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Evidence based public health – the example of air pollution

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Summary

Evidence Based Medicine (EBM) has become a common paradigm in medicine. The notion that action should be based on scientific evidence also applies to public health. The assessment of evidence and the application of evidence based action is a challenge both in EBM and public health. The focus of this article is Evidence Based Public Health (EBPH). We use the issue of air pollution related health as an educational example for EBPH.

Key words: evidence based public health; air pollution; environmental health

Introduction

Evidence Based Medicine (EBM) has become a common paradigm in medicine [1]. It is the conscientious, explicit, and judicious use of current best evidence in making decisions about the care of individual patients. The practice of EBM means integrating clinical expertise with the best available systematic research.

In contrast, the main goal of public health is to assure, maintain, protect, promote, and improve the health of populations. Instead of dealing with patients, public health acts on the level of groups or entire populations of both diseased and healthy or "not yet diseased" people. Preferably, this process ought to be based on scientific evidence, thus Evidence Based Public Health (EBPH) is the natural extension of EBM to the public health field. However the term and concept of EBPH are far less known. A December 2008 PubMed search for EBPH provides 84 entries while EBM results in more then 33 000 items). This article demonstrates the principles and relevance of *Evidence Based Public Health* (EBPH) using air pollution as a classic public health problem. We juxtapose the discussion with EBM.

Evidence of adverse effects of ambient air pollution on human health has substantially increased over the past 20 years with associations between exposure to air pollution and a wide range of health problems established [2–5]. Based on these findings and their implications for public health, many countries have implemented regulatory actions to improve air quality. But do improvements of air quality result in better health?

To discuss this, we first describe the model of evidence based action as applied to public health. We then review and discuss the link between air pollution exposure (or changes thereof), public health, and policy action. The article is not a full review of all related issues but we refer to some recent or particularly relevant reviews and studies to underscore our educational example of EBPH.

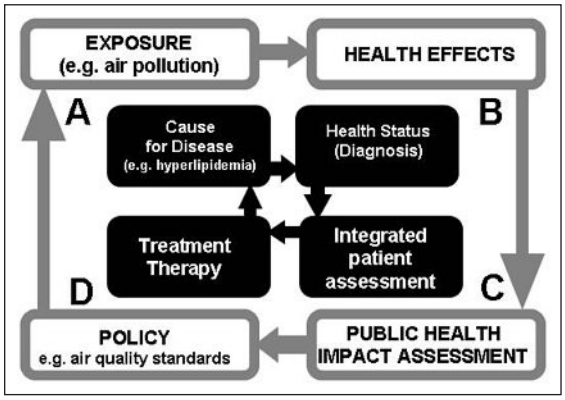
The model of evidence based action

The well established paradigm of evidence based medicine (EBM) is a guiding principle of the work of clinicians, formalised in the inner cycle of figure 1. As people become "patients" due to known or unknown factors (Box A of the inner cycle in fig. 1), they usually approach physicians with their health problem. The disease history (anamnesis) and a range of techniques and tools serve to establish a diagnosis (Box B). In the next step – the overall assessment of the patient's situation (Box C) – the diagnosis plays an important role, but a range of other factors are usually taken

No conflict of interest.

Figure 1

The cycle of clinical work (inner cycle, black boxes) and public health (outer cycle, grey boxes) underlying 'evidence based medicine', and 'evidence based public health', respectively. In clinical work, cause(s) (inner Box A) of health problems (B) result in a doctors' diagnosis. The assessment of the overall situation of the patient (C) determines the treatment strategy (D) to positively affect the causes (A) and/or health (B). In public health, some "exposure" (A) may cause health problems in the population (B). The assessment of its relevance (C) may result in a policy (D) to abate the exposure (A) and improve public health (B). Ideally, all steps in both cycles are based on scientific evidence – evidence based medicine and public health, respectively.



into account. For example, patients' preferences and age, a history of treatment problems, failures or successes, the social setting (as a potential determinant of compliance), or the duration, logistic implications and costs of various treatment options may be considered. This assessment will ultimately lead to some treatment or action (Box D). In an ideal world of EBM, all these steps will be based on scientific evidence. The intention of the therapy (Box D) is to interfere with the causes of the problem (Box A) and to positively affect health and/or quality of life (Box B). Randomised clinical trials play a crucial role in establishing scientific evidence in the EBM model.

