Living conditions, social and psychological determinants of health and epidemiology – addressing the challenges of establishing causality and making evidence-based public health recommendations

Rapporteur’s report of the workshop
Impressum

Herausgeber
Deutsche Akademie der Naturforscher Leopoldina e. V. (Federführung)
– Nationale Akademie der Wissenschaften –
Jägerberg 1, 06108 Halle (Saale)

acatech – Deutsche Akademie der Technikwissenschaften e. V.
Residenz München, Hofgartenstraße 2, 80539 München

Union der deutschen Akademien der Wissenschaften e. V.
Geschwister-Scholl-Straße 2, 55131 Mainz

Redaktion
Dr. Kathrin Happe, Nationale Akademie der Wissenschaften Leopoldina
Dr. Alexandra Schulz, Nationale Akademie der Wissenschaften Leopoldina
Abteilung Wissenschaft – Politik – Gesellschaft (Leitung: Elmar König)
Kontakt: politikberatung@leopoldina.org

Zitiervorschlag
Living conditions, environmental, social, economic and psychological determinants of health – addressing the challenges of establishing causality and making evidence-based public health recommendations

Rapporteur’s report of the workshop
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A working group commissioned by the German National Academy of Sciences Leopoldina, the Union of the German Academies of Sciences and Humanities, and acatech – the German Academy of Science and Engineering prepared a statement on public health. Prior to setting up the working group, the three academies explored this diverse field through a series of workshops that started in March 2013 and continued in June 2013 and October 2013. The workshop topics were set by a planning group of Leopoldina’s Presidium with participation of the Standing Committee of the National Academy of Sciences Leopoldina. Each workshop covered one of seven topics (see below). The workshops were designed to bring together the latest facts and knowledge on each topic. Each workshop brought together the expertise and views of experts from Germany and abroad.

### The workshop series on public health

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Many correlations between living conditions (in a broad sense: encompassing environmental, social, economic and psychological conditions) and health are known. Examples are: mothers without a partner have worse health; low-income individuals have shorter lives; the optimum intake of alcohol for public health is 5 gram of alcohol or around half a glass; individuals living close to chemical waste sites have worse health. The interpretation of almost all of these correlations is controversial. Is it the chemical waste or the poverty of those who live there that causes ill health?

The key questions for this workshop are therefore: which of these correlations are actually based on causal relationships, and which are the causal mechanisms behind the correlation, such that public health recommendations can be built upon them? Examples from the 'WHO European Review of the social determinants of health and the health divide' will be used to illustrate how the challenges of interpreting evidence and making appropriate recommendations can be addressed.

The report documents the presentation and discussion at the Workshop on 19 June 2013. For the reader to be able to follow the chain of reasoning, it was aimed to stay close with the spoken words and to document also the variety of suggestions that were made, even if they are contradictory.
Executive Summary

Many correlations exist between living conditions (environmental, social, economic, and psychological conditions) and health; the problem we face is that the interpretations of nearly all of these correlations are controversially discussed. Scientific papers such as the “WHO European review of the social determinants of health and the health divide” have tried to give us a framework for dealing with the multitude of challenges we face when trying to interpret evidence, in order to develop appropriate recommendations for health interventions. This leads into two key questions of central importance to this workshop and Public health in general: what correlations can actually be proven to be founded on causal relationships, and what are the causal mechanisms behind the correlations themselves?

We still face many challenges when trying to establish causality in public health issues. One of the main challenges is the complex chain of causes we have to engage with when trying to establish a causal relationship between different health indicators. Health and health behaviours are not distributed randomly, and there will always be different individual trajectories over the life course. This produces selectivity effects in addition to the hypothesised causal relationship which are sometimes hard to distinguish. We also run into the issue that behaviours influenced by individual socioeconomic statuses are often affected by more distant structural factors. Even though acting on distant determinants is of crucial importance, it remains difficult because there is little evidence on the effectiveness of specific policies. There may also be reverse causal pathways creating the typical “hen and egg problem” where we have difficulties separating and quantifying the two directions of causality. Without clear and proven causal links, however, the research of Public health experts/practitioners may lead to wrong conclusions and will not have the impact needed in order to convince political decision makers of the evidence and goals.

In order to give science-based policy recommendations, best available evidence needs to be critically appraised with a focus on the development of the concept of causality. The central importance of evidence for our discipline needs to be underlined again; when we are in the process of judging evidence we also need to consider how precise our data collection has been and how robust our initial theories and assumptions are. In all our scientific research
we should be careful to keep in mind whether we can show a causal effect, since only then we can make binding recommendations, which could convince actors within the political decision-making process, through the aid of stringent and robust scientific evidence.

Great progress has been made in the development of the statistical theory of identification of causal pathways and its applications. Key concept is the “instrument”, which drives the pathway to be identified together with the root cause without being driven by the effect. In many circumstances the ideal instrument is random assignment, but oftentimes this is impossible (especially when analysing data after an unexpected event) or unethical (especially when the non-treated in an experiment face serious harm). Then other instrumental variables have been found. The statistical theory also tells us quite clearly that in the absence of such instruments no causal interpretation is possible, and researchers in public health need to acknowledge this.

Finding the casual links between living conditions and health status throughout the life course is especially important in an age of globalisation, where we are facing significant changes in the nature of the work and labour market, especially the increase of work pressure and competition, including “high performance work organisation”. The high demand for flexibility and mobility, the hurried adaptation of workers to new tasks and technologies, coupled with the fragmentation of occupational careers with de-standardised/ataypical work, has led to growing job instability and insecurity, which has led to adverse health effects that will only increase throughout the next decades. On top of this, there is an increasing segmentation of the labour market in combination with rising social inequalities in the quality of work and employment, which naturally also has an effect upon health and health inequalities. We need to keep in mind that health and social inequalities exist in a place or “context,” which possibly has some additional explanatory potential. The concept of place/context comprises a social composition in terms of employment quota, mean income etc., and specific features of the built (air quality, noise, workability) and social (social capital, crime) environment.

When we look at the history of research behind health adverse psychosocial work environments, the traditional focus had been on issues coming out of occupational medicine, specifically chemical and physical hazards, ergonomic conditions, and shift work. The modern focus nowadays has shifted to occupational health research, analysing stress inducing work organisations, employment conditions, and social relationships. Besides these crucial issues it is equally important to figure out the effects of the social determinants of health throughout the different stages of the life-course, and how they interact with health systems, the wider society, and the inherent macro level con-
text we find ourselves in. In order to achieve this we need better collection and analysis of data, especially in cohort studies and in administrative data and record linkage. When it comes to robustness, we need more scrutiny of identification, less parametric statistics and in general more checks for robustness and scientific validity.

Another central aim for public health should be to work on the cooperation and translation between disciplines. We need to find more ways in which the different disciplines can communicate and interact with each other, not only to learn each other’s “languages”, but also to promote inter-disciplinary cooperation for the good of public health. We must realise that health should be a crucial input in all disciplines (health-in-all approach), and that therefore achieving a translational understanding of the interrelation between socio-economic factors and health is a multi-disciplinary goal.
1 Welcome and introduction

Detlev Ganten

Public health is and remains one of the most important topics, not only for academia, but also for the applied sciences. The Leopoldina together with her partner academies has therefore started a process to see what can be done for public health. This process has already led to many fruitful, and at times controversial, discussions. Since public health covers a variety of topics we therefore decided to organise seven workshops, which attempt to do justice to the topic by covering a breadth of issues and closely related scientific disciplines. The central issues we will be dealing with today are living conditions, and the environmental, social, economic and psychological determinants of health, while also addressing the challenges of establishing causality and making evidence-based public health recommendations.

