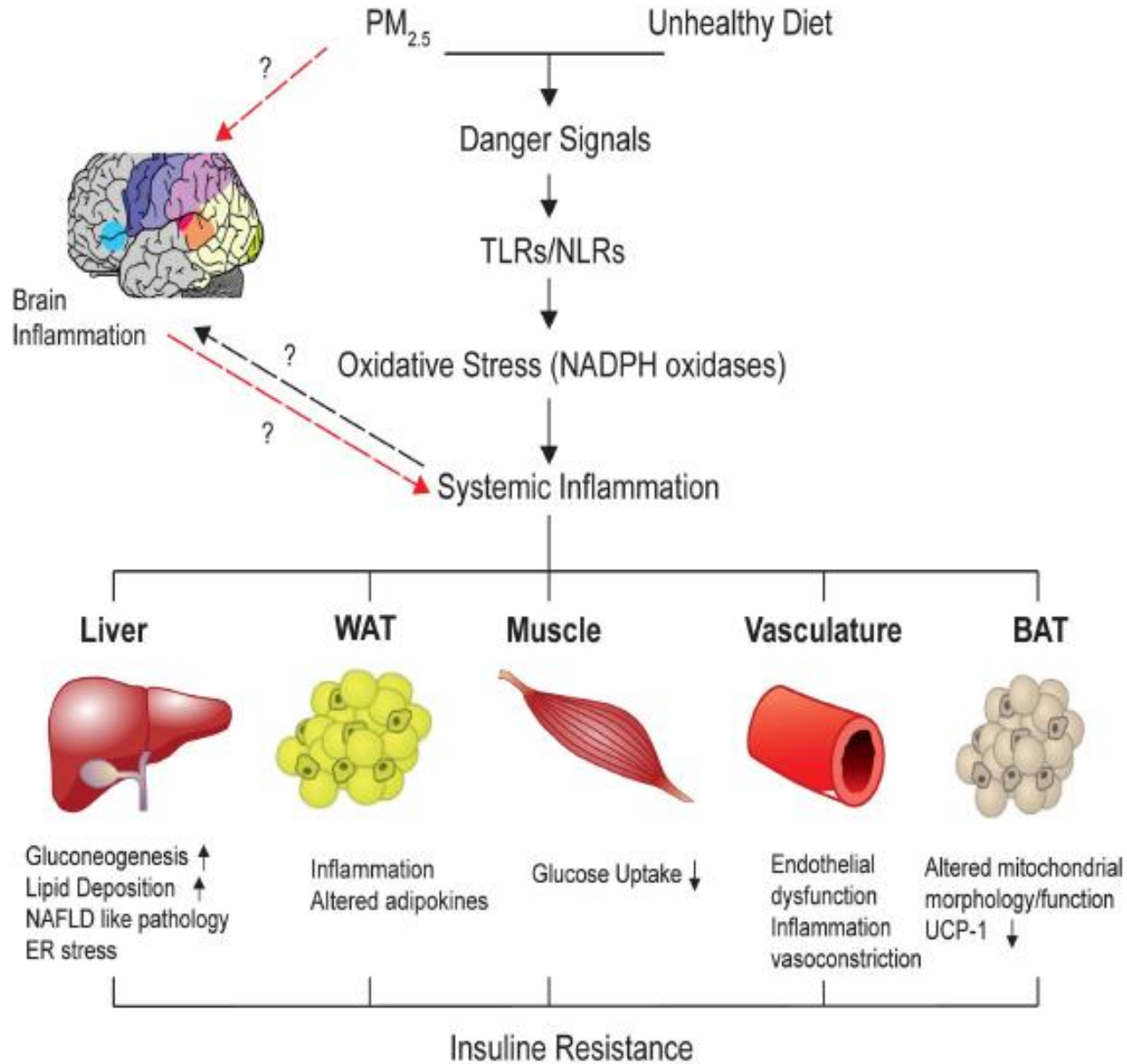


Figure 7.
Hypothesized mechanisms of air-pollution mediated type II DM/insulin resistance.

Ambient Air Pollution and Type 2 Diabetes: Do the Metabolic Effects of Air Pollution Start Early in Life?

