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Stronger sex but earlier death: A multi-level socioeconomic analysis of gender differences in mortality in Austria

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Abstract

Gender inequalities in mortality/life expectancy have been a major area of research in the social sciences since the 1970s. However, the questions posed and the research strategies used are still in a state of flux. In the present paper we shed some light on two related questions: (i) Which socioeconomic variables determine the gender gap in mortality? (ii) Are male and female mortality rates determined by different socioeconomic factors and in different shapes? We use aggregated data from Austria both at the community and district level covering the time period 1969 - 2004. Our two-level empirical design combined with a panel structure at the districts level reveals additional evidence on these questions compared to previous studies at the regional level. By using weighted regression analysis (panel fixed effects, pooled and cross section) we find that the gender gap is negatively associated with higher average net income, a higher educational level, a higher share of immigrants and better familial integration. In general, males are more sensitive with respect to social and economic conditions compared to females, leading to a narrowing gap in mortality when living conditions improve. These results are also confirmed by our Blinder-Oaxaca Decomposition.

JEL classification: I12, I18, J16 **Keywords:** mortality, gender mortality gap, life expectancy, Austria

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1 Introduction

Roughly 250 years ago the famous French mathematician Antoine Deparcieux studied the mortality rates of monks and nuns in French monasteries finding a longevity gap in favour of females. The almost continuous increase of the gender gap in mortality/longevity in the following centuries (for comprehensive empirical evidence see Kalben 2002, Case/Deaton 2003, Case Paxson 2004, Cutler/Meara 2005, Cutler et al. 2006, Trovato/Lalu 2005, Trübswetter/Klasen 2007, Waldron 1986, Waldron 2000, Zielonke 2007) initiated a broad scientific research movement on this topic. The mid of the 1980s to some extend marks a turnaround in the gender gap. Data from OECD30-countries show a sharp increase in the gender longevity gap at birth, starting from 5 years in 1960 to 7.3 years in 1977. The gap then is pretty stable until 1994. Since then it decreased continously to 5.6 years in 2008. Today gender mortality related questions have attracted interest in various scientific disciplines. Roughly speaking, we are able to identify three main strands of approaches, stressing different determinants of the gender mortality/longevity gap: (i) *biological-genetic*, (ii) *environmental-behavioral* and (iii) *economic*.

Biological-genetic differences influence the gender gap in mortality either directly or indirectly. There exist various biological-genetic theories to explain the mortality gap such as the role of the X-chromosomes, androgens, estrogens and progestins, iron overload, natural selection etc. (for an overview see Kalben 2002 or Luy 2002). To isolate the biologicalgenetic effect from behavioral-environmental factors different empirical approaches have been used. These include empirical evidence from different species of animals (see Kalben 2002), investigations of differences in prenatal and neonatal mortality between female and male foetus and babies (see Sahn/Stifel 2002 and Siow/Zhu 2002) or studies about groups of the population where the behavioral-environmental embedding is similar, such as nuns and monks (see, for instance, Luy 2003). Overall, it seems to be clear that biological factors are able to explain the gender mortality gap only to some extent, especially at the aggregate level. However, recent changes in the differences in life expectancy/mortality can hardly be explained by biological factors alone.

Behavioral-environmental approaches focus on the role of working conditions, social roles, environmental behavior, political and civil rights etc. A wide array of studies try to explain mortality differences by consumption behavior, in particular by highlighting the role of smoking (see Pampel 2002, Boback 2003, Valkonnen/Poppel 1997, Preston/Wang 2005 among others), alcohol and accidents (Pampel 2001).

There are only a few papers which apply economic approaches to explain differences in mortality/longevity between men and women. Within these approaches longevity basically is a matter of the optimal amount of health investment in life extension (for the basic model see Galor/Weil 1996). To account for differences in the embedment of the optimisation different models are used. In this line of research, Klasen (1998) developed an intrahousehold resource allocation model to explain the excess female mortality during the Early German Development 1740 - 1860. Felder (2006) studied the gender longevity gap in favour of women assuming different utility functions of singles and couples.

From a conceptual viewpoint differences in health are influenced (i) by individual characteristics, (ii) by the level and structure of individual characteristics at an aggregate level (f. e. local community, region, state) and (iii) by contextual factors at an aggregate level (see Diez Roux 2003). Thus, this would call for multilevel approaches in studying gender differences in mortality. Due to data limitations, multilevel studies of the gender gap in mortality including individual and aggregate data are quite rare. Previous research mainly uses cross-country data (see, for instance, Mustard and Etches 2003, Gjonca et al. 2005, Glei and Horiuchi 2007 or Gray et al. 2006), while studies at the local (regional) level are quite rare. To our knowledge, only two similar studies have been conducted.

Spijker et al. (2007) investigated the gender gap in mortality in the Netherlands, using data from 40 regions with a median size of about 300.000 inhabitants. The exogenous variables chosen for the multivariate analysis are distinguished into four types, namely (i) socioeconomic factors (education and employment), (ii) familial gender roles (fertility level and divorce), (iii) behavioural factors (smoking, consumption of alcohol) and (iv) information on some contextual factors that might confound the regional gender differences in mortality (income inequality, indicators of relative deprivation, urbanisation level, religion, ethnic composition). By using cross-sectional data from 1980-83 and 1996-99, they find that the gender gap in mortality declined between the two periods by 1.2 years, while the determinants used are only significant in some dimensions, leaving others unexplained.

Anson (2003) presents a study of sex differences in mortality at the local level for Belgian municipalities. In particular, Anson stresses the importance of a broader socioeconomic perspective to explain the gender gap. The particular pattern of female and male behavior, the differences between them and their partial convergence over the past quarter of the last century or more, are reflective of male and female roles. Anson explains the gender based mortality differences as a result of an interactive social and biological process, where biological and behavioural facts are socially determined and influenced. In a nutshell, the most important findings are that (i) mortality is lower, for men and for women, in populations with higher socioeconomic status, (ii) in populations with stronger social attachments, and (iii) in populations with a higher concentration of immigrants, whereas (iv) women show a weaker sensitivity than men to socioeconomic conditions. Thus, when social conditions are more favourable to longer life, the male-female mortality gap is smaller. Interestingly, the hypothesis that the female mortality advantage is

inversely related to the male social advantage cannot be confirmed in this study.

In several respects our study follows the research strategy of the Anson (2003) paper. Basically we focus on two questions, namely (i) Which socioeconomic variables determine the gender gap in mortality? (ii) Are male and female mortality rates determined by different socioeconomic factors and in different shapes? To answer these research question we focus on the local community and the district level and use data on both levels of regional aggregation. This can be rationalized by the fact that there are important differences in the social, economic and environmental conditions in different localties, directly affecting mortality risks for men and women. Moreover, based on our hypotheses stated below, we expect that the conditions in a given community affect the mortality risk of men and women differently.

We are aware of the fact that the regional entities chosen are not homogenous, and that therefore the results cannot be assigned to mortality risks for individuals. The considerable variations between communities both in mortality rates and socioeconomic conditions, however, can nevertheless be helpful to identify important determinants of mortality rates, both for males and females. Our findings do not inform on individual mortality risks in a given community or district, it rather enhances our knowledge about general social and economic conditions and their effect on mortality. We are also aware of the possible problems caused by the disconnection between the level of analysis and the level of inference (see, for instance, Sheppard 2003, Diez-Roux 1998 or Greenland/Morgenstern 1989). However, we take account of the possible "ecological bias" problem by applying a multi-level analysis, where we analyze the effect at two different geographical levels. By doing so, we basically follow the suggestion by Robert (1999) who proposed to "include information about self-defined communities or at least purposefully delineate community boundaries to more closely match the theoretical constructs being tested" (p. 509). Although the boundaries of our regions are still exogenously given (and probably arbitrarily chosen), we nevertheless offer a comprehensive study design by including two different geographic levels. Furthermore, as Robert (1999) pointed out, the community socioeconomic context affects health through two major pathways, namely (i) by shaping the socioeconomic position of individuals, and (ii) by directly affecting the social, service, and physical environments of communities shared by residents, which then affect the individual characteristics and experiences that more directly influence health, or mortality, respectively.