Peter Goldblatt

When thinking about this workshop the recent WHO region review was used as a central overarching document in order to add a guiding structure to the work and resulting discussions we will be having today. In the WHO report the review recommendations are grouped under four broad themes:

- Macrolevel context
- Wider society
- Life course stages
- Systems

Of course, the macro-level context influences the other themes, whereas the other themes influence each other (Review of social determinants and the health divide in the WHO European Region: final report).

This scheme also underlines the importance of the effects of the social determinants of health throughout the different stages of the life-course, and how these interacts with health systems, the wider society, and the inherent macro level context we find ourselves in. This workshop is structured around the importance of analysing health throughout the whole life-course, which is
why the first part of the session will focus on the significance of early childhood development upon health. The next session will then deal with the key factors that affect health and its development on the basis of occupational health studies, which will then lead into the third session, which will attempt to take a step back and deal with the question whether the evidence we are dealing with stems out of causality or association. Of course there are other important factors aside from what happens to individuals in the life-course, which is why the fourth session will look at the factors in wider society that influence health, specifically in the development of health inequalities. We need to keep the fact in mind that we are all situated within the wider society in a system that perpetuates inequalities, which arise out of overarching macro-level factors and pathways. Of course it will be impossible to examine every single issue in total detail within the space of a day in a single workshop, but we hope that through this structure we have picked the key issues and examples that will convey the importance of this subject matter to public health.

Axel Bösch-Supan

The active cooperation of disciplines that is taking place under the auspices of the academies plays a critical role in thinking about public health. Public health in Germany still faces many hurdles; we still only have one chair of public health in Germany, while also facing the issue that many theories and terms contained within the concept of public health, when translated to German, still bring up connotations with the time of the NS-regime because of Germany’s historical context. Even though this might seem foreign to non-Germans, we must realise that this still influences the decision-making process, especially when we talk about large-scale interventions, environmental effects, and how to affect the health of entire populations.

Primary goal of public health analysis is to ascertain that the intended effects of a treatment are indeed present, and that there are no harmful unintended side effects. The unintended side-effects of a measure provide a basis for learning for the future. The goal should hence be to isolate cause and effects to leverage this into policy action.

Another central aim of this workshop is to work on the cooperation and translation between disciplines; even though Johannes Siegrist and I speak the same language, after working together for more than ten years we realise more and more that when dealing with specific theories, concepts and disciplines that we speak a different professional language. One of the goals should also be dedicated towards bridging the understanding between each
other when it comes to how we label specific theories and concepts in order to improve cooperation and comprehension.

We are also lacking an overarching common framework of causality accepted by all the experts of the different disciplines represented in the workshop. Hence, I ambitiously would like to focus and further develop the concept of causality. In all our discussions we should be careful to keep in mind whether we can show a causal effect, since only then can we make binding recommendations which could convince actors within the political decision-making process.
2 Influence of economic conditions and other factors in early life

Arie Kapteyn

The following examples provides a context for (a new way of) thinking about public health. Central to this way of thinking about public health is to understand the mechanisms and the causes behind them. Traditionally, in the intellectual and scientific process within the public health community, we think about an issue, then make a theoretical model and proceed to observe and see if it fits. This approach has served us well, but of course this method also depends on the quality and validity of the initial assumptions. Subsequently, two central questions arise from this theorizing: how do we learn, and most importantly, how do we learn more?

When trying to tackle the broad and complex question of “how do we learn” within our disciplines there are two strands of approaches. On the one hand we use theory, structural models and estimation techniques. A second strands is natural and/or field experiments, in which we learn about policy changes that have not been anticipated or learn by observing and analysing policy differences and outcomes at population level between countries and/or jurisdictions.

Oeppen and Vaupel (2002) have studied the development of female life expectancy in record-holding countries from 1840 to the present. They conclude that female life expectancy has risen for 160 years at a steady pace of almost 3 months per year. Whereas in 1840, Swedish women had the longest life expectation with 45 years, nowadays Japanese women enjoy, with almost 85 years, the longest expectation of life. Results suggest that there is no plateau to life expectancy, while also raising several interesting and revealing questions.

Looking at how life expectancy has changed throughout the last centuries and decades provides us with several investigative questions, which could be further analysed. For example, what are the factors that have changed over the centuries to allow for such a rapid and consistent increase of life expectancy, ergo, what has made this development possible? The aforementioned
analysis strongly suggests that our developments in health and other areas have led to higher life expectancies with seemingly no end in sight.

But it is not just life expectancy that is changing over the ages. People’s approach to and dealing with age is also shifting. Not only that people are living longer, there has also been a decrease in the mortality rate. Increasingly, people identify previous age cut-off points as occurring later in life, meaning that people feel younger than their actual age. For example, men age 74 in 2007 had about the same mortality as men age 65 in the 1960s. There has been a ten-year life gain in a 50 year period (Banks, Blundell, Bozio, & Emmerson, 2011).

So we live a lot longer than we used to but not to the same degree in every country. If you consider the interrelations between health care expenditure and life expectancy, which of course is only a correlation but still important nonetheless, we can see, that there is a large variation between countries. For example, the United States have the largest expenditure on health but this does not lead to the highest life expectancy. This tells us that there must be more factors, which affect life expectancy than just health care, and that variations in health care expenditures clearly do not fully explain the variations in life expectancy. Therefore, there is scope for further learning from country differences about what works or not. Some examples will be provided in the following.

Let us begin with the simple question: “Is retirement good for your health?” This question was explored by Coe and Zamarro (2011). Most economists would say that people have to work longer in order to maintain their standard of living in later age. This raises the difficult question whether people are able to accomplish that. In order to figure this out we could take the naive approach of comparing health before and after retirement. This is problematic for obvious reasons. In order to be able to fully explore these issues we would require exogenous shocks causing changes in retirement, for example, retirement windows. The issue of generalisability remains, though. When comparing countries, we also need to keep in mind how we would use and deal with international variation in these cases.

Even though retirement is endogenous, some people who would, for example, still work in country 1 at age 60, have retired in country 2. These people need to be compared. The findings and the conclusions we could draw from this are controversial; they find that retirement seems to be good for overall health, certainly at age 65, but the effects this has on mortality are not studied. Of course there are also other correlated issues that could have an effect upon this curve; for example, in one country pension benefits could make it more beneficent to retire early.
Hernaes et al. (2013) study whether retirement age has an impact on mortality. This was done with Norwegian data after early retirement was introduced in some industries, which also allowed for a comparison between early and late retirement. No effect of retirement on mortality was found in a difference in difference approach.

For all of these examples, we need to keep in mind that there are also scientific papers that show exactly the opposite. This is one of the main reasons why this remains an interesting topic, since it deals with controversial issues where there is no broad consensus within the scientific community.

Another interesting investigative question that we can add to the discussion is the issue whether retirement is also good for cognitive functioning. This was analysed by Rohwedder and Willis (2010), who related percentage decline of mean performance of cognitive tests of men aged 60-64 relative to men aged 50-54 to percentage decline of labour force participation. The relative cognitive performance was based on a ten word recall test. The authors come to the conclusion that “...the relative difference in cognitive performance is about twice as great in countries with early ages of retirement like France, Austria, Belgium, and the Netherlands as it is in countries with later retirement such as the United States, Denmark, Sweden, and Switzerland.” Despite of differences in the age of retirement, on average, retirement reduces cognitive ability relative to remaining in labour. A study from the United States by Bonsang, Adam, and Perelman (2012) comes to similar conclusions in showing that retirement has a significant negative effect on cognitive functioning. Using data from the Health and Retirement Study, they found accelerated decreases in word recall at ages 62 and 65, which were the ages at which most people retire. There is also evidence from Mexico, where in a randomised controlled trial in Yucatan, where some elderly got social security benefits and others did not, it was found that receiving social security benefits significantly helped to improve memory.

