The Contextual Effects of Violence and Poverty on Cardiometabolic Risk Biomarkers: A Longitudinal Multilevel Study in Urban Municipalities in Mexico

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THE CONTEXTUAL EFFECTS OF VIOLENCE AND POVERTY ON CARDIOMETABOLIC RISK BIOMARKERS: A LONGITUDINAL MULTILEVEL STUDY IN URBAN MUNICIPALITIES IN MEXICO

A dissertation
by

Pablo Gaitán-Rossi

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Dissertation Chairs: Dr. David Takeuchi and Dr. Graciela Teruel

Abstract

Poverty and violence within cities frequently concentrate in the same places and evidence suggests these exposures have deleterious consequences on health. The 2007 homicide increase in both rich and poor Mexican municipalities and the available biomarkers in a public panel study offer a unique opportunity to test each contextual effect in isolation on an innovative health outcome. Using an ecological framework, the main hypothesis of the dissertation is that, in urban environments, exposure to higher levels of contextual violence works as a stressor that wears down the body by increasing the levels of cardiovascular risk. This effect was hypothesized to be independent from poverty but with significant interactions and with heterogeneous effects among subpopulations. Multilevel cross-sectional and longitudinal analyses were conducted treating the data as a natural experiment using the homicide rate increase as treatment. The outcomes were two indices and single biomarkers that reflect cardiovascular risk in three waves of data corresponding to the years 2002, 2006, and 2012. Results showed that three complementary statistical approaches provided evidence indicating that exposure to cumulative violence at the municipality level yielded higher cardiovascular risk when controlling for individual covariates like victimization and household expenditure. The significant threshold for homicide rates was 35 and the differences between exposed and unexposed municipalities was between 1.5%

& 8.3%, while the threshold for *changes* in the homicide rates between 2006 and 2012 was 10, with an effect size of 7%. Poverty and violence were not correlated in Mexico during the homicide rate spike, so the effects were independent. Unexpectedly, they did not show interaction effects: affluent and violent municipalities were the most stressful contexts. These effects were higher in women, in individuals in the two lowest socioeconomic quintiles and had significant impact in cohorts younger than 40 years old. The dissertation expands the ecosocial approach by exploring independent effects that shape multiple stressful contexts. It demonstrates that violence is a public health concern in Mexico that has indirect effects in the whole population, which not only worsens the obesity epidemic, but also demands a new perspective on assessing the burden of violence on everyday life.

To Lila,

For her unconditional love and support;

for making me stronger and happier;

for all our exciting adventures.

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I am so lucky for having this amazing team.

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The poor, the vicious, and the delinquent,

crushed together in an unhealthful and contagious intimacy,

breed in and in, soul and body;

would not show such a persistent and distressing uniformity of vice, crime, and poverty,

unless they were peculiarly fit for the environment in which they are condemned to exist. (Park, 1915, p. 612).

"There are no risks, you can wander at ease,

all these people have criminal records indicating their involvement in violent events,

it is a cleansing amongst them and until now any citizen was involved"–

explains a Secretary of Security in Guanajuato, a Mexican state (Lopez Portillo, 2017, p. 1).

"It really only touches those involved in the world of crime

[…] it's better to get used to it. This is not going to change. None of it."–

ascertains Angela Hernandez, while having ice-cream with her 5-year-old son in one of Mexico's deadliest municipality in Colima, a Mexican state (Ahmed, 2017, p. 1).

If you want to understand why health is distributed the way it is,

you have to understand society.

(Marmot, 2015, p. 7).

Chapter I. Introduction

1.1 Purpose

The dissertation examines the contextual impact of violence and poverty on straining the human body. While past studies have shown that both contexts are linked to health and well-being (Kawachi, Kennedy, & Wilkinson, 1999; Sampson, 2003), some pressing questions remain. First, it is not clear whether environmental violence and poverty have independent associations with health. For instance, Wilson (2012) shows how unemployment, family structure, and poverty cluster together in urban ghettos creating interaction effects. Sampson and colleagues continued his insight by examining the convoluted relationship between concentrated disadvantage and violence (Morenoff, Sampson, & Raudenbush, 2001; Sampson, Raudenbush, & Earls, 1997). Nonetheless, since neighborhood poverty and associated structural factors continue to predict individual´s exposure to violence in multiple U.S.A cities (Graif, Gladfelter, & Matthews, 2014), it has been difficult to study each effect in isolation. Furthermore, it is unclear whether these contexts have distinct consequences (Jencks & Mayer, 1990) or rather they have cumulative and interactive effects (Massey, 1990). On the intricate associations between poverty and violence, Massey asserts that by "exposing residents to the combined influence of these maladies […] their independent effects cannot really be disentangled theoretically or empirically" (2013, p. 693).