The paradigm of *Evidence Based Public Health* is similar. The related public health decision process is shown in the outer cycle of figure 1. These boxes and concepts are discussed in more details in the following sections. Ambient air pollution is used as an example of an exposure, hazard, or cause of diseases (Box A), but the paradigm applies to other public health problems.

Air pollution as an hazardous exposure

Ambient air pollution is a complex mixture of hundreds of substances including particles, gases, and semi-volatiles. Being ubiquitous, all people are exposed to air pollution, at least to some degree, throughout their life.

To simplify our EBPH example, we focus on only one aspect of air pollution, namely particulate matter (PM). While the PM content found in the air pollution mixture is unlikely to be the only cause of the range of effects related to ambient air pollution, PM is successfully used as a marker of pollution in epidemiological research which reported in particular associations between PM up to 2.5 or 10 micrometer in diameter (PM2.5 and PM10) and a range of health problems [2, 4]. Experimental studies confirm various health relevant toxicological features of PM and its interactions with other pollutants is subject to most recent investigations [6–8].

Ambient particles are loaded with numerous pollutants, including carcinogens, metals, allergenic compounds, endotoxins, and hundreds of other substances. The finer particles (particles with diameters below 2.5 μm) may be an especially effective media to transport these constituents deeply into the lung. Physicians prescribing glucocorticosterid inhalers make use of this size-dependent physical property of fine particles [9]. Once in the lung PM can activate a cascade of events – desirable in case of treatment, but undesirable in case of ambient PM. The latter cause oxidative stress, and both local pulmonary and systemic inflammatory responses have been described in conjunction with changes in autonomic function and coagulation pathways as most relevant mechanisms induced by PM, PM constituents, and also by other ambient pollutants [10–13]. Pollutants also interfere with ciliary clearance in the airways resulting in increased bacterial and/or viral loads [14].

Air pollution as an exposure that affects health

In the clinical setting it is the patient who actively seeks advice. The public, however, does not by itself disclose its state of health. Research is needed to evaluate the public's health and its determinants. Table 1 summarises the more often investigated effects of air pollution [4]. The acute effects of exposure to ambient air pollution are particularly well investigated. Hundreds of studies confirm that health problems in the population increase on days with increased pollution, with effects ranging from cardiorespiratory death, myocardial infarction, stroke, asthma attacks, or the

exacerbation of respiratory symptoms to more subtle functional changes in the airways and the blood [2, 3].

Cohort studies that repeat measurements of relevant underlying (preclinical) pathologies are providing important evidence that exposure to ambient pollution also contributes to the development of chronic pathologies that ultimately result in morbidity and chronic diseases. While air pollution contributes to disease and death, we focus on two examples using preclinical markers of complex pathologies. This further underscores differences between the clinical approach, where diseases are of primary interest, and public health research on the causes and prevention of morbidity, where preclinical stages can indeed be of high value.

An example of a widely used preclinical marker of chronic disease is functional measurement of the lung (e.g., vital lung capacities, FVC and FEV_1). The lung capacity reflects far more than the state of the lung [15]. Poor lung function (LF) correlates with systemic inflammation and a range of chronic inflammatory diseases, thus LF can be seen as a systemic marker of health. In fact, LF is one of the strongest predictors of life expectancy [15]. Figure 2 provides a life-time model of lung function in 'normal populations' and among those exposed to factors that affect functional growth and decline with ageing.