Our paper extends the results of previous studies on regional differences in the gender mortality gap in several respects. We use data from different levels of regional aggregation, which allows to cope with the "ecological bias" at least partially. Our data cover a long time period of almost fifty years (1969 - 2004) and therefore include changes in the spectrum of diseases. Additionally, at the district level, we are able to use the panel structure in our data in the empirical estimation.

The paper is structured as follows. Section two explains the methodology used to examine the gender mortality gap and presents the most important literature on this topic. Section three includes an overview of our dataset, while our main findings are presented in section four. Finally, section five draws some conclusions.

2 Methodology & Hypotheses

To organize the empirical part of our study we present the hypotheses tested and substantiate them briefly by using the theoretical reasoning and empirical results of previous studies (see, for instance, Anson 2003).

• Hypothesis 1: Mortality will be lower in communities/districts with higher socioeconomic status.

In recent decades, unprecedented decreases in mortality rates are observed, which were accompanied by high growth rates both in income and prosperity. The explanations put forward are quite intuitive, as richer countries have more resources for health care, and a higher income is also often linked to "healthier" jobs, as compared to the situation of non-skilled workers. This relationship is confirmed in several empirical studies (see, for instance, Krieger 1992, Anson 2003, Spijker et al. 2007, Mustard and Etches 2003). Robert (1999), in his extended literature review, states that community socioeconomic level is linked to individual health and mortality, "over and above the impact of individual socioeconomic position" (p. 498). We include three dimensions of the socioeconomic status at the regional level: income, education and labor participation. While the expected influence of income and education seems to be clear, the impact of the labor participation rate is less obvious from a theoretical perspective. A higher participation rate usually corresponds to higher income and educational levels, but higher levels of labor force participation might also reduce the time to invest in health. Moreover, the participation rate is also relevant from a health care service utilization perspective. A higher participation rate as well as a large gender mortality gap may cause problems, particularly in provision of informal care for the elderly. In any case, the conventional explanation that excess male mortality is caused by greater male labor force participation is not supported by empirical evidence (see Waldron 1991 or Pampel and Zimmer 1989).

• *Hypothesis 2:* Mortality will be lower in communities/districts with stronger social networks.

From a broader socioeconomic perspective, social attachments are an important determinant of mortality rates. Hummer et al. (1998) describe mortality as a "socially influenced biological process" (p. 565), where biological and behavioural facts are socially determined and influenced. In recent decades, theories of social capital (Coleman 1988, Putnam 1993) have stressed the importance of social relationships, social organization, norms of reciprocity, and civic participation in promoting social good in the society. Indeed, there are quite a lot of empirical studies confirming the positive effect of social networks on health or mortality (see, for instance, Anson 2003, Klasen 1998, Trowbridge 1995 among others). It has been suggested that lower mortality rates are caused by healthier lifestyles in social networks of strong family structures. Klasen (1998) claims that the marital status is by far the biggest determinant of mortality. Both married males and females have a lower mortality than their single counterparts. Moreover, patterns of social relationships also seem to have different meanings for men and for women, e.g. the beneficial effects of marriage on mortality were consistently stronger for males than for females, especially compared with that of the divorced (see Murphy 2000, Gove et al. 1990, Zick and Smith 1991, Rogers 1995). The reason for this effect is not entirely clear from a theoretical perspective (see Anson 2003). The benefit could be derived from social integration (embeddedness in a network of social relations), social regulation (social control implicit in institutionalized marriage) or even from a selection effect for those who marry. However, there is strong empirical evidence that stronger social and familial attachments lead to better health and lower mortality.

• Hypothesis 3: Mortality will be lower in communities/districts with a higher share of immigrants.

Previous research shows that the share of immigrants within a region seems to have an effect on mortality rates. In fact, as Anson (2003) points out, the social influence of immigration on regional mortality is not yet well studied and understood. Nevertheless the empirical picture seems quite clear, as mortality is significantly lower in regions with a higher share of immigrants or foreigners (see, for instance, Abraido-Lanza et al. 1999). Interestingly, the low mortality rates among immigrants are also confirmed by studies at the individual level (Landale et al. 2000). Common explanations (see Anson 2003) range from selection effects (immigrants might be healthier) to the meaning of voluntary migration (taking control of one's life) and to the solidarity created within marginalized migrant communities. Although there is little evidence in the literature, the overall higher sensitivity of males to socioeconomic circumstances would also suggest a stronger impact of the share of immigrants on males compared to females.

• Hypothesis 4: Mortality will be lower in communities/districts with a more ho-

mogenous population.

Previous research indicates that not only the level of the socioeconomic status, but also its structure (inequality in the distribution) within a region influences health and mortality. More precisely, inequality in the income distribution within a region or a state is associated with higher morbidity and mortality rates, which is not accounted for by the average socioeconomic level (Ben-Shlomo et al. 1996, Kaplan et al. 1996, Wilkinson 1992, 1996, 1997, 2006). Recent literature has proposed several explanations for this phenomenon. First, inequality in terms of income and education within regions leads to lower levels of social cohesion and trust among residents, as well as feelings of relative deprivation, which in turn affects health in a negative way (Kawachi et al. 1997, Wilkinson 1996). Although the empirical evidence seems to be clear in this respect (for an extended literature review see Wilkinson 2006), it is still controversial whether these ecological relationships reflect a causal relationship whereby inequality in society affects or reflects the social cohesion and social capital of a region (Kawachi et al. 1997). Second, regions tolerating higher inequality in socioeconomic variables are also less likely to provide generous human, social, and health resources that ultimately affect the health of residents (Davey Smith 1996, Kaplan et al. 1996). Third, the "relative deprivation" hypothesis (Robert 1999, p. 496) suggests that individuals with lower socioeconomic position are worse off in richer communities as they might experience negative health effects of structural relative deprivation when competing with neighbours for scarce resources, and psychosocial relative deprivation when comparing oneself to neighbours with higher socioeconomic position. A fourth explanation states that the link between inequality and negative health effects reflects a curvilinear relationship between socioeconomic status and mortality at the individual level (Fiscella and Franks 1997, Gravelle 1998). Therefore decreasing returns in the health production function would imply a lower health status in more heterogenous populations. Finally, communities with high socioeconomic inequality often have higher levels of actual or perceived crime (Hsieh and Pugh 1993). This can directly (bodily harm) and indirectly (fear of crime increases stress and promotes social isolation) affect the health of the residents (Macintyre et al. 1993, Sooman and Macintyre 1995).

• Hypothesis 5: Men health shows a stronger sensitivity to environmental conditions compared to women, implying that the male mortality disadvantage will decrease with improving socioeconomic conditions as well as stronger social networks.

There exists empirical evidence that the patterns of social relations have different meanings and effects for men and women, e.g. men benefit more from marriage compared to women (see, for instance, Durkheim 1951, Gove 1973, Rogers 1995, Murphy 2000). Similarly, male mortality levels are more sensitive to differences in standards of living (Park and Clifford 1989, Macintyre and Hunt 1997), as the mortality gap is at its lowest for the highest socioeconomic status (Rogers et al. 1999). In this line of research, LeClere et al. (1997) found that socioeconomic indicators at the community level were better predictors of mortality for men than for women having controlled for the individual-level socioeconomic position. Similarly, Koskinen and Martelin (1994) showed that women's mortality varies less by socioeconomic status than men's mortality. Based on Finnish data they argue that differences in the sensitivity of mortality rates by gender might result from the fact that the relative magnitude of inequalities among women is considerably smaller than among men (particularly in the married subpopulation). Furthermore, the socioeconomic mortality gradient varies by causes of death, and thus, the differences could be restrained to specific death causes (leading to an overall higher male mortality sensitivity to socioeconomic factors). This results are confirmed by an international study by Mackenbach et al. (1999). They analysed mortality data from the US, Finland, Norway, Italy, the Czech Republic, Hungary and Estonia and conclude that the larger socioeconomic inequalities in total mortality among men compared to women is largely due to sex differences in the causeof-death pattern.

• Hypothesis 6: The influence of socioeconomic variables on the gender mortality gap also depends on the corresponding 'gender gaps' in these variables.