Sung Kyun Park

Diabetes 2017;66:1755–1757 | https://doi.org/10.2337/dbi17-0012

PEDIATRIC OBESITY
ORIGINAL RESEARCH
doi:10.1111/ijpo.12106

Prenatal and early life exposure to traffic pollution and cardiometabolic health in childhood

A. F. Fleisch[1], H. Luttmann-Gibson[2], W. Perng[3], S. L. Rifas-Shiman[4], B. A. Coull[5], I. Kloog[6], P. Koutrakis[2], J. D. Schwartz[2], A. Zanobetti[2], C. S. Mantzoros[7], M. W. Gillman[4,8], D. R. Gold[2,9] and E. Oken[4,8]

1418 dzieci z okolicy Bostonu
Śr. w wieku 3.3. i 7.7. lat
Ocena antropometryczna i analiza biochemiczna kardiometabolicznych czynników ryzyka
Ocena korelacji ww. z narażeniem ich matek w okresie ciąży na zanieczyszczenie powietrza i zamieszkanie blisko ulicy

Background: Prenatal exposure to traffic pollution has been associated with faster infant weight gain, but implications for cardiometabolic health in later childhood are unknown.

Methods: Among 1418 children in Project Viva, a Boston-area pre-birth cohort, we assessed anthropometric and biochemical parameters of cardiometabolic health in early (median age 3.3 years) and mid- (median age 7.7 years) childhood. We used spatiotemporal models to estimate prenatal and early life residential $PM_{2.5}$ and black carbon exposure as well as traffic density and roadway proximity. We performed linear regression analyses adjusted for sociodemographics.

Results: Children whose mothers lived close to a major roadway at the time of delivery had higher markers of adverse cardiometabolic risk in early and mid-childhood. For example, total fat mass was 2.1 kg (95%CI: 0.8, 3.5) higher in mid-childhood for children of mothers who lived <50 m vs. ≥200 m from a major roadway. Black carbon exposure and traffic density were generally not associated with cardiometabolic parameters, and $PM_{2.5}$ exposure during the year prior was paradoxically associated with improved cardiometabolic profile.

Conclusions: Infants whose mothers lived close to a major roadway at the time of delivery may be at later risk for adverse cardiometabolic health.

Jerrett et al. Environmental Health 2014, 13:49
http://www.ehjournal.net/content/13/1/49

RESEARCH Open Access

Traffic-related air pollution and obesity formation in children: a longitudinal, multilevel analysis

Michael Jerrett[1*], Rob McConnell[2], Jennifer Wolch[3], Roger Chang[2], Claudia Lam[2], Genevieve Dunton[2], Frank Gilliland[2], Fred Lurmann[4], Talat Islam[2] and Kiros Berhane[2]

4550 dzieci w wieku 5-11 lat
Kalifornia
4 lata obserwacji
Ocena ruchu ulicznego i wywołanego nim zanieczyszczenia powietrza vs. roczny przyrost BMI
Różnica ok. 13.6% pomiędzy najniższym i najwyższym percentylem zanieczyszczeń

Abstract

Background: Biologically plausible mechanisms link traffic-related air pollution to metabolic disorders and potentially to obesity. Here we sought to determine whether traffic density and traffic-related air pollution were positively associated with growth in body mass index (BMI = kg/m^2) in children aged 5–11 years.

Methods: Participants were drawn from a prospective cohort of children who lived in 13 communities across Southern California (N = 4550). Children were enrolled while attending kindergarten and first grade and followed for 4 years, with height and weight measured annually. Dispersion models were used to estimate exposure to traffic-related air pollution. Multilevel models were used to estimate and test traffic density and traffic pollution related to BMI growth. Data were collected between 2002–2010 and analyzed in 2011–12.

Results: Traffic pollution was positively associated with growth in BMI and was robust to adjustment for many confounders. The effect size in the adjusted model indicated about a 13.6% increase in annual BMI growth when comparing the lowest to the highest tenth percentile of air pollution exposure, which resulted in an increase of nearly 0.4 BMI units on attained BMI at age 10. Traffic density also had a positive association with BMI growth, but this effect was less robust in multivariate models.

Conclusions: Traffic pollution was positively associated with growth in BMI in children aged 5–11 years. Traffic pollution may be controlled via emission restrictions; changes in land use that promote jobs-housing balance and use of public transit and hence reduce vehicle miles traveled; promotion of zero emissions vehicles; transit and car-sharing programs; or by limiting high pollution traffic, such as diesel trucks, from residential areas or places where children play outdoors, such as schools and parks. These measures may have beneficial effects in terms of reduced obesity formation in children.