For some more general work on the link between socioeconomic status and health, it is also interesting to look at James P. Smith’s paper from 2005 (Smith, 2005) in which he studied the impact of health on socioeconomic status and vice versa. He found that severe health shocks (cancer, heart disease and diseases of the lung) had major effects on work, income, wealth, but not vice versa. Household income wealth did not seem to have an effect on individual health outcomes. It was found that the economic situation during childhood has an effect on health outcomes later in life. The link between health and education is also studied by Cutler and Lleras-Muney (2006), whereas Silles (2009) looks for causality between health and education.
In conclusion, we must realise that health is a crucial input in production and well-being, and that understanding the interrelation between socio-economic factors and health can make us more productive, can save the government money, and can make us happier.

**Martin Boback “Challenges of establishing causality”**

There are many challenges when we try to establish causality in public health issues. We have done quite well with individual level risk factors. One of the main challenges is, however, the complex chain of causes we have to engage with, when trying to establish a causal relationship between different health indicators. We also need to keep in mind that health and health behaviours are not distributed randomly, and that there will always be different individual trajectories over the life course. It is a challenge to find agreement with policy makers and public health scientists on the fact that the causal chain is long and complex. There is also the issue that behaviours influenced by individual socioeconomic statuses are affected by more distant structural factors, and that even though acting on distant determinants is of crucial importance, it remains difficult because there is little evidence on the effectiveness of specific policies.

When looking at the chain of causes that can lead to poor health, several circumstances at different levels have a direct influence. Starting with the international level (e.g., wars and conflicts) and going down to the national level (e.g., economy, water supply, social benefits), also caused at the group level (e.g., low education, poverty, unemployment) can have a tremendous effect on health. Finally different factors at the personal level (e.g. smoking, drinking, unhealthy diet) can also lead to poor health.

When establishing causality, a lot depends on the assumptions made. Results of a model are not independent of model specification.

When trying to track the causes of the life course, we also need to keep early childhood socio-economic position and education and the crucial effects it has on cognitive development in mind, especially the effects it has upon cognitive reserve and health & lifestyle later in life.

We should also not forget to consider the Bradford-Hill criteria from 1965 (Hill, 1965). This has been a “bible” for epidemiologists, but many of his criteria could be seen as indirect and very weak, with especially many doubts about its consistency. Some critique on the criteria has been raised, e.g. by Ward (2009), on the issue of causality.
Bradford-Hill Criteria

- Strength of association (e.g. RR)
- Consistency (btw studies / populations)
- Specificity (1 cause - 1 effect)
- Temporality
- Biological gradient
- Biological plausibility (biological mechanisms)
- Coherence (with current knowledge)
- Experiment (Randomised control trials)
- Analogy

Hill (1965)

Discussion

- We should expand more on temporality and get the causes and responses into the right timely order.

- The Bradford-Hill criteria should not be thought of as a simple checklist that needs to be completed. It is possible to use only parts of it. A shock itself may not help to identify causality.

- Very often the underlying mechanisms of causality are unknown. Establishing a causal relationship is difficult. Even in the example of cardiovascular diseases, it is not possible to establish a clear causality as long as we look at biological outcomes.

- Establishing causality is a problem we often face when looking at complex health problems and the mechanisms behind them, especially in complicated realms such as epidemiological genetics. But, for public policy measures, we should always attempt to investigate the causal relationships as much as possible. In public health, this is a problematic task.

- There is also the opposing view, which argues that it is always needed to understand mechanisms before acting in public health interventions. If we think about the BSE crisis, there was no understanding of the mechanism, even though it was relatively clear that the epizootic was caused by feeding cattle the remains of other cattle in the form of meat and bone meal (MBM). There may be certain situations in which not every detail is understood but enough is understood to take action.
• We should differentiate between short-term emergency situations, which require immediate actions and long-term issues. To use a metaphor, if a house is burning there should really be no need to know about mechanisms in order to deal with urgent problems that we are facing. Of course when it comes to long-term public health issues, it is of critical necessity to understand mechanisms. To continue with the previous metaphor, when it come to the long-term we should be changing the wiring in houses to prevent future fires from breaking out, but we still need to realise that when it comes to immediate issues we just need to throw water on the fire.

• We need to keep the issue of complexity at the forefront. The more we deal with causes of causes the longer and more complicated the chain of causes becomes, especially if we add wider determinants.

• Also, sometimes the causes of causes cannot be agreed upon. There is still controversy around the question what the exact interaction in the causal relationship between health and social status consists of.

• We often have the issue that different people show different causality, and also that often the models we create to test our own theories often are only a reflection of our own assumptions.

• We need to realise that we don’t have to understand everything, if you can deduce that certain circumstances are acting as a mechanism we should be able to realise that something needs to be done. It is not necessary to know everything about the mechanism in order to be able to know how to act on it.
3 Psychosocial factors in the work environment influencing health

Johannes Siegrist

Causal inference based on results from epidemiological studies remains a methodological challenge, but Hill’s criteria from 1965 of evaluating evidence towards causality of associations remain useful to this end. 25 years of intense international research on associations of an adverse psychosocial work environment with health offer substantial empirical material for this test. This research remains important because adverse working conditions are an important social determinant of health, and therefore potential policy implications of related results may be substantial.

When we look at the history of research behind health adverse psychosocial work environments, the traditional focus had been on issues coming out of occupational medicine, specifically chemical and physical hazards, ergonomic conditions, and shift work. The modern focus nowadays has shifted to occupational health research, analysing stress inducing work organisations, employment conditions, and social relationships.

This is a result of significant changes in the nature of the work and labour market, especially the increase of work pressure and competition, including “high performance work organisation,” a direct impact caused by economic globalisation. The high demand for flexibility and mobility, and the hurried adaptation of workers to new tasks and technologies, coupled with the fragmentation of occupational careers with de-standardised/atypical work, has led to growing job instability and insecurity. Adverse health effects can also be seen because of the increase of service and IT-proessions/occupations, which exhibit a high psychomental/emotional workload. On top of this, there is an increasing segmentation of the labour market in combination with rising social inequalities in the quality of work and employment.

Three theoretical models have been created in order to help us understand health-adverse psychosocial work environments:

(DCM) pays attention to the human factor in the work environment and conceptualises the work environment as purely one of human construction, capable of change to an optimal active learning environment” (Jonge, Dollard, Dormann, Blanc, & Houtman, 2000).

2. The effort-reward imbalance model – focuses on the features of work contracts (Siegrist, 1996): The model of effort-reward imbalance (ERI) claims that failed reciprocity in terms of high efforts spent and low rewards received in turn is likely to elicit recurrent negative emotions and sustained stress responses in exposed people. Conversely, positive emotions evoked by appropriate social rewards promote well-being, health and survival.¹

3. The organisational justice model – focuses on the features of organisational procedures (Elovainio, Kivimäki, & Vahtera, 2002; Greenberg, 1990): The model considers how an employee judges the behaviour of the organisation and the employee's resulting attitude and behaviour.

The demand control model and the effort-reward imbalance model are measured by a standardised self-assessed questionnaire which can be applied to a variety of different occupational groups:

- Job Content Questionnaire (JCQ) (R. A. Karasek)²
- Effort-Reward Imbalance Questionnaire (ERI) (J. Siegrist)³

Both of these questionnaires fulfil criteria of psychometric quality, such as factorial structure of scales, reliability, and discriminant and predictive validity. They are available in a number of languages and have been used in comparative international studies.

When we look at the models using SHARE data we can see that there is an element of social gradient of work stress in the European workforce from age 50-65 (Morten Wahrendorf, Dragano, & Siegrist, 2013). The higher the skill level the lower the work stress level and also the effort-reward imbalance.