There is a controversial debate whether the increasing gender equality in European societies has been a driving factor for the observed narrowing of the gender mortality gap. Given that the higher socioeconomic status of men traditionally coexisted with higher levels of male mortality, it seems obvious that movements towards social and economic equality between men and women will result in a convergence of mortality rates (see Annandale and Hunten 2000 for a review). However, many scholars reject the claim of a causal relationship between the women's low status and their mortality advantage, arguing that the differential will in fact widen with increasing gender equality in the society. Indeed, a wide array of studies tries to explain mortality differences by consumption behaviour, in particular by highlighting the relationship between smoking and longevity (Pampel 2002, Boback 2003, Valkonnen and Poppel 1997, Preston and Wang 2005 among others). Following this line of research, Carey and Lopreto (1995) present a model which interacts biological and behavioural components, suggesting that the biological origins of the female mortality advantage are twofold: an evolutionary process which, on the one hand, favoured robust females who could withstand the rigours of pregnancy and childbirth, and on the other hand, favoured aggressive males who were not averse to risk-taking behaviour. In today's more developed societies, reduced fertility and medical techniques

have overcome the threats of childbearing, while male risk-taking behaviour has not been overcome culturally. The result is excess male mortality, particularly from causes associated with violence, alcohol and cigarettes. Pampel (2001) studied the role of accidents on mortality differences. In essence, Pampel is able to confirm the hypothesis that the female advantage in accident mortality declines over time and that indicators of gender equality reduce the female mortality advantage, albeit no clear pattern of a causal relationship can be shown. Conti et al. (2003) examined gender differentials in life expectancy in Italy, concluding that the slight reduction of the gender differential since 1980 seems to be the result of the recent adoption of unhealthy life styles by women together with a healthier behavior of young men. Similarly, Backhans et al. (2007) investigated whether increased gender equality leads to a convergence of health outcomes for men and women by using data from Sweden. While this hypothesis was confirmed for equality of part-time employment, managerial positions and economic resources to treat morbidity, their main finding is that gender equality was generally correlated with poorer health for both men and women. Interestingly, Anson (2003) is not able to confirm the hypothesis that the female mortality advantage is inversely related to the male social advantage.

3 Data

3.1 Dependent Variables

We extracted sex-specific Standardized Mortality Rates (SMR) for the 2377 communities¹ and 118 districts (including the 23 districts of Vienna)^{2 3} using information from the *Atlas* of Mortality in Austria by Causes of Death of the official central bureau of statistics in Austria (Statistik Austria 2007). Official death records include the information on the place of residence, age, sex and cause of death. This information is combined with the results of

¹Following the NUTS-classification the local community level is LAU2. There were minor changes in the number of the local communities within the observation period due to unification movements. We adjusted for these changes in our data. Vienna is counted as 23 local communities mirrowing the districts in Vienna. In the Austrian political system local communities act as agents in the administration of public functions of the central state and the provinces and fulfill several tasks self-governed. The mean size (population) of the communities is 3373, the median is 1575 (in period two).

²Districts are geographically separated jurisdictions below the NUTS3-level and above the LAU1-level. Their only purpose is to act as agents in the public administration of functions of the central state and the states, their number/size is based on historical reasons and not on the necessities of an optimal spatial organisation of public policy (f. e. in the health care sector). Districts are without legislative functions.

³Local communities and districts are traditional units in various fields of the official statistics in Austria, nowadays at least partially substituted and complemented by the NUTS-classification. On both levels of aggregation we are therefore confronted and aware of the different "boundary problems" of regional epidemiologic analysis (see Diez Roux 2004, Flowerdey et al. 2008).

the population census (1971, 1981, 1991 and 2001) to calculate the corresponding SMR.⁴ To minimize the problems of small numbers (and thus, random variation), mortality rates sorted by age and gender are calculated for longer time periods. At the districts level, for the calculation of the death rates periods of seven years are chosen around the population census years 2001 (1998-2004), 1991 (1988-94), 1981 (1978-84) as well as a five-year period for the 1971 census (1969-73). At the community level, 16 years for the first period (1969-84) and 17 years for the second period (1988-2004) are used. The difference in the age structure between regions and between different time periods has to be taken into account by age-standardization.⁵

All statistical analyses were weighted by the population in the communities to account for the higher variance of the mortality measures in smaller communities. *Figures 1 and* 2 show that the gender mortality gap is almost normally distributed. It is also obvious that the gender mortality gap decreased from period one to period two (from an average of 528.64 to 384.22). In period two, only 22 communities exhibit a negative gender gap, indicating a mortality advantage for men.

Figure 3 shows the geographical distribution of the gender mortality gap in Austria at the community level (second period 1988-2004). Apparently, there is a considerable variation in gender-specific mortality rates across communities. The very few white spots in the map indicate communities with a negative gender gap, indicating a male mortality advantage.

⁴For each death case the registrar must formulate a death certificate which is to be filled in by the coroner stating the cause of death. This death certificate has to be forwarded to Statistics Austria, where these data are centrally processed and codified. The data files on deaths cover persons listed in the resident population who have died in Austria.

⁵In the case of our data set, direct standardization was used. More precisely, the age-specific death rates were broken down into five-year age group intervals for each gender and region. Subsequently, they were applied to the corresponding age group of the standard population, providing the expected number of deaths for the standard population. By summing these expected numbers of death by age group and dividing them by the total standard population, we obtain the SMR, which allow comparison of mortality rates across regions as well as between periods. The same standard population (WHO-European standard) was employed for all analyzed periods.

At the community level the method of indirect standardization was used. This method weights the agespecific reference rates with the age structure of the investigated population (instead of the WHO standard population) and calculates an expected number of deaths within a community. Subsequently, the SMR is then calculated by the ratio of observed to expected death incidences, as explained above. However, as the study population at the community level was chosen gender-specific (the gender-specific SMR in the community relative to the gender-specific average of the whole population), these SMR are not appropriate to compare mortality rates of males and females. Thus, we calculated ratios of the SMR to the genderspecific average by dividing the SMR by the national average by gender. Thereby we get comparable mortality rates for males and females and are able to calculate the gender mortality gap at the community level as well. For details about the direct standardization method of SMR see Statistik Austria (2007).

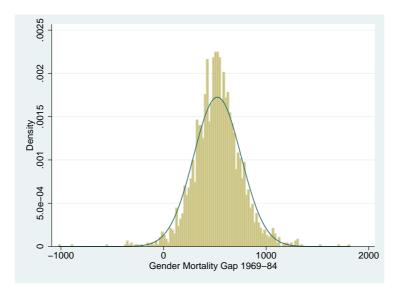


Figure 1: Gender mortality gap 1969-1984 (community level)

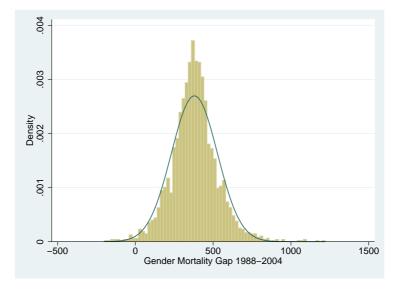


Figure 2: Gender mortality gap 1988-2004 (community level)

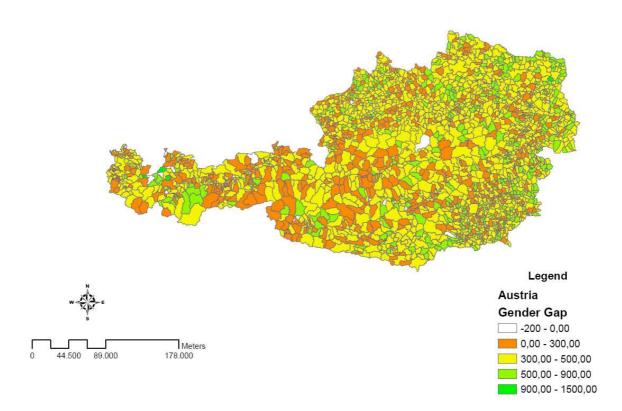


Figure 3: Geographic Distribution: Gender mortality gap 1988-2004 (community level)

3.2 Explanatory Variables

Subsequently, we give an overview of our explanatory variables and how they are calculated.

- Average net income: Unfortunately net income is only available for the second period at the community level (average net income data from tax authorities from 2004). Thus, in the case of communities, we are only able to include it in our cross-sectional estimation of period two. On the district level, we include the gross regional product (GRP) as an explanatory variable.⁶
- Work participation rate: Depending on the estimation, we use overall, genderspecific and/or the gap in the participation rate as explanatory variables.