Keywords: Childhood obesity, Air pollution, Traffic, California

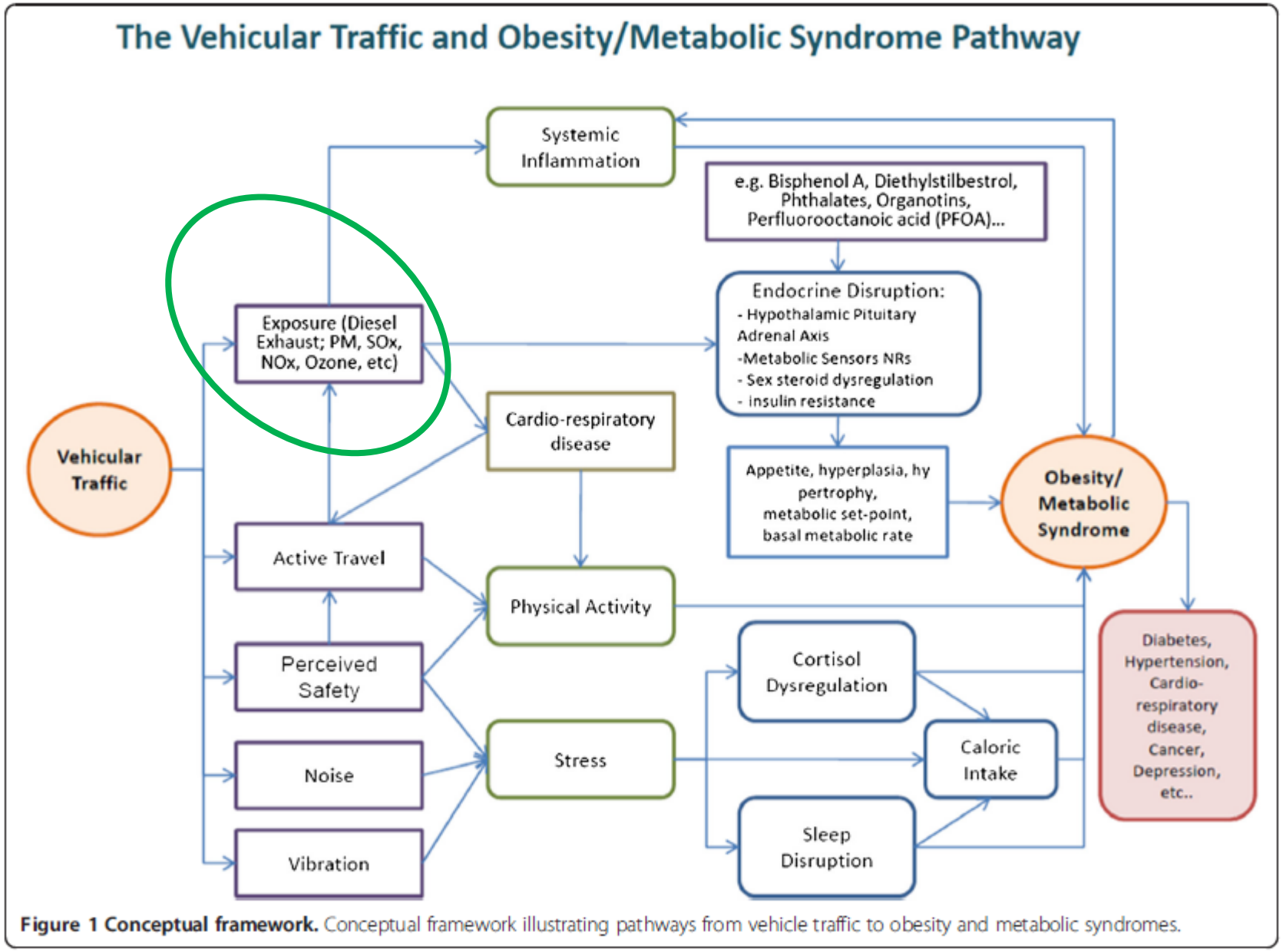


Figure 1 Conceptual framework. Conceptual framework illustrating pathways from vehicle traffic to obesity and metabolic syndromes.