The empirical basis for the test of Hill’s criteria for evaluating causal associations rest on about 20 prospective epidemiological studies with either the demand-control model or the effort-reward-imbalance model (or both) as the exposure measure (> 100,000 employed men and woman). The major health outcomes that were observed were depression, cardiovascular disease,

changes in functioning of sexual and reproductive health (SRH), and cardiovascular risk factors.

When looking at the temporality and strength of association of odds ratios (OR) in cohort studies in regard to risk of depression, the different theoretical models came to the following results:

- **Demand-control model:**
  - 12 of 14 studies: OR varying from 1.2 to 3.4 (full model or components)

- **Effort-reward imbalance model:**
  - 10 studies: OR varying from 1.5 to 4.6 (full model or components)

- **Organisational justice model**
  - 11 studies: OR varying from 1.2 to 2.4 (single components)

In a study by Bosma, Peter, Siegrist, & Marmot (1998) it was shown that effort-reward imbalance is associated with elevated risks of subsequent coronary heart disease. In detail they revealed that for men and women who have high efforts as well as low rewards, the risk for coronary heart disease is about 2.5 times higher as for people with low efforts and high rewards.

In order to prove the consistency of associations, a meta-analysis of cohort studies on job strain were made by Kivimäki et al. (2006). An age- and gender adjusted summary of 10 job-strain studies suggested that the risk ratio for incident coronary heart diseases is slightly increased (relative risk 1.16) for a combination of high work demands and low job control (Kivimäki et al. 2006).

When looking through the different factors in the consistency of associations we see the following results:

- **Gender:** Associations stronger in men than in women
- **Age:** Associations stronger in middle-aged than younger adults (old age: healthy worker effect?)
- **Socioeconomic position (SEP):** Associations stronger in lower SEP than in higher SEP (exception: person-based profession)
- **Culture:** similar strength of associations in western and eastern cultures (more confirmation needed)

There also seems to be a clear dose-response association when we examine some data of the study of (Kivimäki et al., 2006). He found “excess risks for high job strain, low job control, high effort-reward imbalance, and low reward, but not for high demands or high efforts”. Another dose-response association was shown in the study of Wang, Schmitz, Dewa, & Stansfeld (2009). They revealed that people with a persistently high job strain ratio as well as people
with a change from low to high job strain ratio have a 1.5 times higher risk to develop a major depressive episode.

Chandola explained in 2006 the number of job stress assessments over time increase the risk of getting a metabolic syndrome (a cluster of risk factors that increases the risk of heart disease and type 2 diabetes).

Regarding the specificity of association, we can also find falsified association in an implausible case regarding job strain and cancer risk. In a meta-analysis of 5,700 incident cancer events in 116,000 European men and women Heikkila et al. (2013) did not find any clear evidence for an association between job strain and the risk of cancer. Hence, work stress is no relevant risk factor for developing cancer.

In conclusion, many policy-relevant public health topics are imbedded in a complex “web of causation”. To a certain extent, causal inference along Hill’s criteria is nevertheless justified as exemplified by the research on health effects attributed to an adverse psychosocial work environment. We require cumulative research paradigms and sophisticated application of advanced statistical models in order to improve research evidence. Most importantly for us as public health experts, is the necessity to realise that in order to give science-based policy recommendations, best available evidence needs to be critically appraised.

Andrew Jones “Econometrics, health economics and epidemiology – exploring the interface”

There are several recent themes in health econometrics:

- The evaluation problem & identification strategies
- Data and measurement issues
  - Administrative data & sample surveys
  - Health outcomes: Self-reports, anthropometric, biomarkers (ELSA, UKHLS)
  - Modelling costs and expenditure: Two-part models (2PM), generalised linear models (GLM)
- Recent methods for panel data and other complex designs
  - Nonlinear models and unobserved heterogeneity (unmeasured confounders)
  - Conditional estimators, maximum simulated likelihood, Bayesian MCMC, finite mixtures and copulas.

Then what are the recent methods for estimating treatment effects?
Living conditions and causality

- Randomised social experiments
- “Natural” experiments – actual events/policy reforms with controls
- Ex post impact evaluation:
  - “Selection on observables” – adjusting for observed confounders: regression models, matching, inverse probability weights.
  - “Selection on unobservables” – instrumental variables (IV), control functions, panel models (FE/DiD), multiple equations (FIML)
- Ex ante evaluation/structural simulation approach

When looking at natural experiments and the issue of “randomisation by events” we have a long list of examples that we could analyse:

- 1918 influenza pandemic (Almond, 2006)
- Passengers in traffic accidents (Doyle, 2005; Levitt & Porter, 2001)
- Pittsburgh bus strike (Evans & Lien, 2005)
- Reunification of Germany (Frijters, Haisken-DeNew, & Shields, 2005)
- Russian pension crisis (Jensen & Richter, 2004)
- Pension reform after apartheid (Dufl0 2000)
- Lottery winnings (Gardner & Oswald, 2007; Lindahl, 2005)
- Historical business cycle (Van den Berg, Lindeboom, & Portrait, 2006)
- Educational reforms (Arendt, 2005; Lleras-Muney, 2005)
- Hookworm eradication (Bleakley, 2007)
- Romanian ban on abortion and family planning (Pop-Eleches, 2006)

Also of importance are the instruments and natural controls that we use, for example:

- Within individuals (fixed-effects model),
- Within communities (fixed-effects model /MLM),
- Siblings (Holmlund, 2005),
- Twins (Black, Devereux, & Salvanes, 2007), or
- Genetic markers (Norton & Han, 2008).

There are also the issues of anti-tests and sensitivity/robustness that we need to keep in mind. A good example of this is the evaluation by Galiani, Ger-tler, and Schargrodsky (2005) of the impact of the privatisation of local water services on child mortality in Argentina, where they adopted two strategies for assessing the reliability of their difference-in-differences approach that can both be interpreted as anti- or placebo tests. In a placebo regression the model of interest is estimated using only data from the pre-treatment period, but including an indicator of those cases that will go on to be treated. This tests the “parallel trends” assumption required for difference-in-difference...
analysis. As well as measuring deaths from infectious and parasitic diseases they include measures of deaths from causes unrelated to water quality. The fact that they detect a reduction for the former but not for the latter creates confidence in their difference-in-differences identification strategy.

As a second part of this presentation we will be looking at the contractual conditions, working conditions, and health and wellbeing in the British Household Panel Survey. The general aim of this study was to analyse the effects of contractual and working conditions on self-assessed health (SAH) and psychological wellbeing (GHQ) using 12 waves (1991/92 – 2002/2003) of the British Household Panel Survey (BHPS). The motivation behind this study is the vast amount of changes that have been going on in western countries in the past decades:

1. Contractual conditions; non-standard work arrangements (temporary work, part-time contract, etc.) have become much more common.
2. Working conditions; decline of manufacturing jobs, growth of service oriented/computer base jobs.

It is therefore relevant to evaluate whether and how contractual and working conditions affect health and psychological well-being. The results of the BHPS paper can be summarised as following:

- Both contractual and working conditions have some influence on health and psychological well-being. Differences exist between women and men.
- Being unsatisfied with the number of hours worked has a negative influence on the health of individuals who have a part-time job.
- Having a high level of education positively influences both health and psychological well-being of individuals with temporary job arrangements.
- Family structure influences health and psychological well-being of workers with atypical contractual arrangements.

To sum up, in general, we need better collection and analysis of data, especially in cohort studies and in administrative data and record linkage. We also need an increase in experimental studies with simpler econometrics. When it comes to robustness, we need more scrutiny of identification, less parametric statistics and in general more checks for robustness and scientific validity.

