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## **Individual and collective bodies: using measures of variance and association in contextual epidemiology**

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## **ABSTRACT**

### Background

Social epidemiology investigates both individuals and their collectives. While the limits that define the individual bodies are very apparent, the collective body's geographical or cultural limits (e.g., "neighbourhood") are more difficult to discern. Also, epidemiologists normally investigate causation as changes in group means. However, many variables of interest in epidemiology may cause a change in the variance of the distribution of the dependent variable. In spite of that, variance is normally considered a measure of uncertainty or a nuisance rather than a source of substantive information. This reasoning is also true in many multilevel investigations, whereas understanding the distribution of variance across levels should be fundamental. This means-centric reductionism is mostly concerned with risk factors and creates a paradoxical situation, since social medicine is not only interested in increasing the (mean) health of the population, but also in understanding and decreasing inappropriate health and health care inequalities (variance).

### Methods

Critical essay and literature review.

### Results

The present essay promotes (a) the application of measures of variance and clustering to evaluate the boundaries one uses in defining collective levels of analysis (e.g., neighbourhoods), (b) the combined use of measures of variance and means-centric measures of association, and (c) the investigation of causes of health variation (variance-altering causation).

### Conclusions

Both measures of variance and means-centric measures of association need to be included when performing contextual analyses. The variance approach, a new aspect of contextual analysis that cannot be interpreted in means-centric terms, allows us to expand our perspectives.

People are simultaneously social and biological organisms and therefore social epidemiology distinguishes itself from general epidemiology by its inherent multilevel approach that aims to investigate both individuals and their collectives all together.[1] While the limits that define the individual biological bodies are very apparent, the collective body's geographical or cultural limits (e.g., "neighbourhood") are more difficult to discern. Also, it is normal in epidemiology to investigate causation as changes in group means even though many variables of interest may cause a change in the variance of the distribution of the dependent variable and not cause a change in the mean. So far here has been little interest in understanding changes in terms of the variance that underlies averages. [2-6] Variance is often considered a measure of uncertainty or a troublesome entity, rather than a source of substantive information. Paradoxically, this restrictive approach is the norm in many multilevel investigations, [7] whereas understanding the distribution of variance across levels should be the sine qua non of any solid analysis. [8-16] One should always remember that the goal of social medicine is not only to increase the (mean) health of the population, but to decrease health and health care inequalities (variance).

The present essay questions the usual means-centric approach and emphasises the need to deliberately investigate the heterogeneity that underlies averages. We propose, (a) the application of measures of variance and clustering to evaluate the boundaries that are to be used in defining collective levels of analysis (e.g. neighbourhoods). Further, we illustrate how a better understanding of contextual effects may be had by also (b) combining measures of variance with means-centric measures of association, and by (c) investigating the causes of health variation (variance-altering causation), rather than only considering changes in averages.

## **A. MEASURING VARIANCE TO EVALUATE THE BOUNDARIES DEFINING COLLECTIVE LEVEL OF ANALYSIS**

### **Individual bodies, collective bodies, and Frankenstein**

Researchers traditionally, investigate average characteristics of individuals (e.g., blood pressure) or of areas (e.g., social cohesion), but seldom question the “boundaries” that define the units of analysis. These boundaries are often accepted a priori. At the individual level, the limits that define the human body are very apparent, and so is the intra-individual correlation of the individual parts. Without question there is a *generalised individual effect* that maintains a sophisticated homeostasis among an array of cellular and physiological processes (e.g., blood pressure level) within the physical boundaries of our skin. As a result, when performing multiple measurements of individual variables in a group of subjects, a great proportion of the variance between measurements is at the individual level. From an epidemiological perspective this intra-individual correlation is a nuisance that, because of statistical reasons, needs be accounted for when analysing such things as repeated blood pressure measurements, diseases of the eyes or teeth, or multiple bone fractures. However, since the boundaries of human bodies are so well delineated, we can take them for granted. Instead, our research mainly focuses on whether exposure to a variable (i.e., antihypertensive medication) has an effect on, or changes, the mean of the distribution of another variable (i.e., lowering of blood pressure).