⁶Due to statistical changes this variable is only available until 1986. For this reason, we used the value of the year 1986 as a proxy for the third period at the district level (mortality data from 1988-94), while we used the corresponding year of the population census for period one (1971) and two (1981). For the fourth period, we used aggregated (individual) net income data at the community level to calculate a corresponding GRP index at the district level. Subsequently, we calculated a (relative) index for each district and each period that is equal to 100 on average. In order to make the values comparable, we then calculated a consistent index based on the fourth period (where it is equal to 100 on average) and multiplied the indices of earlier periods by the Austrian average GDP (as a percentage of 2004 GDP).

• Level of education: To measure the impact of education on mortality, we consider five groups of educational levels. To calculate an average education level, we multiplied the numbers of persons in each group with the corresponding level of education, and divided the sum of the subgroups by the population above 15 years, as indicated in equation (1),

$$Edu = \frac{\sum_{L=1}^{5} POP_L * L}{POP_{15}} \tag{1}$$

where L corresponds to the level of education, POP_L is the population in each subgroup, and POP_{15} is the overall population above 15 years. The factors used for the education level were (1) compulsory school, (2) apprenticeship or secondary education, (3) higher school certificate (general qualification for university entrance), (4) an additional education after this school-leaving certificate (e.g. a polytechnic education or a college) excluding university education, and finally (5) a university degree or equivalent.⁷ Thus, we get an index measuring the average educational level, (theoretically) ranging from 1 to 5 within regions where increasing values indicate a higher level of education, respectively. Subsequently, the same method was applied to gender-specific educational levels.

• *Education heterogeneity:* To measure inequality in socioeconomic variables within a community or district, we calculated the standard deviation of the educational level, corrected for the average level of education in each region. More precisely, we calculated the education heterogeneity variable by

$$H_{edu} = \frac{\sqrt{\sum_{L=1}^{5} (L-\mu)^2 * s_L}}{\mu}$$
(2)

where L corresponds to the educational level (ranging from 1 to 5), μ is the average educational level within the community, and s_L is the share of the subgroup (by educational level) in the population older than 15. At the district level we use a slightly different methodology, as we use the mean of the above calculated education heterogeneity across communities as an explanatory variable (as we think that districts are too heterogeneous to apply the same methodology as explained for the community level).

• Population origins: As we are able to distinguish between the share of immigrants

⁷As the Austrian education system differs quite strongly from other countries, we also included in this "highest" level of education the degrees for primary and secondary school teachers and similar educations which formally do not belong to university degrees in Austria, but would yield a bachelor's degree according to international standards.

from Turkey or former Yugoslavia, and other foreigners, we have to differentiate. While we expect that mortality will be lower the higher the overall share of foreigners, the effect of immigrants from Turkey and Yugoslavia is not clear. This is mainly due to their traditional employment status, as most of them (or their ancestors) came into the country because there was a lack of unskilled workers in a fast growing economy of the 1960s and 1970s.

- Social and familial attachments: To investigate the effects of different familial networks, we considered the following variables from the census, namely
 - the average number of people living in a household,
 - the share of one-person households,
 - the share of households comprising a couple with children,
 - the share of households comprising a couple without children, where the woman is 40 or older,
 - the share of single-households with children,
 - the average number of children per family,
 - the average birth rate per woman, age-standardized,
 - the share of divorced women, in percent of the ever married, and
 - the share of female singles, age 40-59.

As expected, we observe a high correlation between those dimensions (see Table 6 in the Appendix). Thus, a principal component analysis seems to be appropriate to combine the various characteristics into one single variable. As we included nine variables in our analysis, and the eigenvalue of the first factor amounts to 6.29, the resulting factor explains approximately 70% of the total variance. Factor loadings are reported in Table 7 in the Appendix. Average household size, couples with children, the average number of children per family and the age-standardized number of births per woman are negatively correlated with the factor, while the remaining variables mentioned above influence the factor in the reverse direction (one-person households, couples without children, single with children, the share of divorced women and the share female singles in the age between 40 and 59). To sum up, traditional family structures including a couple with children or more people living in a household exercise a negative influence on the factor. On the contrary, one-person households, couples without children, singles with children and a higher share of divorced or single women increase the resulting factor. By reversing the factor (multiplying it by -1) we are able to interpret the resulting variable as "Social and familial attachments", with increasing values of the factor indicating a higher level of social attachments and familial solidarity, respectively.

On the districts level, we apply the same method to calculate our measure for social and familial attachments within a region. While the factor analysis yield qualitatively the same result as shown on the community level (not shown), we only used eight variables, as the average birth rate per woman was not available for the first period at the district level (1971). Despite of that minor difference between the two geographical levels in terms of calculation of the variable we do not expect any difference in terms of interpretation, as we try to measure a single dimension of social and familial attachments in both cases.

Summary statistics both of our dependent as well as independent variables are reported in Table 1. Means and standard deviations are weighted by the community size (population). Overall, a considerable gender gap in mortality is observable, although there are also a few communities with a "negative" gender gap, indicating a male mortality advantage. As expected, the gender mortality gap decreases from period one to two, as male mortality rates are decreasing more quickly than female mortality rates. Furthermore, we observe a considerable deterioration in terms of social and familial attachments from period one to two, as well as an increasing share of foreigners. The increase in the overall participation rate is (almost only) due to the sharp increase in female participation rates (increasing from 34.2% to 42.5%). Accordingly, the gender gap in participation rates decreases from 22.5% to 14.3% in period two. In terms of education, we observe an increasing level of average education, while female education is increasing more quickly, and thus, the gender gap in education decreases. Interestingly, the heterogeneity in terms of education (as a measure of social status) increases (slightly) over time. In total, as the variables differ considerably between communities, our investigation of socioeconomic determinants of mortality rates by using aggregated data should give interesting results.

4 Empirical Results

We start our multivariate analysis with a weighted regression of the gender gap in mortality on the above presented socioeconomic variables.

Weighted regression results for the gender mortality gap both at the local community and district level are shown in *Table 2*. The first three columns report regression results at the local community level, whereas the first column reports a pooled regression model (including a time dummy for period two), followed by separated cross-sectional

	M	ean	Std.	Dev.	Μ	in.	Max.		
	Period 1	Period 2	Period 1	Period 2	Period 1	Period 2	Period 1	Period 2	
Gender mortality gap	528.638	384.225	162.067	98.544	-1023.08	-199.938	1814.170	1204.319	
Standardized mortality, males	1398.147	954.757	185.770	131.533	501.927	288.993	2875.495	2158.581	
Standardized mortality, females	869.508	570.532	137.555	94.030	402.022	71.325	2508.721	1576.902	
Net income		18043.227		2060.918		6981.000		28236.000	
Social & familial attachments	0.366	-0.344	0.978	0.893	-1.583	-2.130	3.340	2.439	
Foreigners, others	1.398	3.253	1.489	2.368	0.000	0.000	36.131	45.600	
Foreigners, Turkey & Yugoslavia	2.459	5.595	2.881	4.666	0.000	0.000	18.919	25.900	
Participation rate, share	44.939	49.512	2.566	1.812	29.800	38.000	57.800	63.300	
Male participation rate, share	56.746	56.861	2.142	1.508	30.900	31.700	66.300	69.500	
Female participation rate, share	34.244	42.546	4.673	3.074	14.500	21.000	51.400	56.100	
Participation rate, gender gap	22.502	14.316	5.262	3.165	4.200	-5.000	44.000	40.400	
Education, average level	1.676	2.040	0.228	0.239	1.096	1.329	2.516	2.942	
Male education, average level	1.862	2.162	0.276	0.249	1.138	1.324	2.947	3.152	
Female education, average level	1.518	1.927	0.202	0.239	1.000	1.311	2.210	2.761	
Education, gender gap	0.344	0.235	0.093	0.064	-0.037	-0.337	0.743	0.578	
Education, heterogeneity	0.526	0.543	0.042	0.032	0.270	0.329	0.640	0.630	

Table 1: Summary statistics (community level)

Notes: Shares are given in percent. Gender gap variables were calculated as the difference between *male* and the *female* levels of the variable. Means and standard deviations are weighted by population. The values reported correspond to the population census 1981 (period 1) and 2001 (period 2), respectively.