The Southern Californian Children's Health Study is the largest and most extensive air pollution study focusing on air pollution and lung function growth, with measurements taken every year, throughout childhood [16, 17]. The 8-year follow-up showed that ambient air pollution [17], and in particular pollution from traffic [16] affected the development of the lung. At age 18, lung development terminated at substantially lower levels among adolescents growing up in communities with the highest pollution. Having poor lung function (<80% predicted) was some 4 to 5 times more frequent in adolescents with the highest 8-year exposure. This and other studies [18] give strong evidence that air pollution affects the development of children's lungs (fig. 2) [15].

Another chronic pathology of high public health relevance is atherosclerosis [19]. Figure 3 provides a life-time model of atherogenesis in the general population. The model uses the intima-

Figure 2

Life-time course of pulmonary function (lung volume expired during the first second – FEV_1) among a healthy population (solid line). The fast decline among adults (dotted line) reflects a population of smokers. The dashed line is expected under the hypothesis that chronic exposure to ambient air pollution affects lung growth (childhood) and decline (adulthood), leading to lower achieved levels (plateau) and to reaching levels of disability earlier in life.

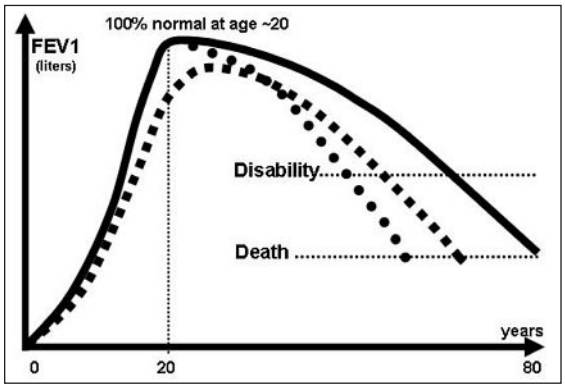


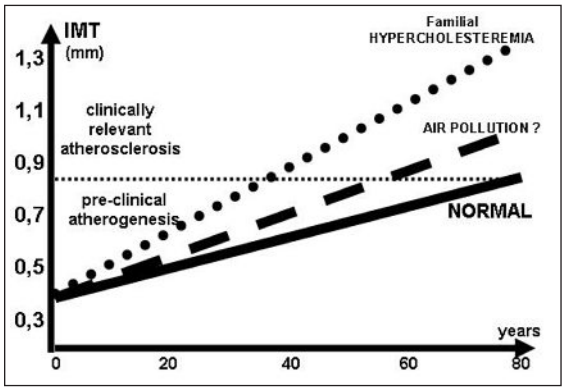
Table 1

Overview of respiratory and cardiovascular physiologic, structural, and clinical outcomes reported to be associated with exposure to ambient air pollution. (? means evidence is sparse) (Adapted from [4]).

Lung	Heart/Vasculature/Systemic
Physiologic & structural changes	
Forced expiratory volume and flows	Heart rate (increase)
Inflammatory mediators (local and systemic)	Heart rate variability (decrease)
Air way remodeling	Blood pressure
	Blood coagulation factors
	Vascular reactivity
	Inflammatory mediators
	Vessel structure
Acute clinical measures of effect	
Upper respiratory symptoms	Thrombosis
Lower respiratory symptoms	Myocardial infarction
Acute exacerbations of chronic bronchitis, asthma	Arrhythmia
Asthma medication use	Stroke
Death	Death
School/work absences	Use of health care resources due to all of the above
Use of health care resources due to all of the above	
Cumulative clinical effects	
Reduced lung growth	Reduced life expectancy (premature cardiovascular death)
Reduced small airway function	? Atherosclerosis
Chronic bronchitis	? Foetal growth retardation
Lung cancer	? Other reproductive outcomes
Reduced life expectancy	? Neurodevelopment
? Asthma onset	