Even since the days of Durkheim [17] we know that when individuals adhere to each other and form a coherent community, a relational [18] and collective effect emerges that becomes more than the sum of each individual action. On some occasions this collective effect may have arisen because of sharing of common geographic environments, [19] and not necessarily as a result of a voluntary individual decision to form a social group. In any case, this *general collective effect* is to some extent analogous to the general individual effect

mentioned above. Like multiple measurements, individuals within a collective are themselves more similar to one another than they are to individuals outside of their group because insiders share common social boundaries and contextual influences. [10] As in the case of the individual body, the collective body maintains a social homeostasis that balances numerous social processes. However, lacking a covering skin such as human bodies possess, the collective body's geographical or cultural limits (e.g., "neighbourhood") are more difficult to discern. A further source of complexity exists: unlike the parts of a human body, [20] individuals often belong to several collectives, [21,22] both in a cross-sectional and in a life course perspective, [23]. Sometimes, however, collective boundaries are relatively easy to recognise, as is the case of schools or health care centres when investigating pupil outcomes [24] or medical treatments. [25]

While the existence of contextual effects on individual health is rather obvious, [26] the validity of many administrative boundaries for defining collective bodies [27,28] requires more investigation. [18] In fact, we cannot exclude the possibility many of the "neighbourhoods" we study are – like contextual Frankensteins – constructed by assembling parts from different collective bodies. Paraphrasing Duncan, Jones, & Moon: "the hierarchical definition of the levels could be criticized as an inappropriately formalistic and mechanistic attempt to capture the cultural geography of lifestyle". [29], p. 732

### **Can we identify appropriate collective boundaries for investigating contextual effects?**

While a number of collective bodies (e.g., neighbourhoods) are assumed to be exclusively delimited by geographical boundaries, other cultural or relational criteria (either alone or combined with geographical information) may be more appropriate for identifying collective bodies. [18,30,31] In any case, if such a collective body exists, one might anticipate finding a correlation in the health of the individuals within it. [10] Moreover, the closer our boundary

definition corresponds to the boundaries of the hypothesized collective body, the greater one may expect the clustering of the health outcome of interest to be. [10,32] Based on this assumption we can try to identify collective boundaries in one of two ways: either by (a) scanning a geographical surface to identify clusters of the outcome (e.g., specific diseases), or by (b) using a priori defined socio-geographical areas and quantifying the observed clustering of disease outcomes within these areas.

In the first instance above, cluster recognition analysis [33,34] may help us to identify geographical clusters of the outcome of interest (e.g., the incidence of cholera). Once the relational [18] boundaries that include the clusters have been identified, we can investigate what makes them different from the rest of the geographical surface (e.g., the water supply) and thereby obtain relevant information for drawing causal hypotheses and planning public health interventions. This idea is close to John Snow's approach in his seminal work on cholera. [35] In the second scenario (b) the most common procedure is to employ geographical divisions found in administrative databases (e.g., census tracts). Alternatively, such areas may be combined on the basis of certain characteristics [31,36,37] or by using Geographic Information Systems (GIS) techniques. [34]

Measures of variance and clustering (e.g., intraclass correlation, [15,38] median odds ratio, [9,39,40] or pairwise odds ratio [32]) allows us to identify the scale (e.g., local neighbourhoods, parishes, or municipalities) [41,42] on which contextual influences operate with different health outcomes.[43] For example, in 1999 Boyle & Willms performed an unconventional multilevel analysis exclusively based on measures of intra-class correlation and places defined by administrative boundaries. [44] They observed that place effects were generally small and were influenced by both the size of the geographical area used to define place and the health indicator selected for study. The authors questioned the usefulness of carrying out health needs assessment surveys within large administrative areas, and casted

doubt on the utility of these geographic boundaries for studying place effects. Boyle & Willms based their conclusions not on means-centric measures of association, but on the analysis of variation, and they raised questions about the context as a whole. Their work, has resulted in some criticism (see, for example, Blakely [45] p. 373).