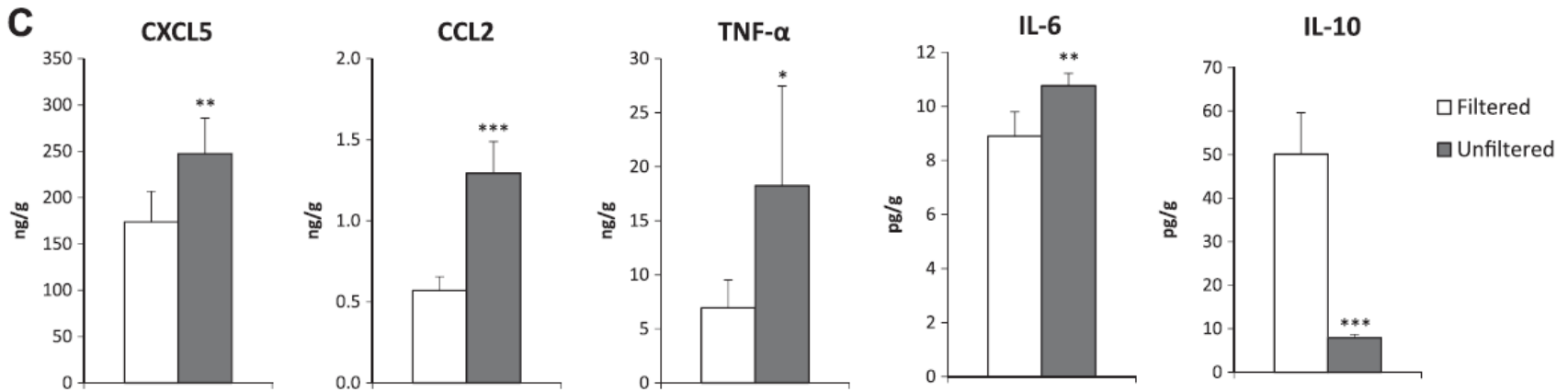
Chronic exposure to air pollution particles increases the risk of obesity and metabolic syndrome: findings from a natural experiment in Beijing

Yongjie Wei,[*,†] Junfeng (Jim) Zhang,[‡,§,1] Zhigang Li,[†] Andrew Gow,[¶] Kian Fan Chung,[‖] Min Hu,[*] Zhongsheng Sun,[#] Limin Zeng,[*] Tong Zhu,[*] Guang Jia,[**] Xiaoqian Li,[†] Marlyn Duarte,[‡] and Xiaoyan Tang[*,1]

ABSTRACT: Epidemiologic evidence suggests that air pollution is a risk factor for childhood obesity. Limited experimental data have shown that early-life exposure to ambient particles either increases susceptibility to diet-induced weight gain in adulthood or increases insulin resistance, adiposity, and inflammation. However, no data have directly supported a link between air pollution and non-diet-induced weight increases. In a rodent model, we found that breathing Beijing's highly polluted air resulted in weight gain and cardiorespiratory and metabolic dysfunction. Compared to those exposed to filtered air, pregnant rats exposed to unfiltered Beijing air were significantly heavier at the end of pregnancy. At 8 wk old, the offspring prenatally and postnatally exposed to unfiltered air were significantly heavier than those exposed to filtered air. In both rat dams and their offspring, after continuous exposure to unfiltered air we observed pronounced histologic evidence for both perivascular and peribronchial inflammation in the lungs, increased tissue and systemic oxidative stress, dyslipidemia, and an enhanced proinflammatory status of epididymal fat. Results suggest that TLR2/4-dependent inflammatory activation and lipid oxidation in the lung can spill over systemically, leading to metabolic dysfunction and weight gain.—Wei, Y., Zhang, J., Li, Z., Gow, A., Chung, K. F., Hu, M., Sun, Z., Zeng, L., Zhu, T., Jia, G., Li, X., Duarte, M., Tang, X. Chronic exposure to air pollution particles increases the risk of obesity and metabolic syndrome: findings from a natural experiment in Beijing. FASEB J. 30, 2115–2122 (2016). www.fasebj.org

Model zwierzęcy
Ciężarne szczury narażone na niefiltrowane powietrze znacznie cięższe przed porodem podobnie jak ich 8-tygodniowi potomkowie
U rodziców i potomków obecne cechy okołonaczyniowego i okołooskrzelowego stanu zapalnego oraz układowego stresu oksydacyjnego i dyslipidemii

The FASEB Journal Vol.30, No.6 , pp:2115-2122, October, 2017



C) Cytokine/chemokine concentrations in epididymal fat tissue of male rats. $^{*}P < 0.05$, $^{**}P < 0.01$, $^{***}P < 0.001$.