Figure 3

Life-time course of atherogenesis measured with the intima-media thickness (IMT) of the carotid artery (in mm) used as a marker of the degree of atherosclerosis. The solid line relates to atherogenesis among a healthy "normal" population. The dotted line is a model of extreme atherogenesis, e.g., due to familial hypercholesteraemia (20). The dashed line would be observed under the hypothesis of accelerated atherogenesis due to life-time exposure to ambient air pollution. Accelerated atherogenesis (thicker arteries) results in earlier occurrence of cardiovascular diseases.



media thickness of the carotid arteries as a measure of the degree of atherosclerosis in the vasculature [20]. It is a simple marker of a rather complex pathology, which results in thicker and stiffer arteries. As shown in figure 3, atherogenesis is a lifelong process related to "ageing" with atherogenic risk factors accelerating the development of atherosclerosis, considered to be an inflammatory disease. The observation of systemic inflammatory responses following exposure to ambient PM raised a provocative hypothesis: does air pollution enhance the most important systemic chronic inflammatory pathology, namely atherosclerosis – "*cause number one*" of mortality and morbidity in our societies? Under this hypothesis, one expects subjects with higher long-term exposure to ambient air pollution to experience faster thickening of the artery walls and earlier signs of calcification (dashed line in fig. 3). The very few studies that have so far investigated this hypothesis in humans appear to confirm abundant evidence from animal studies [21–23].

While exposure to air pollution is ubiquitous, more recent studies highlight hot spots of very high concentrations of a range of toxic substances in proximity to busy roads. Depending on traffic density, concentrations of some pollutants may be some 5–10 times higher along streets compared to levels just ~100 meters off the roads [24]. Numerous studies now investigate the health of those living in the buffer along busy roads. A recent review concluded that children growing up in such locations are at increased risk of developing asthma – the most important chronic disease in children [24]. Further improvements in the identification of susceptible sub-groups – e.g., defined by genetic variants – may expand the list of diseases considered to be caused by such traffic-related pollutants [25].

Despite a range of open questions, the evidence clearly points at air pollution as the cause of many health ailments. The list of acute and chronic cardio-respiratory morbidities that are due to ambient air pollution is ever increasing and explains the observed shortening in life expectancy among those experiencing higher exposure to air pollutants. Ongoing research addresses in particular prenatal and early life reproductive effects of air pollution and novel lines of research start to integrate cognitive function to assess neurodevelopment and neurodegenerative pathways [26]. Further research is needed in the area of chronic diseases among adults, including asthma, COPD, and chronic cardiovascular pathologies. The relevance of factors that may amplify or reduce (or eliminate) adverse effects of air pollution (e.g., diet, atopy, obesity, genetic factors, environmental co-exposures, medical treatments etc.) are subject of ongoing investigations, and results may become of clinical relevance.

Assessing the size of the problem

Similar to clinical decision making, an integrated assessment is needed to explore the relevance of the public health problem (Boxes C in fig. 1) prior to prescribe an adequate "treatment" and take action (Boxes D).

In the air pollution domain, this step – often called "impact assessment", translates research evidence into estimates of the overall burden of health that can be attributed to air pollution [27]. A range of such assessments have been conducted in the past years employing established methods to provide crude estimates for a few health problems such as death (or life expectancy), hospital admissions, and respiratory problems attributable to air pollution [28–30]. These studies have usually concluded that a large proportion of common health problems is attributable to air pollution providing a strong quantitative argument to take action. Table 2 provides some examples of the burden attributable to air pollution for selected outcomes in studies considering different geographical scales around Europe. In Switzerland, government took a leading role in the mid 1990's in triggering such studies [29, 31].

Similar to the clinical situation, this step (Box C) may take into account a broad range of factors that go well beyond health alone. In fact, the Swiss studies mentioned above were part of the govern-

Table 2

Examples of the health burden attributed to air pollution in selected areas of Europe.