## **B. COMBINING MEASURES OF VARIANCE AND MEAN-CENTRIC MEASURES OF ASSOCIATION FOR A BETTER UNDERSTANDING OF CONTEXTUAL EFFECTS**

### **Small variance but conclusive associations**

It may appear paradoxical that a conclusive (“significant”) association between a contextual variable and an individual outcome can be detected alongside a very small fraction of overall variance in the outcome at the contextual (e.g., neighbourhood) level. [46] Thus, means-centric measures of association indicate the existence of contextual effects, while measures of variance suggest the opposite. In an attempt to justify this paradox [47,48], it has been commented that **standardized mean differences (d) between intervention and control neighbourhoods, that program evaluators commonly view as medium (d = 0.4) or even large (d = 0.6) translate into ‘small’ intra-neighbourhood correlations of 4% and 8% respectively.** Several criticisms can be raised to this reasoning, with the most fundamental being there is no need for such a justification. As our essay argues, such paradox does not exist but, rather, the question is to distinguish between the different information provided by measures of association and by measures of variance. Furthermore, it has been **previously** revealed that **standardised coefficients [47] are inappropriate measures of effect as they can be “confounded” by the variance of the specific setting where the study is performed (see Greenland [49] and Cummings[50] for an extended explanation).** Standardized effects are



actually distorted measures of association and also hide the information contained in the variance being standardized..

In addition, while intraclass correlations ranging from 4% - 8 % may be considered small by some [47], they may be highly relevant for others particularly when compared with the intraclass correlations of many “neighbourhood” outcomes relevant for social epidemiology. [51]

The fundamental difference between measures of variation and means-centric measures of association becomes clear if one understands that the interpretation of variance is often temporo-spatially constrained and that for every individual outcome there may be a pattern of variance produced by different environmental conditions. [52] Thus, in seeking useful information for planning public health interventions, measures of variance and clustering from a specific context provide insight into the areas being investigated during the period of the study. By contrast, measures of association intend to provide causal information that can be generalised and applied to contexts beyond the one where the study was performed.

Further illustration of this idea using a continuous contextual variable is given in the figure 1 and its accompanying legend. See also a previous study published elsewhere in this Journal.[11])

*-[Figure 1 about here]*

We can even clarify this apparent contradiction in an equation that can be calculated from a two level logistic regression model. As an example, imagine individuals are nested within neighbourhoods and the aim is to analyze the relation between neighbourhood deprivation (X) and presence of hypertension. Without loss of generality, assume the

contextual variable is centred on the mean. It can be shown that the total neighbourhood variance  $\sigma^2_T$  (as originally estimated in the simplest “empty” model with only individual nested within neighbourhoods) is a function of the regression coefficient of the contextual variable ( $\beta^2_X$ ) the variance of this contextual variable ( $\sigma^2_X$ ), and of the residual variance ( $\sigma^2_u$ ) once the contextual variable is included in the model. Therefore it is possible to find a similar  $\beta_x$  with very different scenarios of variance.

$$\sigma^2_T = \beta^2_X \cdot \sigma^2_X + \sigma^2_u \quad \text{Equation 1}$$

Failure to distinguish between the two types of measures explained above, and specially, interpreting means-centric measures of association as if there were measures of variance may lead to inappropriate conclusions, a situation that is unfortunately rather common in many multilevel analyses performed today. As one example, in 2001 Diez-Roux et al, [53] performed a state of the art and thoroughly conducted multilevel analysis of the relationship between characteristics of neighbourhoods and the incidence of coronary heart disease. Possibly, since the samples of individuals within blocks in this Atherosclerosis Risk in Communities Study (ARIC) were very small, the authors did not estimate neighbourhood variance. They did, however, assume the existence of intra-neighbourhood correlation in the outcome, which was considered a statistical nuisance and overcome by adjusting standard errors for clustering using the statistical software SUDAAN. The size of this conjectural intra-neighbourhood correlation was never reported. The study drew two main conclusions: (1) “Neighbourhood characteristics are related to the incidence of coronary heart diseases” and (2) “Strategies for disease prevention may need to combine person-centered approaches with approaches aimed at changing residential environments.” While these conclusion are of clear academic interest for understanding the contextual causes of coronary heart disease, they are also vague and may even be misleading. An approach aimed at changing residential environments may be effective in the ARIC context, but since we do not have information on

the intra-neighbourhood correlation (or similar measures of variance), we cannot be certain. For example, although an analogous multilevel study in Malmö, Sweden, [54] demonstrated a clear association between the socioeconomic characteristics of the urban areas and individual blood pressure (in agreement with the ARIC study) [53], differences between these same areas explained less than 1% of the individual variance in blood pressure. Consequently, the study concluded that, with regard to Malmö, an intervention focused on urban areas with a higher mean level of deprivation would be ineffective [4,11]