Discussion

- There are two main methods of data linkage used in Europe. Probabilistic linkage is mainly used in the UK and Scandinavia, where we have identifi-
cation numbers in the data sets, i.e. record linkage. This is because in the United Kingdom we have a universal National Health Service (NHS) system with a unique NHS number for everyone within the British health system. If we have these numbers available in two different data sets, it enables us to do record linkage, even though one must be aware that sometimes the information is badly recorded. It is also possible to do occupational health surveys with these numbers, and then link health outcomes to these numbers.

- One can observe especially interesting findings if one looks at the link between unemployment and health. In the survey data that we have seen unemployment was always associated with worse health in advanced age.

- Another useful application for administrative data is in helping us to understand non-responses in survey data. With administrative data there is always an element of selectivity involved that we need to keep in mind.

- After the presentations we have heard so far, it should be clear that we should extend the causal pathways that we are investigating into early life. In general, we should be investing more into life course research, because the evidence behind it seems to be quite robust.

- Is robustness really a criterion? If we continue to always make the same mistake in our procedures, we would still get robust results. There is really no proof that you’ve found any worthwhile evidence if there is a systematic mistake in the process.

- If there is an identification problem, getting more data will not solve it, because you will not escape the problem of faulty premises. We therefore must ask ourselves whether the level of robustness is related to the nature of the health outcomes. Personally for many of the issues of social exposures, I would prefer to see hard outcome effects.

- The general problem seems to be that many researchers do not always base their conclusions on the intrinsic value of the data that they are working with.

- Any well-written scientific paper on these topics should be talking about the strength and weaknesses of data. The issue is that papers are often written by people who do not understand the modus operandi of data collection methods.
• We should also stress that survey data tends to be prone to errors, for example when we look at questionnaires that deal with the gradient of blood pressure, since the way you posit these questions also has an impact on the outcome. Measurement therefore remains and will remain a huge problem in the social sciences.

• Sometimes we may be forced to act on the best available evidence in situations where we cannot wait on the best evidence. Of course we should be strict in our scientific research, but we should also be careful to not be too strict with our criteria in everyday settings. There should, however, be a limit to the pragmatism that is often expressed, as there are many examples in public health, where interventions were either done too early, or based on faulty research/evidence.

• Doing nothing is an action in itself when we talk about public health. There has to be a balance between decisive action and careful interventions.
4 Evidence for causal pathways from socio-economic position to health

Joachim Winter

In health economics there is the “chicken and egg” problem around the socio-economic gradient of health. We know that socio-economic status (SES) and health are positively correlated, but we are not sure why this is so and in which direction causality goes: Does SES cause health (Hypothesis A)? Does health influence SES (Hypothesis B)? Or are health and SES dependent on some other common factors (Hypothesis C)?

Causal inference faces two methodological challenges: the problem of simultaneity and reverse causality (Hypothesis A vs. Hypothesis B). The observed correlation between SES and health may likely also be caused by omitted variables and/or individual heterogeneity (Hypothesis A/B vs. Hypothesis C). The arguments in this presentation are based on Adams et al. (2003), Stowasser et al. (2012) and Stowasser et al. (2013), all of which make use of the Granger-causality-test (Granger, 1969).

Adams et al. (2003) use entire observed (partly endogenous) variation, and a one-sided test for the absence of causal links (either direction). Within this paper Granger-causality from SES was rejected for most health conditions. Stowasser et al. (2012) revisited the paper by Adams et al. with more comprehensive data including the data gathered from the United States Health and Retirement Study (HRS). They came to the conclusion that Granger-causality can no longer be rejected for most health conditions, the exception being acute conditions. The evidence found by Stowasser et al. suggests that the original conclusion by Adams et al. were partly driven by low test power in small samples.

The starting point for the third paper (Stowasser et al., 2013) was the Granger-causality model as used by Adams et al. We added retrospective life history data from the RAND Health and Retirement Study (HRS) to the analysis. Exploiting this data gave us three distinct advantages:

1. It gives us an improved model of health dynamics (so far: 1st-order Markov)
2. It provides us with improved control for individual heterogeneity and initial conditions.
3. It offers us a substantive research agenda with the central question of when the gradient is established, in childhood or adulthood.

Retrospective data enables us to look back into the „black box“ of early life by better control of initial conditions and individual heterogeneity (Stowasser et al., 2013).

- **First advantage: Improved model of health dynamics**: Adams et al. (2003) modelled health as 1\textsuperscript{st}-order Markov model, which delivered an unrealistic description of stock characteristics of latent health capital as envisioned by Grossman (1972). In order to accommodate long-memory characteristics of latent health we modified the model in two specific ways: first, we increased the length of the adult health histories that we controlled for, which made a 3\textsuperscript{rd}-order Markov approach feasible without losing too much data. The second modification was to add a control for health status as a child, which provided us with a much longer health history and quasi-initial conditions. We therefore wanted to study whether the gradient survives with a richer model of health dynamics in place.

- **Second advantage: Improved control for individual heterogeneity**: The downside of a Granger causality approach is that there is no clear discrimination between “true” causality and common effects. Individual heterogeneity (genetics, family background, early-life experiences) influences both health and SES, but it is unobserved. We therefore added a proxy control in order to allow for some individual heterogeneity. We specifically added childhood SES and other retrospective data that would help us capture characteristics of family and home environments. This will not completely solve the methodological problems faced with the Granger causality approach but it does help to alleviate most issues. We therefore need to examine whether the gradient survives with these additional controls in place.

- **Third advantage: Pathways between SES and health - When is the gradient established?** The first two improvements we made is to study if the gradient is causal in nature or not, but from a substantive perspective. We also wanted to understand when and how causal links are established. Since we control for both historic and contemporary variables, it allows us to consider several interesting question.
Living conditions and causality

- Is there a link between SES and health that is predetermined during childhood?
- Are contemporary factors still important?
- Are there gender differences in the inter-temporal transmission of SES and health?

This raises important policy implications, specifically the issue of whether resources should be invested in order to provide educational and financial support to young families?

The data we used to explore this question was a combination of RAND HRS panel data and HRS early life data. The RAND HRS panel data were taken from waves 2-9 (1993-2008) with the following SES variables: Wealth, income, education, dwelling conditions, and neighbourhood conditions. For better interpretability we took 20 health conditions and then combined them into 6 disease clusters:

1. Acute: cancer, heart disease, stroke
2. Chronic: lung disease, diabetes, hypertension, arthritis
3. Functional: incontinence, severe falls, hip fractures, ADL/IADL impairments, obesity
4. Mental: cognitive impairment, psychiatric illness, depression
5. Self-rated health
6. Mortality

The HRS early-life data included information on childhood health (3 disease clusters) and the family background (9 family SES markers, 5 family behaviour makers, parental mortality, proxy for in utero and early life environment).

As for improving the model of health dynamics one can conclude that richer models of health dynamics help us to describe the evolution of health better than short-memory models, the exception being acute health conditions that are sufficiently described by 1st-order Markov processes. Our results for SES Granger causality tests do not substantially change; there is no Granger causality for acute conditions, while mental conditions, SRH, and mortality, all exhibit Granger causality. We received mixed results when testing for chronic and functional conditions, but these results appear to be driven by a reduction in sample sizes.

As for improved control for individual heterogeneity and common effects, results are similar than for the richer health dynamics models. There is no Granger causality for acute conditions, while chronic and mental conditions, SRH, and mortality, all exhibit Granger causality. There are mixed results for functional conditions, but just as previously, these results appear to be driven...
by a reduction in sample sizes. The results when controlling for all retrospective variables (childhood health and family effects, smallest sample) suggest that Granger causality is most robust for mental health and SRH, even though this arguably warrants a causal interpretation of the gradient for these health dimensions.