Pollutant/scenario[a]/ health burden	Europe 25 [44]	Europe 26 urban cities [45]	Switzerland [29]	13 Italian cities [46]	Barcelona metropolitan area [35]
Population exposed (millions)	~450	~41.5	~7.3	~10.0	~3.9
PM_{10}					
Current levels (annual mean)	–	54 µg/m³ [b]	21 µg/m³	45 µg/m³	50 µg/m³
Reduction scenario	–	Reduction annual level to 40 µg/m³	Reduction annual level to 7.5 µg/m³	Reduction annual level to 40 µg/m³	Reduction annual level to 40 µg/m³
Health burden by outcome					
All cause mortality (long-term exposure)	–	8,550	3,314	2,270	1,200
Life year lost	3,618,700	–	–	–	8,200
Hospital admissions for respiratory causes	62,000	–	1,308	225	390
Hospital admissions for cardiovascular causes	38,300	–	2,979	176	210
Chronic bronchitis adult	163,800	–	45,446	1,114	1,900
$PM_{2.5}$					
Current levels (annual mean)	–	33 µg/m³ [c]	–	–	–
Reduction scenario	Adopt regulated emissions in year 2000 in all countries	Reduction to 20 µg/m³	–	–	–
Health burden by outcome					
All cause mortality (long-term exposure)	347,900	11,375	–	–	–
Life year lost	3,618,700	–	–	–	–
Hospital admissions for respiratory causes	62,000	–	–	–	–
Hospital admissions for cardiovascular causes	38,300	–	–	–	–
Chronic bronchitis adult	163,800	–	–	–	–
Ozone					
Current levels (annual mean)		–	–	–	–
Reduction scenario	Adopt regulated emissions in year 2000 in all countries	–	–	Reduction countries all days ≥70 µg/m³	–
Health burden by outcome					
Acute mortality	21,400	–	–	516	–
Respiratory hospital admissions	14,000	–	–	228	–

[a] Assumes a reduction of current levels to levels proposed in scenario
[b] Only 8 cities with levels above 40 µg/m³
[c] Only 11 cities with levels above 20 µg/m³

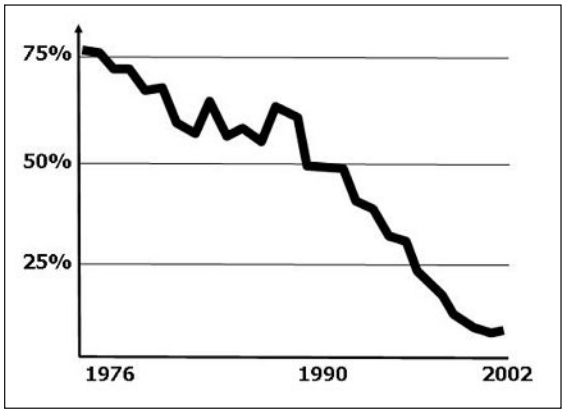
mental strategy to internalise the so-called "external" costs of goods movement – usually covered by the tax payer – into Swiss road pricing policies. Thus, like other side effects of road transport, the health effects attributable to traffic related air pollution had first to be quantified [32]. So far, impact assessments do not integrate the new evidence of increased health hazards occurring among those living along busy roads [24], but the respective methods are now under development [27].

Taking action – the "treatment" step

Whether the health burden estimated in the above studies – and thus the benefit of preventive action – ought to be considered large or small is not just a scientific question but a political one. Policy makers in many countries have considered the problem severe enough to take action (outer Box D in fig. 1). In clinical setting, guidelines may set a target for the treatments – for example the

Figure 4

Example of the improvements of air quality in the Los Angeles area due to rigorous implementation of a range of policies to comply with the most stringent air quality standards in the world (State of California). % of days per year exceeding 2002 standards for carbon monoxide, ozone and PM_{10} (1976–2002) [55].