Second, the links between poverty, violence and health are not well established. While stress has been implicated as a mediator between social structural factors and health (Evans & Kim, 2010; Matthews, Gallo, & Taylor, 2010), poverty and violence are usually considered as individual experiences (Haushofer & Fehr, 2014), and have not been fully examined at the community level (Massey & Brodmann, 2014). Community contexts are important units of

analysis and should not be considered as individual "traits" (Raudenbush & Sampson, 1999b). They can be studied for their ecological results on biological embodiment, the lived experience that in turn shapes population patterns of health and disease (Krieger, 1999; Krieger, 2011a). Community contexts conceptualized as stressors in interaction with personal emotions and behaviors illuminate key pathways to understand how social influences *get under the skin* (Berkman, Kawachi, & Glymour, 2014).

Third, most of the studies on violence, poverty, and health have used cross-sectional designs (Sharkey & Faber, 2014). While these designs help to identify associations between persons, they are not useful in establishing within-person change and, especially, the patterns of change over time in different populations like longitudinal designs do (Wodtke, Harding, & Elwert, 2011).

Finally, most of these studies have been conducted in the U.S.A and Europe (Sampson, 2012). It is difficult to establish if the link between poverty and violence are universal or pertinent only to high-income country contexts (Sen, 2008). Moreover, the few studies addressing these topics in Mexico examine either the individual (Vilalta, 2013) or the municipality level (Osorio, 2012; Torche & Villarreal, 2014) but rarely test cross-level interactions and longitudinal designs.

The dissertation moves beyond these knowledge gaps by disentangling the longitudinal consequences of contextual poverty and violence on individual health over time in Mexico. The intersection of theories stemming from Social Ecology and Social Epidemiology provide a sound framework to analyze context effects on a wide array of outcomes, especially those related with cardiovascular disease. The study seeks to enhance the understanding of how long-term exposure to stressful contexts is embodied through biosocial processes that decrease individual health.

1.2 Significance

Since 2008 a majority of the world's population were living in cities and it is projected that by 2050 two out of three persons will live in one (UN-HABITAT, 2016). In 1950, only in Latin America, 41% of the population inhabited urban settlements and this proportion increased up to 78% by 2007 (UN-HABITAT, 2016). Following global trends, Mexico experiences an intense process of urbanization. In 1960, one out of four people lived in Metropolitan areas but, by 2010, 63% of the population lived in localities with more than 2,500 people (CONAPO, 2012). Rapid urbanization calls for innovative ecological ways to understand old social problems.

Urbanization crucially shapes social processes such as poverty, violence, and health (UN-HABITAT, 2016). For example, in the U.S.A, during the 70's, poverty became increasingly concentrated within segregated inner-city neighborhoods creating a new type of underclass and thus provoking greater sensibility to economic shocks and an important reduction in well-being, eventually strengthening the reproduction of poverty (Massey, 1990; Wilson, 2012). However, poverty in Latin American middle-income countries is somewhat different. Mexico is the second largest economy in Latin America and the United Nations categorized it as a country with high human development (UN, 2016). Nonetheless, in 2012, 45% of Mexicans lived below the poverty line and 42% of that population inhabited urban localities, where inequality is greatest (CONEVAL, 2014). In spite of the changing face of poverty, official counts do not distinguish between urban and rural poverty and therefore a more precise conceptualization is required to understand its main features (Teruel, 2014).