TABLE 2. *Biomarkers of male rats exposed prenatally and continuously for 8 wk to unfiltered or filtered Beijing air*

	Filtered ($n = 10$)		Unfiltered ($n = 10$)		
Biomarker	Mean	SD	Mean	SD	*P*
LDL (μM)	50.7	8.02	63.0	9.18	0.005
HDL (μM)	58.8	7.84	46.5	3.49	0.001
TG (mM)	2.93	2.07	5.45	1.41	0.006
TC (mM)	23.4	5.39	27.2	2.15	0.058
MDA (nM)	13.4	6.84	25.5	6.05	0.001
GSH (ng/ml)	8.69	3.51	4.69	2.66	0.011
GLP-1 (pM)	30.9	14.0	17.4	12.0	0.033

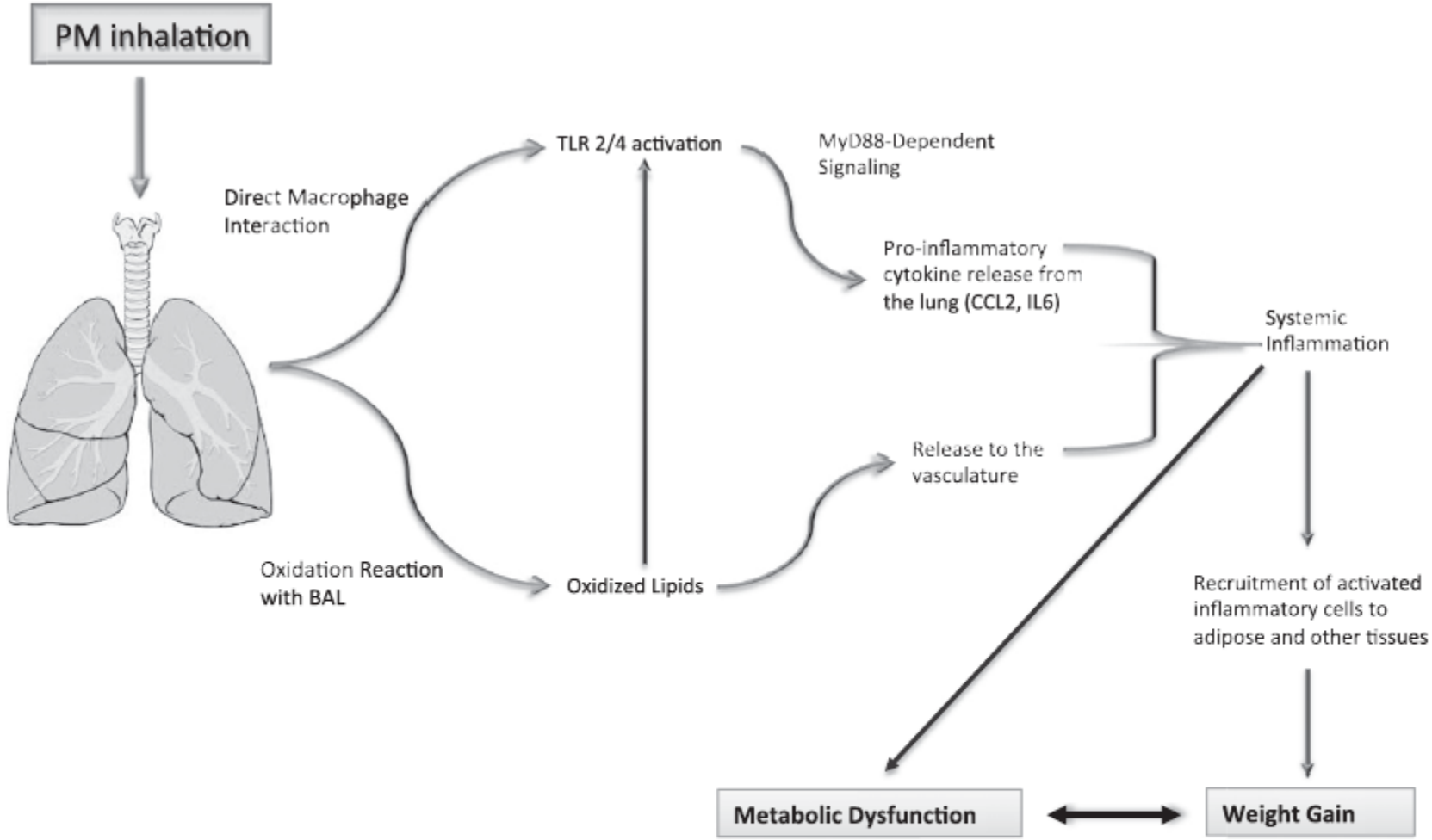


Figure 4. Mechanistic framework explaining how inhaled air pollutants disrupt metabolic state. Inhalation of air pollutants (especially particulate matter) can lead to direct activation of alveolar macrophages through TLR2/4-dependent mechanisms and generation of oxidized lipids within lung lining. These oxidized lipids can further activate inflammatory processes through TLR2/4 binding or be released to vasculature, where they will initiate systemic inflammation and oxidative stress responses. TLR2/4-dependent inflammatory activation, through activity of MyD88, will lead to release of proinflammatory cytokines, such as CCL-2 and IL-6, from lung, also generating systemic inflammation. These systemic inflammatory activation processes, along with loss of anti-inflammatory functions from incretins such as GLP-1, will lead to