We should not avoid a contextual analysis because the area variance or the intra-class correlation is very small, since means-centric associations between contextual variables and individual health may still be detected. On the other hand, neither can one recommend a contextual public health intervention based on a “significant” means-centric association if the clustering of individual health within areas is unknown or very low. Means-centric measures of association do not provide sufficient information for deciding to launch public health interventions **at some specific areas but not at other**. In fact, if clustering is small, a public health intervention would be ineffective, even where a contextual variable is associated with the individual outcome and serves to explain 100% of the area variance. As Singer has stated “You can explain a large amount of very little” [55], p. 332. A different scenario is also possible in which we observe a clear association between the contextual variable and the individual outcome side by side with a very large residual area variance. In this case, if the contextual variable does not go very far toward explaining the original area variance, a public health intervention aimed at changing this contextual variable would not be very ineffective.

As discussed earlier, measures of variance and clustering are useful for identifying boundary limits of the “collective body” that we assume influence the outcome under study. In this section we sought to demonstrate that such collective boundaries (i.e., “the geographic scale”) cannot be properly identified solely by means-centric measures of

association. For this purpose, means-centric measures of association must be combined with measures of variance and clustering.

### **New analytical approaches – solving the paradox**

In an earlier publication, [9] we have given empirical examples of the two scenarios depicted above, and have also sought to apply a pair of useful measures proposed by Larsen et al [9,40]: the median odds ratio (MOR) and the interval odds ratio (IOR) (see the [Tutorial published elsewhere in the Journal for an extended explanation of these measures](#))[39]. The MOR quantifies neighbourhood variance on the odds ratio scale, and the IOR incorporates both the means-centric effect (i.e., odds ratio) and neighbourhood variance in one interval, allowing for a more detailed description of the means-centric effect.

In our [previous](#) study, [9] we found the administrative neighbourhoods of Malmö were very suitable for identifying the “collective body” that conditions certain individual behaviours, such as choosing to visit a private rather than a public physician. For this condition, the area variance (SE) with adjustment for age and individual education was 1.815 (0.278) and the correspondent MOR = 3.61. However, the same neighbourhoods seemed inappropriate for identifying the “collective body” that conditioned hospitalisation for ischemic heart disease, since the corresponding variance (SE) was [only](#) 0.028 (0.025) and the MOR = 1.17. In the same study, the socioeconomic characteristics of the neighbourhood appraised by aggregated educational achievement (low vs. high) were, [however](#), associated with both outcomes. [Nevertheless](#), in spite of this observed association (and disregarding concerns about counter-factuality) we concluded that a possible public health intervention directed to specific neighbourhoods would be ineffective in either instance. In the case of [hospitalisation for](#) ischemic heart disease the inefficiency [would depend on the](#) very low neighbourhood variance. Finding such a low area variance in cross-sectional studies is rather common for chronic diseases which, like arteriosclerosis, develop over a whole life-course

and have little to do with the place where an individual may be currently residing. **When it comes to change individual's choice of physician, a neighbourhood intervention** would similarly be ineffective because of the large variance that remains unexplained after including the variable “neighbourhood level of education” in the model. The latter expressed itself in a very broad IOR = 0.28–27.3. There is little doubt that the current neighbourhood context influences individual behaviour much more than chronic disease, but “neighbourhood educational level” – in spite of being associated with the choice of physician – **does not explain very much in the city of Malmö.**

We state that combining variance-based measures with means-centric measures of association provides useful and complementary information on contextual effects. **These considerations may be relevant when attempting to determine the efficacy of focusing intervention on places rather than on people.** For example, imagine that a City Council has been informed that average blood pressure is higher in deprived neighbourhoods than in wealthy neighbourhoods. As a consequence, decision makers are considering the allocation of resources in the most deprived neighbourhoods for the creation of new health care centres specialized on blood pressure control. However, if the neighbourhood variation represents only a very small part of the total individual variation in blood pressure, then many people with high blood pressure would be ignored simply because they reside in wealthy neighbourhoods. When the clustering of individual health status within neighbourhoods is small, focusing intervention on specific places may be a rather inefficient strategy. [4,11] Using the words of Clarke: “without knowledge of the random components, the interpretation of area-level fixed effects parameters becomes *decontextualized*”. [37] p. 315.