Both globally and in Mexico, violence tends to concentrate in urban environments when it is operationalized as homicide rates (Leenen & Cervantes-Trejo, 2014; UN-HABITAT, 2016). In Mexico, during the 1990's, political violence was distributed on rural localities in the poorest southern states, but during the last 15 years it moved to urban municipalities, where it is now

mostly caused by firearms and is strongly linked to criminal gangs and drug trafficking (Magaloni & Razu, 2016). In 2005, the national rate of homicides by 100,000 inhabitants was close to 10 but, at the end of 2006, former president Felipe Calderón launched the so-called "War on Drugs" and by 2011 the homicide rate rose to 24 (Leenen & Cervantes-Trejo, 2014). The increment was not equal in other types of crime, like kidnappings; see figure 1. The homicide rate increment was not widespread either, but concentrated in some municipalities. For example, homicide in Ciudad Juárez, reached alarming rates of 108 that justified a comparison with war zones (Osorio, 2015). Regardless of the label, Mexico does not face a traditional war with state actors, but an internal conflict of huge proportions where lines between non-state actors constantly blur and the role of the state is ambiguous at best (Schedler, 2015). The conflict shares characteristics with extremely violent societies (Gerlach, 2015), especially because several types of actors independently engage in violent actions despite state responses and become victims and perpetrators at the same time (Rodriguez Ferreira, 2016).

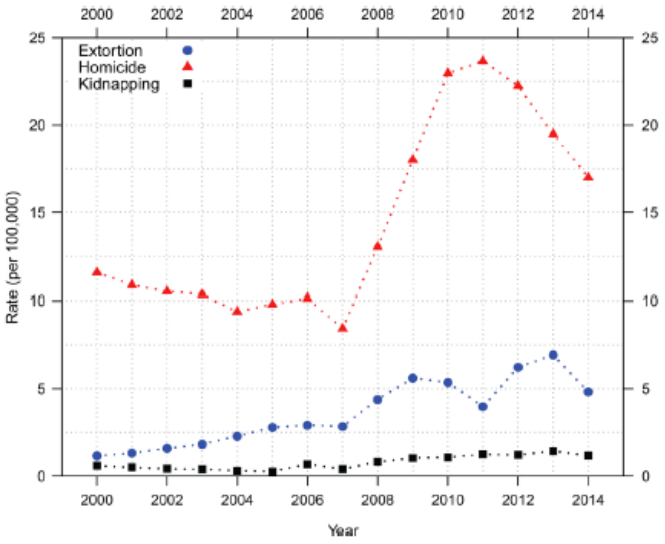


Figure 1. Rates of extortion, homicide and kidnapping for the Mexican population from 2000 to 2014 with data from INEGI and Secretaria de Gobierno. Source: Canudas-Romo, Aburto, Garcia-Guerrero, and Beltran-Sanchez (2016).

There are several reasons why the Mexican violence differs from the one in the USA, particularly in its inextricable association with poverty. Foremost, the recent "War on Drugs" and the increasing militarization of the country created a singular period (Enamorado, López-Calva, Rodríguez-Castelán, & Winkler, 2016) that could be reduced to a tougher domestic drug reduction policy (Osorio, 2015) i.e. homicides committed by traffickers battling to control a market and casualties generated by law-enforcement (Rios, 2012b). Similar explanations attribute the violent increase to external shocks, such as the price of Colombian cocaine (Hope, 2013). But geographical variations in the homicide rates suggest specific social dynamics, such as wins of mayors from the ruling party (Dell, 2015). Even though different sources of violence converge simultaneously, the 2006 to 2012 increase is mostly attributed to organized crime and weak security institutions (Pereyra, 2012; Rodriguez Ferreira, 2016).

The Mexican government has been frequently held responsible for the increase in violence. Mexico has a corrupt and dysfunctional set of law enforcement institutions and practices (Magaloni & Razu, 2016, p. 62). Weak, fragmented, and decentralized institutions contribute to impunity (Rios, 2012a) and not even additional funding to local polices is clearly related with a reduction in the homicide rates (Chaidez, 2014). In the 2016 Global Impunity Index Mexico was in the 58th position out of 59 countries (Le Clercq Ortega & Rodriguez Sanchez, 2016). Even though impunity is a multidimensional problem, in Mexico the poor functioning of the security and justice systems, paired with a severe human rights deficit, are mostly responsible for it. For instance, among the reported crimes only 4.5% receive a sentence; but 93% of crimes are not reported, thus in Mexico less than 1% of crimes committed receive any punishment. In spite of the fact that homicides have to be prosecuted by law, only 27.5% begin the sentencing process (Le Clercq Ortega & Rodriguez Sanchez, 2016). Moreover, a detailed study found over one thousand clandestine graves between 2009 and 2014 and the authors persuasively claim the number is likely underestimating the horror of

the violence crisis in Mexico (IBERO & CMDPDH, 2017). They also highlight that mass graves appeared all along the country, but some states had a higher level of violence and statistical patterns indicate that some municipalities concentrate many of them. The authors conclude that these mass graves aim to generate terror and boast about the impunity in which criminals can act in Mexico (IBERO & CMDPDH, 2017).