increased recruitment of activated inflammatory cells to tissues and in particular adipose. Recruitment of such cells to adipose will worsen metabolic profile, leading to weight gain and metabolic disease state. Prenatal exposure may enhance cellular and functional responses shown here.

Rev Endocr Metab Disord (2017) 18:207–214
DOI 10.1007/s11154-016-9388-6

Low serum vitamin D-status, air pollution and obesity: A dangerous liaison

Luigi Barrea[1] · Silvia Savastano[2] · Carolina Di Somma[3] · Maria Cristina Savanelli[1] · Francesca Nappi[1] · Lidia Albanese[2] · Francesco Orio[4] · Annamaria Colao[2]

otyłość

↓

zmniejszone stężenie wit. D3

bo mniejsza lokalna aktywacja (alfa1hydroksylacja) prohormonu, magazynowanie lipofilnej wit.D w tkance tłuszczowej; każdy wzrost BMI o 1 połączony jest ze spadkiem stężenia 25OHwit.D o 1.15%

zanieczyszczenie powietrza

↓

zmniejszona synteza wit.D3 z 7-dehydrocholesterolu w skórze

↓

Wit.D wpływa na ekspresję genów włączonych w metabolizm tkanki tłuszczowej (m.in. hamuje różnicowanie preadipocytów, lipolizę, produkcję prozapalnych cytokin przez tkankę tłuszczową…, wtórnie pobudza PTH, które poprzez wzrost stęż. Ca 2+ w komórkach adipocytów powoduje wzrost ekspresji syntetazy kwasów tłuszczowych- w efekcie tworzenie depozytów lipidowych i zmniejszenie lipolizy…)