estimations of the two periods. Columns four and five regress the gender mortality gap on socioeconomic determinants at the districts level, while column four (five) reports a fixed-effects (pooled) model. The female mortality advantage decreases with increasing social and familial attachments, a higher educational level and a higher share of foreigners (both from Turkey and Yugoslavia and other countries). Interestingly, the negative effect of foreigners applies to both groups included. This is interesting, as it shows that the effect of foreigners on mortality rates is driven by both groups included, and even people from Turkey and Yugoslavia, who typically came to the country as unskilled workers, exhibit the above mentioned positive selection effect of immigration on mortality. Moreover, the gender gap in education shows significantly positive coefficients, indicating an increasing mortality gap with higher differences in education between men and women. The influence of work force participation rates appears less significant in our estimations. While both the participation rate as well as the corresponding gender gap influence the gender gap in mortality negatively in the cross-section estimation for period two, both variables appear insignificant in the remaining estimations. This might be due to the high negative correlation between these labor market variables ($\rho = -0.504$), leading to collinearity, and thus, to non-significant coefficients. The influence of education heterogeneity appears ambiguous in our estimations, while a higher net income, as expected, reduces the gender mortality gap. The time dummy for period two shows a negative coefficient, confirming the result that the gender gap in mortality decreased over time.

By reporting standardized beta-coefficients in *Table 2*, we are able to compare the relative importance of various variables in our estimation, as the included explanatory variables

Regional Level	*	Sults - Gende Communities	U	Districts				
Method	Pooled	CS (t=1)	CS (t=2)	FE	Pooled			
Income / Gross regional product			-0.006***	-1.451***	-1.348***			
			(-3.291)	(-3.640)	(-3.995)			
			-0.127***	-0.326***	-0.303***			
Social & familial attachments	-80.864***	-102.326***	-55.317***	17.095	68.009***			
	(-20.473)	(-14.164)	(-12.188)	(0.867)	(7.630)			
	-0.546***	-0.497***	-0.505***	0.163	0.650***			
Foreigners, others	-2.354**	-4.746*	-1.777	-27.544***	-11.169***			
Ċ,	(-1.981)	(-1.924)	(-1.409)	(-5.606)	(-3.131)			
	-0.036**	-0.043*	-0.043	-0.448***	-0.182***			
Foreigners, Turkey & Yugoslavia	-2.277***	-4.593***	-0.746	0.551	-1.813			
	(-3.526)	(-3.403)	(-1.125)	(0.199)	(-1.148)			
	-0.067***	-0.073***	-0.035	0.019	-0.063			
Participation rate, share	-0.221	-1.715	-3.494**	12.147***	11.376***			
	(-0.196)	(-0.916)	(-2.423)	(3.340)	(4.778)			
	-0.005	-0.028	-0.064**	0.393***	0.368***			
Participation rate, gender gap	-0.369	-1.035	-3.276***	3.000*	1.553			
	(-0.490)	(-0.884)	(-3.124)	(1.706)	(0.996)			
	-0.015	-0.03	-0.105***	0.162^{*}	0.084			
Education, average level	-281.022***	-538.413***	-163.585***	15.094	-184.420***			
	(-20.465)	(-15.941)	(-8.652)	(0.170)	(-4.699)			
	-0.577***	-0.654***	-0.396***	0.043	-0.521***			
Education, gender gap	141.733***	431.855***	61.533^{*}	-149.941	236.249***			
	(5.014)	(8.343)	(1.851)	(-1.507)	(3.694)			
	0.083***	0.226***	0.040*	-0.112	0.177***			
Education, heterogeneity	-135.625**	79.150	22.012	439.324*	107.827			
	(-2.026)	(0.724)	(0.261)	(1.847)	(0.843)			
	-0.035**	0.021	0.007	0.175^{*}	0.043			
Period	-84.928***			-74.833**	-55.952***			
	(-11.474)			(-2.527)	(-7.064)			
	-0.284***			-0.767**	-0.573***			
Constant	1168.024***	1395.420***	1005.863***	-16.370	373.881*			
	(14.534)	(10.531)	(10.213)	(-0.064)	(2.423)			
N	4739	2358	2377	449	449			
R^2	0.327	0.158	0.147	0.804	0.664			

Table 2: Empirical Results - Gender Mortality Gap	Table 2:	Empirical	Results -	Gender	Mortality	Gap
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Notes: The first value reports regression coefficients, t-statistics are reported in parentheses. The third value corresponds to standardized beta coefficients. *, **, *** denote 10%, 5% and 1% significance levels. Regressions are weighted by community/district size (population). CS corresponds to cross section estimations, period in parenthesis. The *Pooled* Model includes all observations from both periods (including *Period* time fixed effects), while *FE* corresponds to a fixed effects model at the district level.

have been standardized to variances of 1. According to this, social attachments and the educational level exercise by far the strongest (negative) influence on the gender mortality gap, which is confirmed by all our estimations. The positive effect of the gender gap in education is also fairly large. Remarkably, this implies that a larger gender gap in education leads to an increase in mortality differentials, and thus, that a convergence in educational levels between men and women leads to a decrease in the mortality differential.

For robustness checks, we applied the same model to our data at the district level, where we have a short panel of four time periods. As the random effects model is rejected by a hausman test, we report both a panel fixed effects as well as a pooled model. Although we are aware of the fact that certain structural breaks between the periods might lead to inconsistent results in our pooled model, we nevertheless report the estimations for the sake of completeness.

We confirm the decreasing impact of income (measured as gross regional product) on the gender mortality gap at the district level. Moreover, the decreasing effect of foreigners is also confirmed (even though non-significant for foreigners from Turkey and Yugoslavia), similarly to the decreasing effect of the educational level (insignificant in the fixed effects model). Furthermore, we find (weak) evidence for an increasing effect of the gender gap in education as well as the participation rate and the corresponding gender difference in labor participation. Thus, this indicates that the current increase in gender equality in European societies is likely to lead to a decrease in the gender mortality gap, although the effects of a gender equalization in terms of participation rates yield ambiguous results in our estimations. Surprisingly, the variable for social and familial attachments appears non-significant in our fixed-effects estimation (and even positively significant in the pooled model). We conclude that social structures might only play a role in smaller communities, while the heterogeneity at the district level (including both rural and urban regions in most of the cases) is likely to lead to this surprising result.⁸

⁸ Table 8 (Appendix) reports the cross-section estimations at the districts level for the observed four periods. While most of our explanatory variables appear non-significant (most likely due to the large heterogeneity of the observed entities), we nevertheless confirm some of our main findings with respect to the gender mortality gap. The mortality differential is significantly negatively linked to the average educational level, while a higher gender gap in education (and thus, a higher status difference between men and women) lead to an increase of the gender mortality gap. Once again, this suggests that the advancement of female educational levels leads to a decrease of the female mortality advantage. Thus, the equalization of life styles between men and women, which have been proposed to result in unhealty and risky behaviour among women, seems to dominate the effect of the increase in the socioeconomic status among females, as stated in our literature review.