The results we encountered come from parameter estimates of the underlying prediction models, which we interpreted as follows. Regarding acute health conditions, we saw no effect of childhood/adult SES, which is in line with Granger causality tests, which shows us that SES does not matter for acute health shocks. Concerning chronic health conditions we found no evidence that a link between SES and health is established during childhood, it seems to be adult SES and not childhood SES that really matters. However, childhood health is a good predictor for adult health outcomes.

When looking at functional mental health conditions we see that there are strong gender differences. For women, the gradient appears to be established during childhood, ergo, only childhood SES matters. For men on the other hand, the gradient appears to be established later in life during secondary education and occupational period, meaning that only years of education and current status of wealth/income matter. Irrespective of gender, childhood health has predictive power for health shocks later in life. This implies that for future research we should be looking at SES outcomes over the life cycle, specifically in labour market participation.

In summary, the availability of retrospective early-life data brings us closer to being able to give causal interpretations of results from Granger causality frameworks. We can also determine with some confidence that adult SES is unlikely causal for acute health conditions, while mental health, SRH, and mortality, are likely causal. Regrettably, the evidence for chronic and functional health conditions is mixed, which does not allow us to draw any clear conclusions there. We also find that longer health histories do a better job in explaining health innovations than 1st-order Markov models, since even childhood health has a strong predictive power for health outcomes among retirees. Especially for women, part of the SES gradient in health is established during childhood.

Therefore, we require more support by health practitioners and the health community to convince political decision makers to push through public policies that are specifically targeted at young families since this should have the largest long-term impact.
Living conditions and causality

Peter Goldblatt - Discussion

First off, we need to distinguish between the issue of whether health causes or leads to SES, or whether SES simply influences health? Those issues and the related methodological problems raised can be seen as quite separate. Maybe we could look at the previously used concept called the “healthy worker effect” (Li & Sung, 1999).

Naturally, health as a variable always has a strong effect in workplace studies; also, there is already a natural selection bias even before the selection process for such studies takes place, because if you are severely unhealthy, you are unlikely to be working in the first place. The impact of health on SES will also differ depending upon the type of health shock/impact. For example, chronic health problems will naturally have a much more long-term effect on SES.

When trying to figure out the direction of causality, analysing time trends is one way we could simplify the long and complex causal chains which we have to deal with. The issue of complexity in the life course and in the causal chain will remain with us because there are such a large number of variables that not only influence and interact with health status, but also influence and interact with each other as well. This leads us back to the “chicken and the egg” problem of the causes of causes mentioned earlier. We therefore need multi-stage models that can apportion how much certain selected variables have an effect upon health; the truth is that with most data sets available nowadays, you can only do this in a very simplistic way. In Markov-chain models we should look at the attributable risk at each stage of the chain. An interesting approach we could maybe learn from is the Barker hypothesis, or thrifty phenotype hypothesis (Barker, 1997)

Discussion

- The point was raised that the bigger the sample size, the less likely Granger causality can be ruled out because tests have more power to reject the null, which is no Granger causality. This is indeed an important feature of "testing for the absence of Granger causality", and McFadden

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4 “The healthy worker effect (HWE) is a phenomenon initially observed in studies of occupational diseases: Workers usually exhibit lower overall death rates than the general population because the severely ill and chronically disabled are ordinarily excluded from employment. Death rates in the general population may be inappropriate for comparison if this effect is not taken into account.”(Porta & International Epidemiological Association, 2008)
was certainly aware of this problem, as is apparent from his original paper. In a way, the new study can be viewed as verifying methodological concerns that have been discussed in this literature for a long time. More generally, such discussions highlight how difficult it is to establish causality, and a fortiori, specific causal channels, between socio-economic status and health using observational data.

- A further complication is that there is no unidirectional causality for most diseases. There are many causal mechanisms which operate in parallel, but in opposite directions. With the current methodology, such multi-causality will be difficult to capture even with larger and more comprehensive data sets.
5 Overview of the influence of contextual characteristics in the development of health inequalities

Oliver Razum

Before giving a basic overview of contextual factors in health inequalities, we should appreciate the “classical” vertical (income, status, education) and horizontal (gender, marital status, nationality) inequalities. We need to keep in mind, however, that health and social inequalities also exist in a place or context, which possibly provides us with additional explanatory potential. The concept of place/context comprises a social composition in terms of employment quota, mean income etc., and specific features of the built (air quality, noise, workability) and social (social capital, crime) environment.

There are three specific public health challenges this presentation would like to deal with:

1. Is there really an East versus West divide in Germany or does differentiation take place at a smaller area level?
2. Do small area “contextual” factors contribute to health inequalities?
3. Can we reduce such social and health inequalities?

When looking at social inequalities in Germany we should realise that the basic constitutional law (Grundgesetz Art. 72, Abs. 2) postulates strategies against inequalities by advising for the “creation of equal living conditions.” Inequalities between the East and the West of Germany were referenced by then Federal President Köhler in 2004, who noted that: There “… were and are (...) major differences in living conditions throughout this republic – between North and South as well as between West and East. Those trying to level them will cement the subvention state and will lay upon the young generation an intolerable burden of debt. (...) What is important is to create room for peoples’ ideas and initiatives”

Interview in the German magazine “FOCUS” with the then federal president Horst Köhler on 13. September 2004: “Aber unabhängig davon gab und gibt es nun einmal überall in der Republik große Unterschiede in den Lebensverhältnissen. Das geht von Nord nach Süd...”
There are in fact obvious differences between the East and West of Germany, such as mean GDP per capita in the East being around 70% of the West. Once we look at the few available summary health measures it becomes clear that the differences are not as large as one would expect. For example, in 2008/2010 average life expectancy at birth for women in the East was 0.2 years below the average of the West (1.2 years for men), in spite of persisting socioeconomic differences between the two parts of Germany.

This shows that the connection between inequalities and health is not a simple association. What we could consider is that the areas which we compared were too large. When we try to analyse the socioeconomic uncoupling of regions by looking at unemployed and social benefit recipients per hundred inhabitants aged 18-65 years from 2003, we notice major differences, with disadvantaged areas clustering in the East (Kröhnert, Medicus, & Klingholz, 2006). However, mapping can be deceptive. The map shows an unequal distribution of social benefits recipients in Germany. Initially it may seem that most recipients live in East Germany. But when we look at the regions in more detail and take population numbers into account, a different picture emerges. Mecklenburg-Vorpommern in the East has 1.4 million people affected, while the core area of the Ruhr Valley, the former industrial heartland of the West, has 5 million people affected, even though the latter information only shows in tiny dots on the map.

We also need to keep in mind that a major population movement has been going on in Germany, which is affecting living conditions and the economy. Initially, this again seems to be an East-West phenomenon (e.g. between 1991-2008 the net migration from the East to the West was 1.74 million people (Kubis & Schneider, 2008)). However, when looking at the population decline in the Ruhr area it becomes evident that the demographic transition takes place in other regions of Germany as well. There again is not a simple east/west divide (Kröhnert, Medicus, & Klingholz, 2006).

We therefore have a situation in Germany where a redistribution of a shrinking population is taking place, both in the east and west, and in a selective manner (e.g., more young and active women migrate than men). A shrinking population of course has a direct effect on local communities. There is a decline in local income tax, and they experience losses in federal financial transfers to cities. Net infrastructure, such as roads, water pipes, sanitation systems, cannot be partitioned, meaning that local communities are forced to

pay upkeep for basic services that are not being fully used. The shrinking of cities is not a simple contractive process. What we are seeing instead is the development of so called “perforated cities.”

So what consequences do these developments have on health? If we look at the life expectancy in the Ruhr area for example, male life expectancy in 2002-2004 is 1.3 years below the average of Nordrhein-Westfalen, while life expectancy for women is 0.9 years below the average. Contrast this with the East/West difference, which is also 1.3 years for males and only 0.2 years for females. When we look at excess mortality in the Ruhr area (Klapper, Bardehle, & Razum, 2007) we can see that the middle age group from 40-49 years is the most affected, with high rates of avoidable deaths from cardiovascular and alcohol related diseases.