levels of lipids to be achieved through prescription of lipid lowering agents [33, 34]. A similarly effective strategy in air quality regulation is to set targets for ambient concentrations of pollutants. Table 3 summarises current air quality standards set for different areas of the world. A range of local, regional, or national actions are usually implemented to achieve the pre-set targets. Several examples exist confirming that such policies can dramatically improve air quality. For example the Southern Californian Los Angeles region gives an impressive example of radical improvements achieved over the last 50 years with policies targeting all key emissions such as those from traffic, power plants, industries, small businesses, and households (fig. 4). The same is true for the London metropolitan area. Today London as well as large parts of Southern California have better air quality than many European cities where air quality regulation was not a priority [35]. Unfortunately, targets set for the European Community (EC) lag far behind other national or local policies, and largely ignore scientific evidence. Ironically, the latter is to a substantial degree based on acclaimed European research, partly funded by the EC [36].

Multiple causes are behind the discrepancies in policy making (table 3) as legislature considers factors other than public health. This is comparable to the clinical situation where treatment decisions often differ between countries and even between physicians within the same region. This occurs despite EBM and guidelines for clinical practice, and the same applies to EBPH. While costs are often used in the argumentation against clean air policies, risk assessors conclude that the benefits of the U.S. Clean Air Act to be far larger than its costs [37].

Table 3

Examples of clean air policy targets set by different authorities. Note that "Air Quality Standards" are not reflecting "levels of no effect" or "safe levels". Researchers failed to identify thresholds of "no effect" and it is assumed that susceptible individuals to be affected by exposures at very low concentrations. (Modified from [47]).

Source	Sulfur dioxide (µg/m³)				Nitrogen dioxide (µg/m³)			PM_{10} (µg/m³)		$PM_{2.5}$ (µg/m³)		Ozone (µg/m³)	
	1 year	24 hours	1 hour	10 minutes	1 year	24 hours	1 hour	1 year	24 hours	1 year	24 hours	8 hours	1 hour
WHO [47]		20		500	40		200	20	50[a]	10	25[a]	100	
European Union [48]		125	350		40		200	40	50[b]			120	
Switzerland [49]	30	100[d]			30	80[d]		20	50[d]			120[d]	
France [50]	50	125[a]	350[f]		40		200[e]	40	50[b]				
Sweden [51]		100	200		40	60	90	40	50				
United Kingdom [52]		125[a]	350[f]	266[b]	40		200[e]	40	50[b]	25		100	
United States [53]	78	366			100			50	150	15	65	157	
California [54]		105[c]	655				470[c]	20	50	12	65	137	180[c]

[a] Not to be exceeded more than 3 days per year
[b] Not to be exceeded more than 35 days per year
[c] Photochemical oxidants
[d] Not to be exceeded more than one time per year
[e] Not to be exceeded more than 18 times a year
[f] Not to be exceeded more than 24 times a year

Cleaner air – better public health?

The ultimate EBM question of a clinician is whether his or her treatment and action truly affects the course of the disease (inner Box D in fig. 1). In fact, treatment should not only change a marker of health e.g., lower the lipid levels, but improve health, quality of life, or health prognosis (Box B) in a relevant way [33]. Similarly, the ultimate question of our EBPH example is not only whether clean air policies reduce air pollution (Box A) – which has been proven to be the case time and again – but indeed whether they result in improved public health (Box B).

For obvious reasons, double blind randomised experiments are not a choice in this field to establish "the ultimate evidence" as done in EBM. In fact, a formal assessment of the health consequences of public policies is often entirely lacking. In the field of air pollution instead, the term "*accountability studies*" has been coined for studies that address health effects of (often policy driven) air quality changes [38]. Such studies are not easily implemented and come with many caveats. Improvements of air quality usually take many years, thus, the interpretation of health changes may be flawed as many other health relevant factors may change too. However, an increasing number of studies successfully investigate the "accountability evidence". We mention in particular two examples that relate to the objective health outcome discussed above, namely lung function development.