Dependent variable		N	Iale Mortalit				Fe	male Mortal		
Regional Level		Communities			Districts		Communities	Districts		
Method	Pooled	CS(t=1)	CS (t=2)	FE	Pooled	Pooled	CS(t=1)	CS (t=2)	FE	Pooled
Income / Gross regional product		. ,	-0.002	-1.111**	-1.897***		. ,	0.005***	0.257	-0.063
			(-1.019)	(-2.243)	(-3.982)			(2.890)	(0.619)	(-0.170)
			-0.039	-0.103**	-0.176***			0.106***	0.035	-0.009
Social & familial attachments	-107.736***	-109.267***	-89.382***	-45.118*	72.213***	-25.601***	-2.386	-35.492***	-55.529***	12.600
	(-22.669)	(-13.301)	(-14.812)	(-1.753)	(5.548)	(-7.175)	(-0.399)	(-7.898)	(-2.594)	(1.283)
	-0.399***	-0.463***	-0.612***	-0.178*	0.285***	-0.137***	-0.014	-0.340***	-0.322***	0.073
Foreigners, others	-9.888***	-22.199***	-8.974***	-10.402*	-18.140***	-3.224***	-10.808***	-4.471***	15.796***	-0.708
	(-7.280)	(-8.074)	(-5.539)	(-1.767)	(-3.708)	(-2.967)	(-4.955)	(-3.561)	(3.184)	(-0.184)
	-0.084***	-0.177***	-0.162***	-0.070*	-0.122***	-0.039***	-0.117***	-0.113***	0.156^{***}	-0.007
Foreigners, Turkey & Yugoslavia	-5.202***	-10.743***	-3.594***	8.816**	-2.383	-2.242***	-6.325***	-2.625***	11.473***	-1.403
	(-6.737)	(-7.066)	(-4.095)	(2.400)	(-1.024)	(-3.858)	(-5.312)	(-4.137)	(4.316)	(-0.822)
	-0.084***	-0.150***	-0.128***	0.126**	-0.034	-0.052***	-0.119***	-0.130***	0.241^{***}	-0.029
Education, heterogeneity	-296.232***	-217.29*	35.301	-21.999	-504.046***	-256.187***	-101.269	-27.869	-327.926	-718.974***
	(-3.714)	(-1.761)	(0.316)	(-0.083)	(-2.719)	(-4.243)	(-1.053)	(-0.335)	(-1.353)	(-5.668)
	-0.042***	-0.051*	0.009	-0.004	-0.083***	-0.052***	-0.032	-0.009	-0.079	-0.174***
Period	-422.539***			-157.262***	-218.895***	-293.921***			-135.347***	-145.163**
	(-55.398)			(-4.525)	(-23.308)	(-45.590)			(-4.351)	(-20.065)
	-0.775***			-0.665***	-0.925***	-0.777***			-0.841***	-0.902***
Male participation rate, share	-1.920	-9.912***	-2.254	1.780	-0.311	-1.006	-10.963***	2.184	-7.317**	-7.006**
	(-1.333)	(-4.478)	(-1.085)	(0.507)	(-0.084)	(-0.903)	(-6.292)	(1.395)	(-2.552)	(-2.536)
	-0.013	-0.116***	-0.026	0.012	-0.002	-0.010	-0.173***	0.035	-0.075**	-0.071**
Female participation rate, share	1.856***	1.612^{*}	3.373**	3.435	7.982***	2.396***	2.331***	2.961***	1.590	3.611^{***}
	(2.733)	(1.867)	(2.909)	(1.175)	(4.695)	(4.547)	(3.452)	(3.393)	(0.663)	(2.865)
	0.041***	0.038^{*}	0.079**	0.079	0.183^{***}	0.077***	0.074***	0.097***	0.054	0.122***
Male education, average level	-287.720***	-382.084***	-208.701***	-241.452**	-82.660*					
	(-19.521) -0.324***	(-12.617) -0.482***	(-8.603) -0.395***	(-2.393) -0.287**	(-1.928) -0.098*					
	-0.024	-0.402	-0.335	-0.201	-0.050					
Female education, average level						-75.794***	-147.673***	-101.741***	-45.864	-17.489
						(-5.954)	(-4.964)	(-5.698)	(-0.485)	(-0.419)
						-0.127***	-0.189***	-0.259***	-0.079	-0.03
Constant	2625.989***	2820.578***	1425.745***	1903.622***	2070.761***	1410.374***	1713.218***	457.689***	1573.201***	1767.093**
	(27.113)	(19.173)	(10.940)	(5.741)	(9.149)	(18.897)	(14.721)	(4.675)	(6.024)	(10.441)
N	4739	2358	2377	449	449	4739	2358	2377	449	449
R^2	0.707	0.173	0.148	0.953	0.874	0.636	0.075	0.057	0.932	0.849

Table 3: Empirical Results - Male and Female Mortality Rates

Notes: The first value reports regression coefficients, t-statistics are reported in parentheses. The third value corresponds to standardized beta coefficients. *, **, *** denote 10%, 5% and 1% significance levels. Regressions are weighted by community/district size (population). CS corresponds to cross section estimations, period in parenthesis. The *Pooled* Model includes all observations from both periods (including *Period* time fixed effects), while *FE* corresponds to a fixed effects model at the district level.

Male and Female Mortality Rates

Table 3 reports the sensitivity of male and female mortality rates on our socioeconomic variables. The first five columns report mortality rates for males, whereas the following five columns report the regressions for female mortality rates. In both cases, results are reported at the community (pooled model and cross-section regressions for both periods) as well as the district level (including a fixed-effects and a pooled model). To begin with, the intercept for male mortality is always higher than for female mortality (both at the community and district level). This is caused by the overall female mortality advantage. Both for males and females, mortality is ceteris paribus lower in regions with stronger social and familial attachments, a higher share of foreigners and a higher level of education. As expected, in all cases the influence is much higher on male mortality rates compared to females (as indicated by the standardized beta coefficients in Table 3). The results for social and familial attachments and foreigners are rather mixed at the district level, indicating the above mentioned heterogeneity of districts, and thus, that the influence of these variables are restricted to smaller regions and communities, respectively. Interestingly, although we only included net income in our cross-section estimation for period two (due to data restrictions), the coefficient appears negative, but non-significant for male mortality, while it is even significantly positive for females, indicating an increase of female mortality with increasing income. Regarding the district level, we get similar results. In this case, a higher income (gross regional product) lowers male mortality, but does not have any (significant) influence on female mortality. This not only confirms the proposed (partly) negative effect of an increase in the socioeconomic status for women (because of unhealthy life styles and risky behaviour due to the equalization of gender roles), but also the lower influence of social and economic variables on female mortality in general. While the results for education heterogeneity are quite mixed at both geographical levels, the influence of workforce participation rates seems quite interesting. While a higher male participation rate decreases both male and female mortality rates (or appears insignificant), an increasing female participation rate is linked to higher mortality rates, both for males and females. This finding seems to be one of the most robust in all our estimations (see *Table 3*). As explained above, the impact of labor participation is not clear from a theoretical perspective. While a higher participation rate usually corresponds to higher income and educational levels, higher levels of labor force participation might also lead to less time investments in health as well as weaker social attachments. While the conventional explanation proposed that excess male mortality is due to greater male labor force participation is also not supported by earlier empirical studies, our results rather indicate that higher levels in female participation rates increase male (and female) mortality rates. Moreover, when taking into account the zero-order correlations, we also know that higher participation rates (particularly among females) are also linked to lower social attachments and familial solidarity, which would also lower life expectancy or increase mortality, respectively.

Furthermore, we are also able to confirm our hypothesis that male mortality is more strongly affected by differences in socioeconomic variables, as we observe a much higher effect of social attachments, education and net income on male than on female mortality levels. This is also supported by the fact that the goodness of fit is much higher for males, indicating that we are able to explain a higher share of the variance in male than in female mortality levels by means of our explanatory variables. Overall, the larger coefficients thus indicate a stronger sensitivity of male mortality to social and economic conditions, and the higher goodness-of-fit values confirm a higher explanatory power for the male than for the female mortality rates.

Blinder-Oaxaca Decomposition

To improve our knowledge of the sensitivity of mortality rates by gender we applied the Blinder-Oaxaca decomposition (Blinder 1973; Oaxaca 1973) to decompose the mean differences in mortality rates in a counterfactual manner. This method divides the mean differential between two groups into a part that is "explained" by group differences, in our case by differences in socioeconomic factors, and a residual part that cannot be accounted for by differences in mortality determinants (Greene 2002, p. 55; Jann 2008). In our case the "unexplained" part can be interpreted as a measure for the influence of other factors (i.e. biological characteristics), but it also subsumes the effects of group differences in unobserved socioeconomic predictors. To implement the Blinder-Oaxaca decomposition we have to reshape our data set in the following way. Each community/district now includes two observations, one for male and one for female mortality. Additionally, we add the gender-specific variables to the observations (participation rate, educational level). Subsequently, we compare the predictions for male and female mortality to decompose the differential into an "explained" and an "unexplained" part. *Table 4* and *Table 5* present the Blinder-Oaxaca decomposition for the community and the district level, respectively.