↓

otyłość

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zaburzenia węglowodanowe

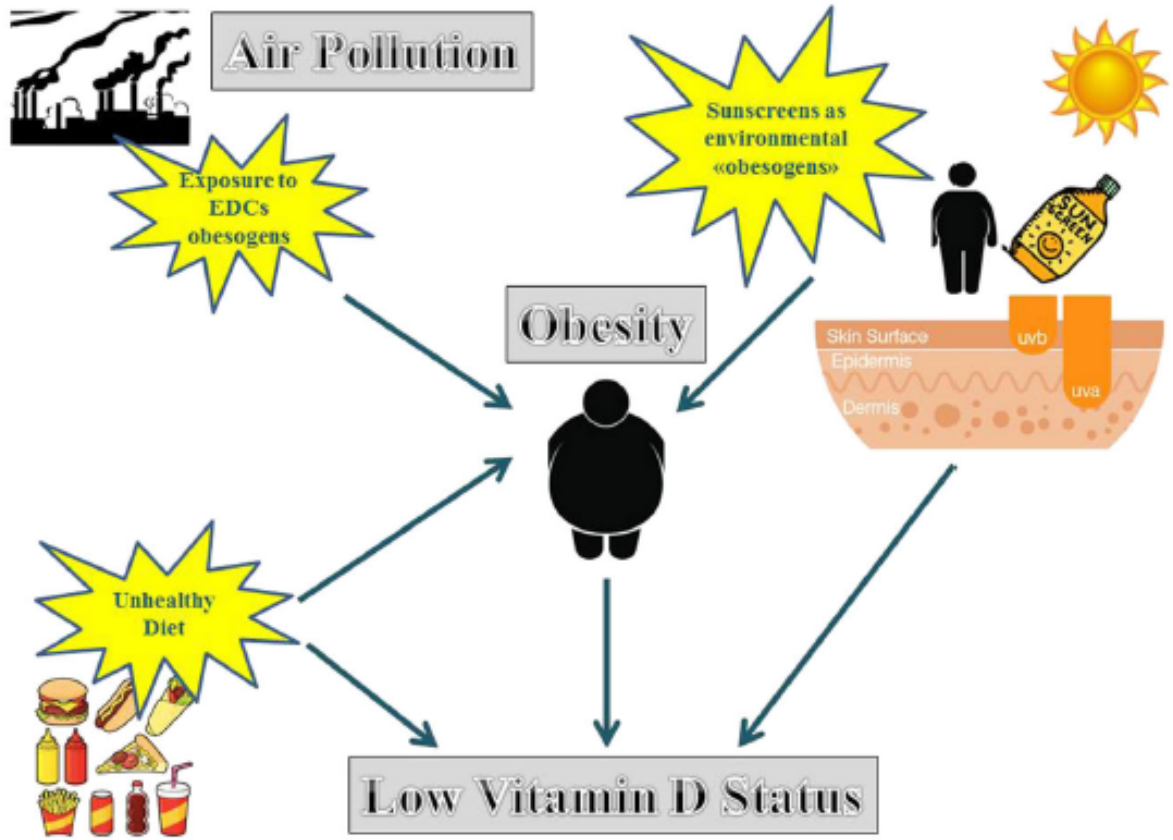

Fig. 1 An intriguing hypothesis can be proposed, that, combined with unhealthy diet and lifestyle, the inadequately wearing of some sunscreens might cause a low vitamin D status either directly, by reducing the absorption of solar UVB radiation, and also indirectly, by inducing obesity, possibly by acting as environmental "obesogens". Sequestration of vitamin D in the adipose tissue further worsens the low vitamin D status and increases the metabolic risk in obese individuals

Podsumowanie

- Zanieczyszczenie powietrza jest istotnym czynnikiem ryzyka wystąpienia cukrzycy i innych zaburzeń gospodarki węglowodanowej
- Mechanizmy oddziaływania zanieczyszczenia powietrza na metabolizm węglowodanów są złożone i nie do końca poznane.
- Działania ograniczające zanieczyszczenie środowiska są obiecującą opcją profilaktyki zagrażającej światu pandemii cukrzycy


risk
glucose
treatable
insurance
health
surveillance
addiction
lifestyles
injected
complications
bloody
hypodermic
finger
drug
human
pharmaceutical
hyperglycemia
pharmacology
diagnosed
lancet
medicine
analyzing
surgery
glucometer
control
emergency
patient
glucuose
physical
symptoms
adults
needle
people
bleed
sugar
pain
immunization
diagnostic
produces
prevention
representation
doctor
equipment
prescription
cells
illness
hemoglobin
care
food
mellitus
sickness
glucometer
analysis
stop
measurement
factors
hospital
level
capsule
disease
diabetes
fear
sampling
clinic
biotechnology
pill
glucosemeter
blood
instrument
stick
hypoglycemia
healthcare
protection
treatment
vegetables
test
healthy
pharmacy
vaccine
microscope
bad
insulin
research
examining
causes
chronic
medical
hurt
prick
laboratory
syringe
meter
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heart
vial
drop
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agnessang.flog.pl


doctor
equipment
prescription
cells
illness
hemoglobin
care
food
mellitus
stop
air pollution