Let us go back to the issue of contextual effects on health. We linked socio-economic panel data (SOEP) with small area data (INKAR) for all 439 cities/counties of Germany. What we found was that there are “…Substantial (and) increasing regional disparities … across the whole of Germany…” and “This study finds a significant association between area deprivation and physical health. The association between neighbourhood deprivation and physical health can be partly explained by specific features of the neighbourhood environment.” (Voigtländer, Berger, & Razum, 2009)

So how do we explain these inequalities in the regional distribution of health? Is it merely an aggregation of individual disadvantages (poor and ill people do not manage to move out of a disadvantaged area), or is there a causal effect on health of contextual factors? In order to figure this out we would need an experimental study, randomly assigning people to regions with a different degree of deprivation and then observing their health over time. This, of course, would be impossible for ethical reasons.

However, a scenario similar to the experimental study described above actually occurred, providing a natural experiment. Large numbers of ethnic German re-settlers (“Aussiedler”) migrated from the former USSR to Germany since the 1990s. They were distributed to the federal states and in Nordrhein-Westfalen were randomly distributed to the counties and cities. We analysed their mortality experience over time after aggregating the cities and counties into 6 deprivation clusters (Strohmeier et al. 2007): tertiary sector with few families, poverty poles, heterogeneous cities, heterogeneous counties, family zone and prospering regions.

What we found was that the re-settlers assigned to the “poverty poles” cluster experienced the highest mortality rate. Cox modelling with “poverty poles” as reference was done, showing a significantly lower mortality risk in the less deprived clusters:
• RR = 0.82 in “Prospering regions” cluster
• RR = 0.87 in “Family zone” cluster

Because of the quasi-random assignment of re-settlers to the various clusters, the study provides us with supporting evidence for a causal effect of regional (contextual) factors on health.

In conclusion it is understandable that a focus remains on comparing socioeconomic and health status between the east and west of Germany. But we also need to keep in mind that living conditions are increasingly diverging in the whole of Germany at the local area level. This contributes towards health inequalities, which is apparently happening via contextual effects. The selective migration from the East to the West of Germany is likely to compound the situation via compositional effects. Affected regions could find themselves in a vicious circle of declining population health and socioeconomic disadvantage.

When trying to solve these problems, we cannot simply rely on interventions that aim to change individual behaviour (“Verhaltensprävention”), since these interventions often have the paradoxical effect of actually increasing health inequalities (because uptake is much higher among the healthy, well-educated people). Prevention therefore needs to address contextual factors as well (“Verhältnisprävention” or situational prevention), since it is the prerequisite for creating the “room for peoples’ ideas and initiatives” previously mentioned by Köhler, in order to reach the goal of reducing health inequalities. Specifically what Germany requires are targeted regional investments in the provision of social goods, such as education, subsidised housing, employment, unemployment benefits, and urban planning.

Gabriele Doblhammer “The causality of context factors”

This presentation looks at context factors and natural experiments.

Let us first look at the health of native Germans compared to ethnic German immigrants, the “Aussiedler”, coming from the former Soviet Union, and specifically at the age- and sex-standardised morbidity prevalence for native Germans. Kreft & Doblhammer (2011) looked at the impact of three contextual factors on the health of “Aussiedler”: centrality (proportion of persons driving to the next regional centre in less than half an hour), GDP and the proportion of foreigners in a region. GDP is positively correlated with better health among “Aussiedler” and native Germans independent of their socioeconomic status. Centrality, on the other hand, has detrimental effects on health.

On the topic of exogenous shocks and the effect they have on health we can look at a study on the great Finish famine that lasted from 1866-1868,
which examined the long-term effect of famine early in life on survival at old age. The famine had a negligible effect upon determining survival at old age, which is mostly due to strong selection effects for mortality and fertility. Negative effects of famine on adult health and mortality, could however be observed when differences in the frailty distribution of the cohorts were taken into account (Doblhammer, van den Berg, & Lumey, 2011).

Another experiment was made in order to see whether there is a connection between boom years and GDP and if they made a difference in developing good cognitive functioning later in life (Doblhammer, van den Berg, & Fritze, 2011). Even though the data seems to suggest that being born in a boom year does not affect the life course, the results showed that being born during a boom (recession) period reduces (increases) the risk of low cognitive functioning later in life.

Another example is the effect of the month of birth on adult life expectancy at ages 50+. For Austria and Denmark Doblhammer and Vaupel (2001) showed that being born in May/June has a negative effect on mortality, while the situation is exactly reversed in Australia. We would, however, require much more administrative data in order to conclude what the different risk factors are that are causing these deviations in mortality rates depending upon month of birth.

In conclusion, we should exploit more natural experiments and exogenous shocks for causal explanations of context factors. We should also make use of routine data and link them with epidemiological surveys.

Discussion

- When talking about regional disparities, one should look for the causes of the health differences. The picture of causalities is diverse. With regard to the effect of the month of birth on life expectancy, infectious diseases could be one such reason. There are other studies, however, which support infectious diseases as a predictor of infant mortality; others find that it is the mother’s nutrition. With all studies, it is still an interpretation coming from theory. Causality can only be established with certainty if it can be measured separately.

- There is some evidence that regarding behavioural prevention measures are taken-up by those, who do not really need it (the better-off) and that measures may increase inequality. Health inequality should not be a policy goal. Under these circumstances, we should therefore ask ourselves, whether changing individual behaviour should still be a policy goal? Inter-
ventions should be universal in that they level up the gradient so that everybody improves.

- Analysing East/West differences and developments over time should be extended beyond Germany to Central and Eastern Europe and the countries of the former Soviet Union. These types of analyses are really unique considering the fact that simple first steps after 1989 made a great difference in the development in these countries. It will be interesting to see if life expectancy in the East, for example, will remain flat. It will be worthwhile to study not only differences between East/West but also regions across one country.
6 General discussion

6.1 Key findings

- The key question for this workshop is: what correlations can actually be proven to be founded on causal relationships, and what are the causal mechanisms behind the correlations themselves such that we can build public health recommendations upon them. Examples from the 'WHO European Review of the social determinants of health and the health divide' can be used to illustrate how the challenges of interpreting evidence and making appropriate recommendations should be addressed.

- Great progress has been made in the development of the statistical theory of identification of causal pathways and its applications. The key concept here is that the “instrument”, which drives the pathway, is to be identified together with the root cause without being driven by the effect. In many circumstances the ideal instrument is random assignment, but often times this is impossible (especially when analysing data after an unexpected event) or unethical (especially when the non-treated in an experiment face serious harm). Then other instrumental variables have to be found. The statistical theory also tells us quite clearly that in the absence of such instruments no causal interpretation is possible, and researchers in public health need to acknowledge this.

- We need to keep in mind that health and social inequalities exist in a place or “context,” which possibly has some additional explanatory potential. The concept of place/context comprises a social composition in terms of employment quota, mean income etc., and specific features of the built (air quality, noise, workability) and social (social capital, crime) environment.

- The primary goal of public health analysis is to ascertain that the intended effects of a treatment are indeed present, and that there are no harmful unintended side effects. The unintended side-effects of a measure provide a basis for learning for the future. The goal should hence be to isolate cause and effects to leverage this in to policy action.
• We are lacking an overarching common framework of causality accepted by all the experts of the different disciplines represented in the workshop. Hence, we should ambitiously focus on and further develop the concept of causality. We should always be careful to keep in mind whether we can show a causal effect, since only then we can make binding recommendations, which could convince actors within the political decision-making process.