The first component is the part of the outcome differential that is "explained" by group differences in the explanatory variables (the "quantity effect") and the second summand is the "unexplained" part. While the unexplained part, once again, can be caused either by gender differences in biological-genetic conditions, it also captures the potential effects of differences in unobserved variables. Nevertheless, the significant explained part (both at the community and district level) of the differences also shows the higher sensitivity of

SMR	Coef.	Std. Err.	p-value
Differential			
Prediction female mortality	698.656	2.720	0.000
Prediction male mortality	1144.768	3.921	0.000
Difference	-446.112	4.772	0.000
Decomposition			
Explained	-20.844	10.120	0.039
Unexplained	-425.268	10.051	0.000

Table 4: Blinder-Oaxaca: Two-fold Decomposition (community)

Notes: Decomposition is formulated from the viewpoint of male mortality rates. Thus, the group differences in the predictors are weighted by the coefficients of male mortality rates.

Table 5. Diffider-Gazaca. Two-fold Decomposition (districts)										
SMR	Coef.	Std. Err.	p-value							
Differential										
Prediction <i>female mortality</i>	710.761	8.286	0.000							
Prediction male mortality	1159.629	12.247	0.000							
Difference	-448.867	14.786	0.000							
Decomposition										
Explained	-52.332	28.378	0.065							
Unexplained	-396.535	26.900	0.000							

Table 5: Blinder-Oaxaca: Two-fold Decomposition (districts)

Notes: Decomposition is formulated from the viewpoint of male mortality rates. Thus, the group differences in the predictors are weighted by the coefficients of male mortality rates.

male mortality rates on socioeconomic conditions as compared to females. Although the unexplained part is larger in comparison, the explained part, mainly due to differences in the coefficients, is also of considerable magnitude. This is also confirmed by the three-fold Blinder-Oaxaca decomposition (not shown), which distinguishes between an *endowment effect* (differences due to group differences in the predictors), a *coefficient effect* (differences in the coefficients of the explanatory variables) and an *interaction effect* (accounting for the fact that differences in endowments and coefficients exist simultaneously between the two groups). The decomposition at the district level shows that the significant "explained" part in the mortality differencies in the sensitivity to socioeconomic variables), while both the endowment as well as the interaction effect are non-significant. At the local community level, however, all three parts appear significant in our estimation. According to this, based on their endowments, males should have lower mortality than females (due to higher education level etc.), but the negative coefficient effect leads to higher mortality rates among men, and thus, to a negative "explained" part in *Tables 4 and 5*.

5 Discussion & Conclusion

This study focused on the determinants of mortality differentials between males and females in Austria by investigating data both at the community and district level. As shown in our descriptive statistics, communities and districts exhibit considerable variations in our explanatory variables, such as average net income, social and familial attachments, education, participation rates etc. We were able to give empirical support to the assumption that there is a considerable covariation between mortality and socioeconomic conditions, even when analysing data at an aggregated level. We are able to conclude that

- mortality rates are lower in communities/districts with higher socioeconomic status and income, both for men and women (including net income, the gross regional product and the educational level),
- mortality rates are lower in communities/districts with stronger social networks, both for men and women (including a variable "social and familial attachments" that included a principal component model of household structures, birth rates, children per family etc.),
- mortality rates are lower in communities/districts with a higher share of immigrants,
- men show a stronger sensitivity than women to environmental conditions, implying that the male mortality disadvantage decreases with improving socioeconomic conditions, stronger social networks and a higher share of immigrants, and
- the influence of socioeconomic variables on the gender mortality gap also depends on the corresponding 'gender gaps' in these variables, particularly in the case of the educational level, where a lower gender gap in education (indicating a higher educational level among females) leads to a lower female mortality advantage.

The effect of converging participation rates among males and females is not entirely clear in our empirical estimations. Both the participation rate as well as the participation gap between genders do not show consistent results that could be interpreted in one or the other direction. Thus, regarding participation rates, the (negative) effect of a higher socioeconomic status among women (as indicated by higher female employment rates) seems to be cancelled out by the adoption of unhealthy life styles of women (smoking, alcohol etc.) that can probably be attributed to the equalization of gender roles in modern societies. On the contrary, the equalization of the education level between genders shows a clear picture: The narrowing educational gap between men and women decreases the gender gap in mortality. Thus, the proposition by Kalben (2002) that the gender mortality gap would be even wider if men and women had the same social and economic characteristics, cannot be confirmed in our study. Regarding our remaining hypothesis, that mortality rates will be lower in communities/districts with a more homogenous population, our data cannot confirm this proposition (Wilkinson 2006, etc.).

In a nutshell, we could find empirical support in our data for five of our six hypotheses derived from the literature. We find that although mortality rates are affected by similar factors, the way and intensity of the effect differ considerably by gender. In particular, as male mortality declines more quickly with improving conditions, this implies a convergence of the gender gap in mortality as the conditions affecting mortality improve. However, we also observed a negative correlation between net income and higher educational levels with social attachments within a community/district. That is, as people get better educated and therefore, earn more money, the time restriction might lead to weaker social networks and thus, to a break-up of traditional family structures. Hence, the overall effect is ambiguous, as higher levels of income lower mortality rates, while lower social attachments clearly lead to higher mortality rates, as shown in our results. Contrary to Anson (2003), who could not confirm his hypothesis of a narrowing gender gap as the social status of genders converges, we were able to confirm this assumption, at least for the gender gap in the educational level. This finding is confirmed by the study done by Backhans et al. (2007), who find that increasing gender equality in the society leads to a convergence in health outcomes. Interestingly, we could also confirm their finding that increasing gender equality was generally correlated with poorer health for both men and women, as a higher female participation rate leads to higher mortality rates both for men and women. This result is somehow contradictive to the findings of Waldron (2000), who suggested that "trends in women's labour force participation have had little effect on trends in gender differences in health-related behaviour or mortality" (p. 174). On the contrary, the results of Leung et al. (2004) confirm our hypothesis, as they emphasize the importance of the narrowing gender pay gap, which leads to an increasing female employment rate, and thus, to reduced time investments in health by women. Simultaneously, men are able to increase both time and goods investment in health, leading to an overall smaller gender gap in mortality. Of course, this phenomenon is strengthened by the indirect effects of reduced gender inequalities in societies through the adoption of unhealthy lifestyles by women (smoking etc.). The decreasing effect of immigrants on aggregated mortality rates fits to the findings of individual-level studies, as done by Landale et al. (2000).

For robustness purposes, we also tried a slightly different specification by linking the mortality rates to lagged explanatory variables. More precisely, we linked the mortality rates of period two (one) at the community level to the population census from 1991 (1971). As expected, all main findings stated above were also robust in this specification

(not shown). Additionally, we also ran Seemingly Unrelated Regressions (Felmlee and Hargens 1988) to account for the fact that we are regressing on multiple outcomes from the same set of cases (not shown). Once again, our findings from the baseline specifications are confirmed. The mixed evidence regarding our education heterogeneity variables might be due to considerable correlations with the educational level, or due to issues in terms of calculating the variable, as we are not sure whether the (arbitrary) size of communities and districts is able to catch our intended concept of heterogeneity.

Interestingly, the influence of socioeconomic variables on mortality rates seems to decline over time, as indicated by decreasing values of the R^2 in our (cross-sectional) regressions in recent periods. This impressions holds true both for the community and the district level, and is also confirmed for the gender mortality gap as well as male and female mortality rates. This finding might indicate decreasing returns for improving socioeconomic conditions due to the shape of the underlying health production function.

Although our study gave interesting insights into the socioeconomic determinants of gender-specific mortality rates, it comes nonetheless with some methodological limitations. Our analysis focused on administratively defined political units (communities and districts), which might have been chosen arbitrarily. From a methodological point of view, we took account of this problem by weighting our regressions by the relative size of the community/district, as well as by applying our analysis at two different geographical levels. Nevertheless the question about the "right" level of aggregation still remains. Moreover, our findings do not give any information about individual mortality risks in a given community. Our study rather gives information about general social and economic conditions and their effect on mortality. However, although death is an individual phenomenon, we also have to consider that mortality, the pattern of deaths in a given region by age and sex, is both a biological and a social phenomenon. Similar to Gjonca et al. (2005), we have to conclude that the biological reasons for a female mortality advantage is at least not sufficient to explain the gender gap in mortality. As the female mortality advantage is not a universal phenomenon from a historical point of view, as the gender gap in mortality considerably vary over time, we claim that although biological and environmental factors are also relevant, they have to be embedded in a social and economic context. The results from our study show that biological factors might be influenced by socio-economic factors quite differently. Similarly, social circumstances also strongly influence the behaviour of individuals. Following Hummer et al. (1998), we therefore have to conclude that mortality differences are a result of a "socially influenced biological process", and that a mere biological approach to explain this conundrum is insufficient to encompass a satisfactory explanation. Instead, the social, economic and environmental circumstances have to be taken into account for a comprehensive analysis.