The Los Angeles based Children's Health Study team visited children who had moved, during follow-up, to other places in the Western U.S. [39]. As a side effect of residential changes – usually driven by parents' careers – exposure to air pollution may also change. "Movers", thus, became participants of a "natural experiment", and, in fact, lung growth correlated with the change in air quality: those moving into cleaner communities benefited with a faster lung development while the others experienced growth deficits [39].

Among adults, ageing is expected to result in a decline in functional lung volumes such as the vital capacity or the volume expired in the first second (FVC and FEV_1) (fig. 2). The natural annual decline in FEV_1 of some 30–40 ml can be affected by many factors. The best known is smoking where smoking cessation leads, within a couple of years, to a normalisation of the annual decline. But do improvements in air quality slow down the functional loss related to ageing? The Swiss Study on Air Pollution and Lung Diseases (SAPALDIA) had the unique opportunity to investigate this question [40]. The development of state-of-the-art air pollution modelling tools allowed the characterisation of home outdoor air quality for each subject and all residential locations [41]. Thus, each subject's 11 year change in exposure could be estimated and compared with the 11 year change in lung function. Due to the long-standing record of air quality regulations implemented by Swiss authorities, the majority of SAPALDIA participants experienced an improvement in home outdoor air quality. As in case of smoking cessation, a reduction in air pollution was significantly associated with a slower decline of lung function. Other studies confirm the benefit of clean air policies [38]. In fact, improvements in Swiss air quality were also paralleled by reductions in respiratory health problems among Swiss children [42] – an observation very similar to the ones seen during the dramatic reductions in air pollutants in former Eastern Germany [43].

Conclusion

The air pollution example demonstrates similarities and differences between evidence based *medicine* and *public health*. Both adhere to the vision to base action on scientific evidence. The central role of research is evident in both cycles of figure 1 although our example of EBPH research involves many more scientific disciplines than EBM usually does. The main difference between the two is in the implementation of action. In EBM, the central role of the physician is evident and a great opportunity (Box B, C, D of the inner cycle). Applied EBM is rewarding for the patient and the physician – the two key "actors" and partners in the clinical cycle of figure 1. This is very different in EBPH, at least in the example of air pollution.

First, the "actor" is neither the physician nor any uniquely defined constituency. Policy defines the action. The translation of evidence (Box B) into a policy framework (Box D) requires the sustained collaboration between scientists, health professionals, the complex world of policy making, and the public.

Second, the implementation of policies depends on a complex network of actors and agencies, ranging from engineers to industries, urban planners, or users of products. To line up these actors is more complex than letting physicians follow the vision of evidence based medicine and the current debate in European air quality standard setting gives a vivid example of a failure to line decision makers up to agree on science based standards [36].

Third, in our EBPH example – and in strong contrast to the clinical work – the beneficiary of the action cannot be individually identified. All

one can say is, e.g., that "many asthmatics" will suffer fewer attacks if air pollution decreases, fewer myocardial infarctions will show up in the emergency room, fewer cardio-respiratory problems will prevail etc. Lack of the "lobbying power" of beneficiaries further complicates the implementation of public health action. Instead, the impact assessment (Box C) becomes much more important (and complex) in EBPH than in most cases of clinical work. In EBPH, impact assessment tools are needed to communicate the overall size of the public health problem and the potential benefits of policies [30, 37].

In case of air pollution, many open questions that are not addressed in this article remain along the cycle shown in figure 1. However, the "unknowns" should not cloud the "knowns": a) scientific evidence is sufficient to consider current levels of ambient air pollution a cause of morbidity and mortality; b) the disease burden associated with current levels of air pollution is substantial; c) clean air policies provide successful tools that can indeed improve air quality in a sustained manner; d) such improvements of ambient air quality do result in better health and quality of life. The latter is and remains the ultimate goal of both evidence based medicine *and* evidence based public health.

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