### C. INVESTIGATING THE CAUSES OF HEALTH VARIATION

Epidemiologists commonly understand causation in terms of group means, so the statement - “X causes Y” is taken to imply that, *ceteris paribus* (see references [14,48,56,57] for a discussion on this aspect), an increase in the value of X changes the mean of the distribution of Y. However, many independent variables of interest in epidemiological studies may cause a change in the variance (not the mean) of the distribution of the dependent variable [2-6] (see figure 2).

*[Figure 2 about here]*

The distinction between the variance altering and means-centric altering approaches is still not widely observed in social epidemiology; most researchers only discuss classic means-centric measures of association. This means-centric reductionism goes hand by hand with an epidemiology mostly concerned with risk factors [58] and drug safety and creates a paradoxical situation, since social medicine is not only interested in increasing the (mean) health of the population, but also in decreasing health inequalities (variance). Likewise, it is of major relevance to understand and prevent inappropriate health care variation, as it leads to inefficient resource utilization. Modelling variance itself as a dependent variable may provide useful information on health inequalities and suggest a different kind of contextual effect. [6,13,25,59]

When investigating such variance-altering causation in contextual analysis, a fundamental independent variable is the definition of boundaries that we use to operationalise collective bodies (as we have attempted to show in the first section above). For example, we include ‘neighbourhood’ as a random term in multilevel regression analyses. We can consider the boundaries that define a specific level of analysis as an independent variable in an equation that models variance. In this way we can investigate (potential) causation where the

chosen boundaries – assuming they delimit a true collective body – “cause” a certain pattern of individual differences/similarity. This **has been the** approach adopted, for example, by Boyle & Willms [44], **Reijneveld [60], Petronis & Anthony [32]** and by ourselves [6,25,46,54] as well as recently **by Uthman, Moradi & Lawoko [61] and by Naess et al [62].** **This last work** explores area variation across a life-course as a way of elucidating potential (causal) influence of area on mortality.

Modelling individual and area variance may yield valuable information on how contextual factors shape health inequalities for different individuals. In a previous study based on the MONICA project [6] we investigated contextual effects on individual systolic blood pressure, and modelled variance as a function of antihypertensive medication use and body mass index. Among other results we found that contextual effects were particularly strong in overweight women on antihypertensive **medication. Actually, around 20% of the individual differences in blood pressure were conditioned by the MONICA population where these women were included. This contextual phenomenon** possibly reflects **disparities in the effectiveness of antihypertensive treatment among different** national health care systems (see figure 3 in reference [6]).

In a recent study [25] we have explicitly investigated variance altering causes and presented a conceptual illustration showing that a change in the characteristics of a context (e.g., the implementation of a decentralized health care budget) not only changes the mean of the distribution of the variable studied (i.e., increasing compliance with prescription guidelines), but alters the variance between the collective units as well (i.e., decreasing inequality between health care centres). Downs & Rocke [2] and Braumoeller [5] also provide illustrative examples in their work.

## CONCLUSIONS

Both measures of variance and means-centric measures of association need to be included when performing contextual analyses. However, more research is needed to (a) identify appropriate boundaries for collective bodies like neighbourhoods, (b) develop statistical methods that facilitate the use of measures of variance in social epidemiology, (c) identify variance-altering causes and their mechanisms, and (d) comprehend the relationship among the degree of clustering of individual health within administrative areas, the size of means-centric measures of association, and the possible efficiency of public health interventions.

**Seeking causal explanations in social epidemiology is a challenge in itself,[63]**

**but** focusing on the (causal) circumstances that condition variance reveals a neglected theoretical dimension for understanding health disparities in social epidemiology. The variance approach, a new aspect of contextual analysis that cannot be interpreted in means-centric terms, allows us to expand our perspectives.

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**Contributorship Statement**

JM had the original idea of this study and wrote the manuscript. HO, KFL, BC and SS have actively contributed to the discussion and interpretation of the ideas presented in this essay. All authors have critically revised this article for important intellectual content. All the authors have read and approved the final version to be published.