6 References

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7 Appendix

Table 6: Socia	l Atta	chmer	ts: Co	orrelat	tions				
Variable	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
(1) Average household size	1.000								
(2) One-person households, share	-0.881	1.000							
(3) Couple with children, share	0.769	-0.755	1.000						
(4) Couple without children, woman age $40+$, share	-0.730	0.665	-0.861	1.000					
(5) Single with children, share	-0.001	0.139	-0.289	-0.159	1.000				
(6) Average number of children per family	0.501	-0.365	0.425	-0.504	0.213	1.000			
(7) Birth per woman, age-standardized	0.757	-0.654	0.681	-0.646	0.048	0.653	1.000		
(8) Divorced women, share	-0.677	0.683	-0.733	0.508	0.335	-0.193	-0.603	1.000	
(9) Female singles, age 40-59, share	-0.504	0.504	-0.459	0.297	0.306	0.104	-0.298	0.552	1.000

Table 6: Social Attachments: Correlativ

Notes: Correlation coefficients of variables included in the principal component analysis (community level).

Table 7: Principal Component Factor - So	cial Attachments: Factor loadings
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Variable	Factor	Uniqueness
(1) Average household size	-0.9338	0.1280
(2) One-person households, share	0.9614	0.0757
(3) Couple with children, share	-0.9751	0.0491
(4) Couple without children, woman age 40+, share	0.8088	0.3459
(5) Average number of children per family	-0.3865	0.8506
(6) Birth per woman, age-standardized	-0.8954	0.1983
(7) Divorced women, share	0.9327	0.1301
(8) Single with children, share	0.7127	0.4920
(9) Female singles, age 40-59, share	0.7460	0.4435

Dependent variable	Gender Gap	(() () ()	(3.6	Male SMR	()	(a	(Female SMR	(24. 2	() () () ()	(3.4
Method	(Model 1) CS (t=1)	(Model 2) CS (t=2)	(Model 3) CS (t=3)	(Model 4) CS (t=4)	(Model 5) CS (t=1)	(Model 6) CS (t=2)	(Model 7) CS (t=3)	(Model 8) CS (t=4)	(Model 9) CS (t=1)	(Model 10) CS (t=2)	(Model 11) CS (t=3)	(Model 12 CS (t=4)
Method	C5 (t=1)	0.5 (1=2)	C5 (t=3)	C5 (1=4)	CS (t=1)	CS (t=2)	C5 (t=3)	C5 (t=4)	C5 (t=1)	CS (t=2)	C5 (t=3)	C5 (t=4
Income (gross regional product)	-2.282	-0.129	-0.151	-0.995	-2.947*	-1.388	-0.449	-0.246	0.001	0.147	0.580	0.074
	(-1.41)	(-0.15)	(-0.28)	(-1.50)	(-1.74)	(-0.89)	(-0.63)	(-0.23)	(0.00)	(0.10)	(1.39)	(0.15)
	-0.180	-0.020	-0.043	-0.296	-0.202	-0.125	-0.086	-0.047	0.000	0.016	0.193	0.025
Social & familial attachments	219.023***	99.779***	29.354^{*}	19.663*	207.062***	-13.098	14.722	37.833**	-26.013	-80.954**	0.150	21.480**
	(5.68)	(4.54)	(1.82)	(1.84)	(5.01)	(-0.32)	(0.68)	(2.20)	(-1.12)	(-2.21)	(0.01)	(2.17)
	1.196	1.013	0.459	0.372	0.983	-0.075	0.155	0.461	-0.221	-0.573	0.003	0.462
Foreigners, others	5.341	-0.680	-0.097	1.736	-34.501**	-68.728***	-3.344	0.393	-23.143**	-46.842**	0.714	2.271
	(0.32)	(-0.06)	(-0.02)	(0.46)	(-2.17)	(-3.49)	(-0.44)	(0.07)	(-2.10)	(-2.59)	(0.17)	(0.66)
	0.051	-0.009	-0.003	0.077	-0.287	-0.532	-0.061	0.011	-0.344	-0.445	0.023	0.114
Foreigners, Turkey & Yugoslavia	-5.021	-7.137*	0.730	-1.203	-4.852	-0.943	0.374	-3.153	-7.081	-1.453	-1.635	-1.137
	(-0.60)	(-1.69)	(0.31)	(-0.77)	(-0.59)	(-0.12)	(0.11)	(-1.26)	(-1.31)	(-0.21)	(-0.90)	(-0.82)
	-0.074	-0.239	0.052	-0.109	-0.063	-0.018	0.018	-0.184	-0.163	-0.034	-0.135	-0.117
Participation rate, share	15.864^{*}	-1.268	-3.990	-6.686								
	(1.91)	(-0.18)	(-0.82)	(-1.53)								
	0.349	-0.031	-0.131	-0.210								
Participation rate, gender gap	6.633	-1.879	-7.647**	-14.133***								
	(1.30)	(-0.39)	(-2.17)	(-3.98)								
	0.264	-0.107	-0.580	-0.750								
Education, average level	-884.260***	-601.791***	-263.404***	-163.206***								
	(-4.16)	(-5.50)	(-4.52)	(-3.30)								
	-1.522	-1.596	-1.015	-0.758								
Education, gender gap	435.019^{*}	653.351***	247.970**	190.853^{*}								
	(1.74)	(4.36)	(2.30)	(1.93)								
	0.379	0.658	0.247	0.190								
Education, heterogeneity	612.628	229.223	-165.334	-190.554	-4.055	-1757.010^{***}	-1166.091***	-549.405	-603.909**	-1591.338^{***}	-968.553***	-501.746
	(1.64)	(0.76)	(-0.64)	(-0.90)	(-0.01)	(-3.10)	(-3.20)	(-1.62)	(-2.56)	(-3.19)	(-4.78)	(-2.57)
	0.317	0.104	-0.081	-0.117	-0.002	-0.450	-0.384	-0.217	-0.486	-0.501	-0.552	-0.350
Male participation rate, share					10.411	-38.767**	-24.918***	-18.923**	-5.195	-36.614^{***}	-16.625***	-0.870
					(1.10)	(-2.51)	(-3.28)	(-2.22)	(-0.89)	(-2.78)	(-3.95)	(-0.18)
					0.156	-0.468	-0.561	-0.287	-0.139	-0.543	-0.649	-0.023
Female participation rate, share					1.714	10.168**	15.285***	19.186***	0.697	8.333*	9.333***	10.245**
					(0.64)	(2.06)	(4.93)	(4.80)	(0.41)	(1.96)	(5.45)	(4.45)
					0.056	0.278	0.635	0.656	0.041	0.280	0.672	0.619
Male education, average level					-423.691***	186.578	-205.888***	-226.963***				
					(-2.67)	(1.47)	(-3.35)	(-2.87)				
					-0.805	0.340	-0.586	-0.701				
									149.794	385.095**	-128.373***	-81.141
Female education, average level												
Female education, average level									(1.08) 0.325	(2.30) 0.632	(-2.98) -0.553	
Female education, average level									0.325	(2.30) 0.632	(-2.98) -0.553	(-1.88) -0.431
Female education, average level Constant	862.190	1367.994***	1271.801***	1351.342***	1948.885***	3913.018***	2987.942***	1915.938***	0.325	0.632	-0.553 1932.456***	-0.431 534.313
	862.190 (1.44) 95	1367.994^{***} (2.70) 118	1271.801^{***} (3.39) 118	1351.342*** (4.26) 118	1948.885*** (3.19) 95	3913.018*** (4.01) 118	2987.942*** (5.86) 118	1915.938*** (3.75) 118	0.325	0.632	-0.553	-0.431

Table 8: Empirical Results (district level - cross section)

Notes: The first value reports regression coefficients, t-statistics are reported in parentheses. The third value corresponds to standardized beta coefficients. *, **, *** denote 10%, 5% and 1% significance levels. Regressions are weighted by district size (population).