• We must understand that health is a crucial input in production and well-being, and that understanding the interrelation between socioeconomic factors and health can make us more productive, can save the government money, and make us happier.

• We need to keep the issue of complexity at the forefront. The more we deal with causes of causes the longer and more complicated the chain of causes becomes, especially if we add wider determinants.

• When we look at the history of research behind health adverse psychosocial work environments, the traditional focus had been on issues coming out of occupational medicine, specifically chemical and physical hazards, ergonomic conditions, and shift work. The modern focus nowadays has shifted to occupational health research, analysing stress inducing work organisations, employment conditions, and social relationships.

• Many policy-relevant public health topics are imbedded in a complex “web of causation”. To a certain extent, causal inference along Hill’s criteria is nevertheless justified as exemplified by the research on health effects attributed to an adverse psychosocial work environment. We require cumulative research paradigms and sophisticated application of advanced statistical models in order to improve research evidence. Most importantly for us as public health experts, we need to realise that in order to give science-based policy recommendations best available evidence needs to be critically appraised.

• We need to decide whether robustness is really a criterion? If we continue to always make the same mistake in our procedures, we would still get robust results. There is really no proof that we have found any worthwhile evidence if there is a systematic mistake in the process.

• The availability of retrospective early-life data brings us closer to being able to give causal interpretations of results from Granger causality frameworks. We can also determine with some confidence that adult SES
Living conditions and causality

is unlikely causal for acute health conditions, while mental health, SRH, and mortality, are likely causal. Regrettably, the evidence for chronic and functional health conditions is mixed, which does not allow us to draw any clear conclusions there. We also find that longer health histories do a better job in explaining health innovations than 1st-order Markov models, since even childhood health has a strong predictive power for health outcomes among retirees. Especially for women, part of the SES gradient in health is established during childhood.

• It is understandable that a focus remains on comparing socioeconomic and health status between the East and West of Germany. But we also need to keep in mind that living conditions are increasingly diverging in the whole of Germany at the local area level. This contributes towards health inequalities, which is apparently happening via contextual effects. The selective migration from the East to the West of Germany is likely to compound the situation via compositional effects. Affected regions could find themselves in a vicious circle of declining population health and socioeconomic disadvantage.

• When trying to solve these problems, we cannot simply rely on interventions that at behavioural prevention, since these interventions often have the paradoxical effect of actually increasing health inequalities. Prevention therefore needs to address contextual factors as well as situational prevention, since it is the prerequisite for creating the “room for peoples’ ideas and initiatives” previously mentioned by former president Köhler, in order to reach the goal of reducing health inequalities. Specifically what Germany requires are targeted regional investments in the provision of social goods, such as education, subsidised housing, employment, unemployment benefits, and urban planning.

• We should exploit more natural experiments and exogenous shocks for causal explanations of context factors. We should also make use of routine data and link them with epidemiological surveys.

• The central importance of evidence for our discipline need to be underlined again; when we are in the process of judging evidence, we also need to consider how precise our data collection has been and how robust our initial theories and assumptions are.
6.2 Conclusions

- These three points are directed at the research community, since there seems to be much potential to enrich further research in three specific ways:
  - extend the time periods of research,
  - monitor the impact of variables on the life course in more detail,
  - find innovative ways to enrich the empirical evidence, since in the long run such an approach could affect the quality of output.

- To further promote inter- and multidisciplinary research in public health, we need to find more ways
  - to promote inter- and multidisciplinary cooperation between different research communities, particularly between econometrics and epidemiology and between quantitative sciences and fields focusing on qualitative social research,
  - to institutionalise interdisciplinary exchange, for instance, in (more than one) strong school of public health in Germany,
  - to better communicate and interact across disciplines not only to learn each other’s “languages”,
  - to collect and organise data in a way that they can be used for multi-disciplinary research,
  - to further develop a good statistical methodology for multi/interdisciplinary research.

- Causality and causal mechanisms are very important. This should be kept in mind especially for further research, which should be designed around being able to address and convince policy makers with stringent and robust scientific evidences.

- The central importance of evidence needs to be underlined; when we are in the process of judging evidence we also need to consider how precise our data collection has been and how robust our initial theories and assumptions are.

- If we look at the effect of medical interest groups, and public bodies on the public discourse in the media, they seem to be very good at selling their message, especially to political decision makers. We are lacking in this regard, even though we have lots of evidence that should interest political stakeholders and other people of interest, which is why we should do our best to improve the links between theory, practice and policy.
Even though it has been said that doing nothing is also an action in public health as well, we must underline that in this case and for the future, doing nothing cannot be a valid position for public health and its practitioners to take in the face of rising inequalities, both in health, and socially.
7 References


8 Appendix

8.1 Workshop Programme

Date: June 19, 2013, 11:00 a.m. - 05:00 p.m.
Location: Langenbeck-Virchow House, Room „August Bier“, Luisenstr. 58/59 in 10117 Berlin

11:00-11:30: Welcome and introduction
Detlev Ganten, Coordinator of the Planning Group “Public Health”
Peter Goldblatt, Coordinator of the Workshop
Axel Börsch-Supan, Coordinator of the Workshop

11:30-12:30: First Session
11:30-12:55 Influence of economic conditions and other factors in early life
Arie Kapteyn, Center for Economic and Social Research, University of South California
11:55-12:10 Discussion of the paper by Kapteyn
Martin Boback, Research Department of Epidemiology and Public Health, University College London
12:10-12:30 General discussion

12:30-13:30 Second Session
12:15-12:55 Psychosocial factors in the work environment influencing health
Johannes Siegrist, Institute of Medical Sociology, University Düsseldorf
12:55-13:10 Discussion of the paper by Siegrist
Andrew Jones, Department of Economics and Related Studies, University of York
13:10-13:30 General discussion

14:30-15:30 Third Session
14:30-14:55 Evidence for causal pathways from socio-economic position to health
Living conditions and causality

Joachim Winter, Seminar for Empirical Economics, Ludwig-Maximilian-University Munich

14:55-15:10 Discussion of the paper by Winter
Peter Goldblatt, Institute of Health Equity, University College London

15:10-15:30 General discussion

15:30-16:30 Fourth Session

15:30-15:55 Overview of the influence of contextual characteristics in the development of health inequalities
Oliver Razum, Faculty of Health Sciences, University Bielefeld

15:55-16:10 Discussion of the paper by Razum
Gabriele Doblhammer, Faculty of Economic and Social Sciences, University Rostock

16:10-16:30 General discussion

16.30-17.00 Wrap up and lessons learned
8.2 Workshop Participants

Speakers

- Johannes Siegrist, Institute of Medical Sociology, University Düsseldorf
- Joachim Winter, Seminar for Empirical Economics, Ludwig-Maximilian-University Munich
- Oliver Razum, Faculty of Health Sciences, University Bielefeld
- Arie Kapteyn, Center for Economic and Social Research, University of South California

Discussants

- Martin Boback, Research Department of Epidemiology and Public Health, University College London
- Andrew Jones, Department of Economics and Related Studies, University of York York
- Peter Goldblatt, Institute of Health Equity, University College London
- Gabriele Doblhammer, Faculty of Economic and Social Sciences, University Rostock

Member of the planning group and coordinators of other workshops

- Axel Börsch-Supan, Max Planck Institute for Social Law and Social Policy, Munich
- Peter Goldblatt, Institute of Health Equity, University College London
- Detlev Ganten, Stiftung Charité, Berlin

Leopoldina Secretariat

- Kathrin Happe, Leopoldina, Halle
- Barbara Döhlha, Leopoldina, Halle

Rapporteurs

- Julian Kickbusch, Berlin
- Kathrin Happe, Leopoldina, Halle

Guest

- Tabea Bucher-Koenen, Max Planck Institute for Social Law and Social Policy, Munich