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## LEGEND TO THE FIGURES

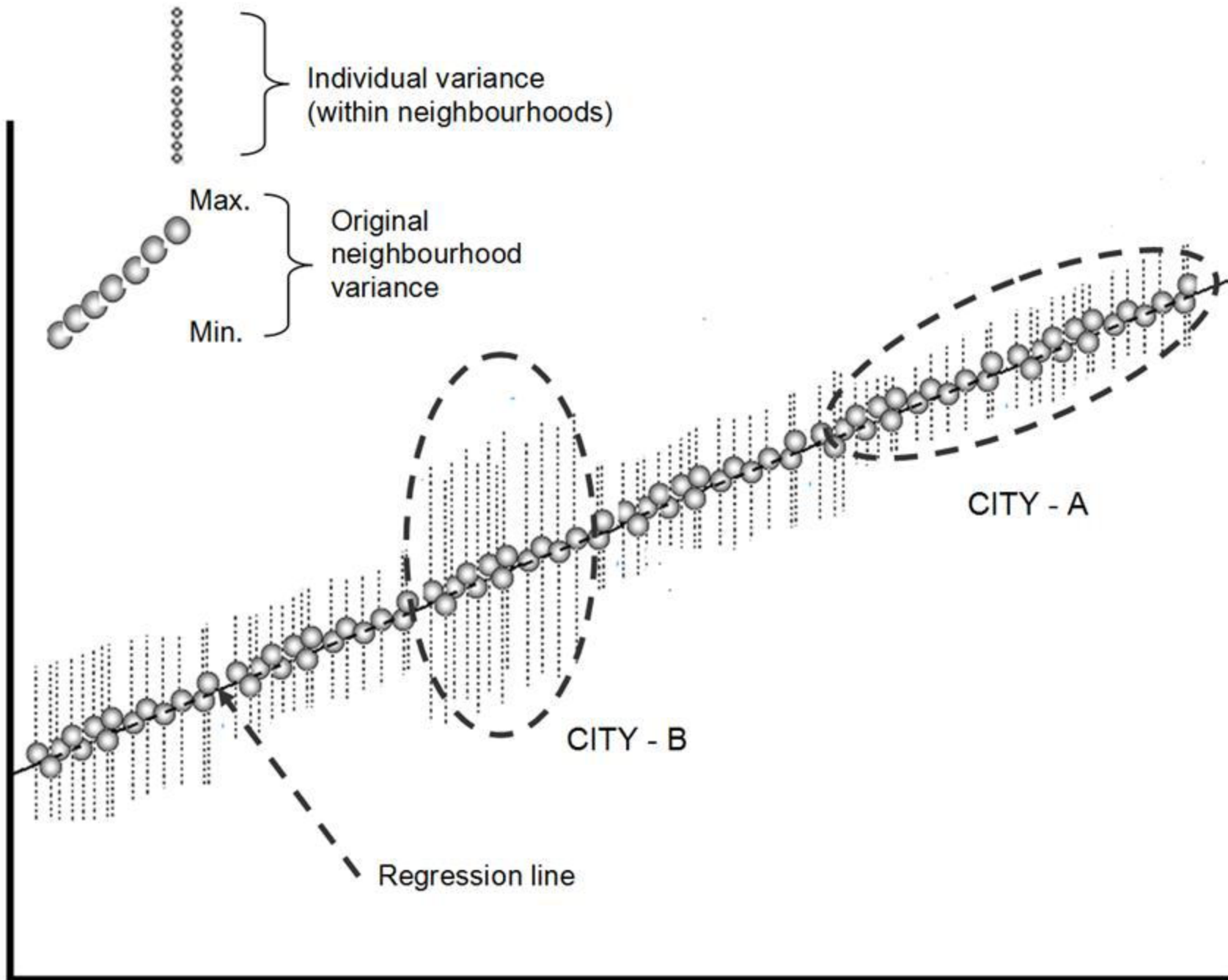
**Figure 1.** Visualization of a hypothetical association between a contextual variable (e.g., extreme poverty to extreme wealth) and individual **blood pressure**, based on data on from neighbourhoods in different cities around the world. In this representation the contextual variable explains 100% of the variance between neighbourhoods, since all areas are located on a regression line. However, while the regression coefficient is identical in all cities (i.e., a one unit increase in wealth results in an equivalent increase in health), the original neighbourhood variance before the introduction of the contextual variable was lower in city B than in city A. The original neighbourhood variance also corresponds to a much larger proportion of total individual variance in city A as opposed to city B. Consequently, a contextual public health intervention would be more efficient in city A than in city B. The key for this intervention is the size of the neighbourhood variance.

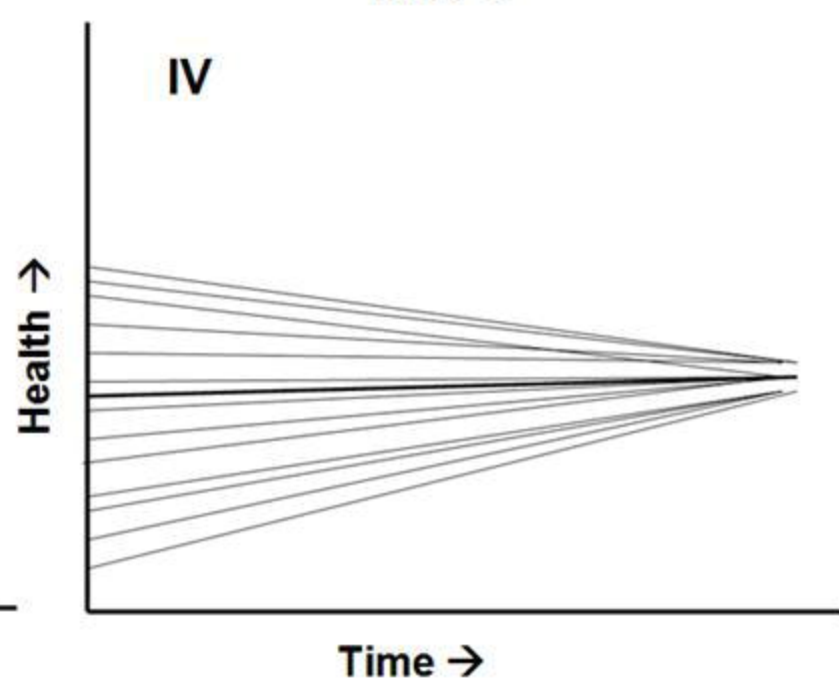
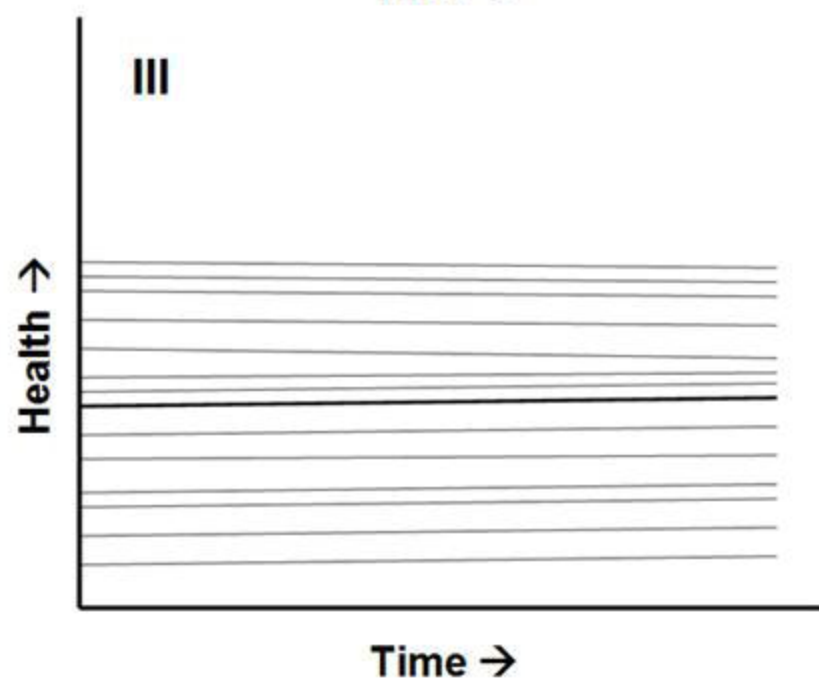
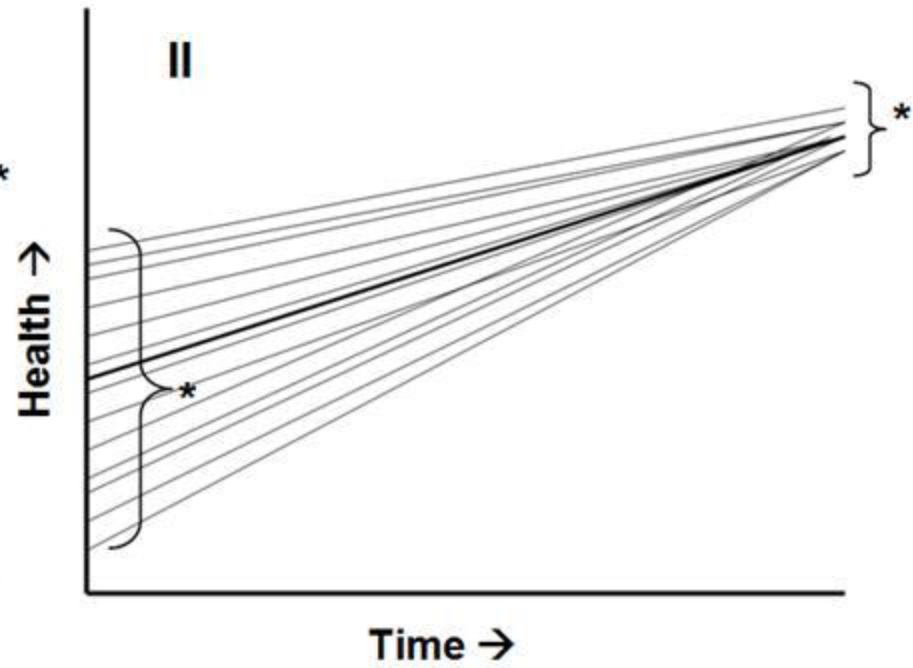
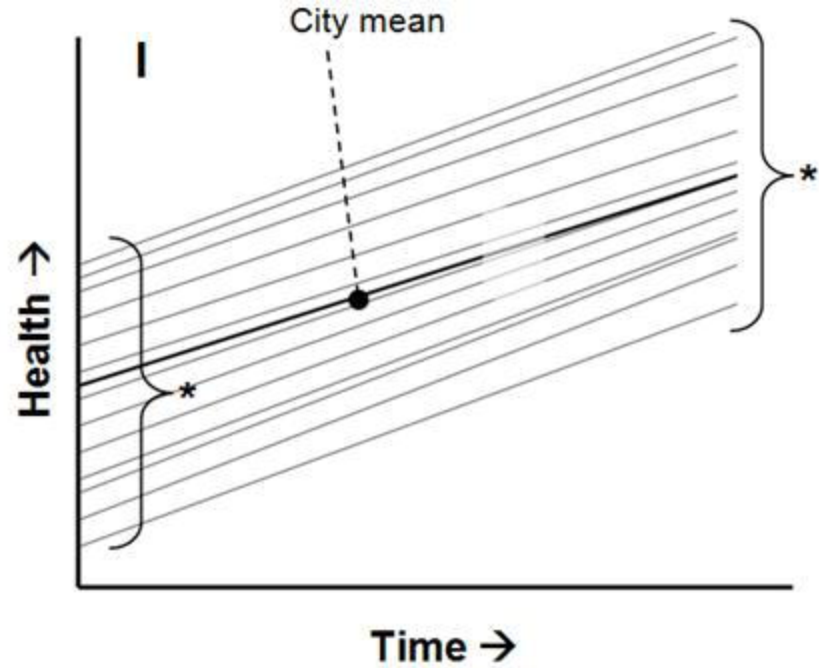
**Figure 2.** Visualization of an imaginary association between the interval of time after the start of a contextual intervention in a hypothetical city and mean individual health measured across (1) the entire city (thick line) and (2) in each of the city's neighbourhoods (thin lines). The measure of association (regression coefficient  $\beta$ ) is positive; it is similar in I and II and shows an increase of the overall mean from the start of the intervention. However, health inequalities (i.e., neighbourhood variance) do not change along the time axis in scenario I, but are considerably reduced in scenario II.

The regression coefficient ( $\beta$ ) is zero in both III and IV, which might be interpreted as an absence of contextual effects, since the intervention does not seem to influence health.

However, while the health inequalities represented by neighbourhood variance have not changed in scenario III, they have been greatly reduced in scenario IV.

Individual health →





\*Neighbourhood variance