It is surprising that few analysts link poverty with violence in Mexico, perhaps because poverty rates remained stable through the same years that homicide rates spiked (CONEVAL, 2014). Nonetheless, there is evidence of the negative impact of drug-related crimes on income growth (Enamorado, López-Calva, & Rodríguez-Castelán, 2014). A longitudinal examination of the homicide rates at the municipality found an inverted U shape with poverty (Osorio, 2012). A similar investigation concluded that, rather than poverty, income inequality accounted for the increase of homicide rates (Enamorado et al., 2016). While the U.S.A suffers from a race-based mass-incarceration closely associated with poverty (Moore & Elkavich, 2008), Mexico has a different context from the US in relation with the root-causes of violence. Therefore, Mexico is an ideal site to test and elucidate long-held assumptions about violence and poverty.

The notorious increase of violence spurred a number of studies in Mexico to assess its consequences. Some investigations focused on the direct impacts of violence, particularly documenting that they were happening mostly in the northern, richer states, and that male youths were dying at a higher rate (Leenen & Cervantes-Trejo, 2014). Other studies did not focus on homicides but on enforced disappearances (HRW, 2013) and individual strategies from lay people to protect themselves from general crime (Vilalta, 2012). An appalling finding is that Mexico's "epidemic of violence" caused life expectancy to stagnate at age 20 between 2005 and 2014 for females and males and also generated that, on average, 40% to 70% of the number of years of the Mexican population are lived with important feelings of vulnerability; "if perceived

vulnerability remains at its 2014 level, the average Mexican adults would be expected to live a large fraction of his/her life with perceived vulnerability of violence" (Canudas-Romo et al., 2016, p. 1).

In addition to the direct impact of violence –measured as victimization rates– researchers found that violence has had several negative consequences on the Mexican's social fabric and well-being (Diaz-Cayeros, Magaloni, Matanock, & Romero, 2011) that range from sleep deprivation (Braakmann, 2012) and worrisome levels of fear of crime (Vilalta, 2013) to forced migration (Rios, 2012a). Notably, from a public health perspective, there have been few attempts to understand the political, economic, and structural causes of violence, as well as its indirect effects on health (Gamlin, 2015).

In spite of the epidemic levels of violence and its documented consequences, evidence suggests that large portions of the population seem indifferent and may be starting to normalize daily violence (Schedler, 2015). Even though fear of crime is also shockingly high in many parts of the country, it is closely associated with personal victimization but not as much with municipality-level homicide rates (Gaitán-Rossi & Shen, 2016). The elevated levels of fear of crime in Mexico follow the victimization paradox in which the most fearful members of the population are in less danger than the least fearful. It is interesting to note that high levels of insecurity decrease the indices of subjective well-being in Mexico (Martinez-Martinez, Vazquez-Rodriguez, Lombe, & Gaitan-Rossi, 2017). Yet, Mexico scores a mean 7.8 in life satisfaction on a 0-10 scale, placing it among the highest levels in the world (INEGI, 2015). The population's apparent indifference to violence may be related to the official discourse that tends to isolate the danger and minimize its consequences. The official mantra in the present and last administrations has been to calm the population by assuring that the "killings are amongst criminals" and thus the innocent can keep on with their lives as usual; as the epigraph on the beginning, an example from

a police authority in Sinaloa illustrates this discourse: “homicides in the municipality occur in focalized areas and among members of criminal gangs, they do not affect the population” (Lopez Portillo, 2017, p. 1). The evidence indicates that the consequences of violence exceed the direct effects measured with victimization rates and subjective measures may not capture the full range of its burden.

Cities influence health and well-being both positively and negatively; for example, on one hand there is easier access to social and health care services but, on the other, there is more exposure to pollution (Galea, Freudenberg, & Vlahov, 2005). The urban environment is of etiological significance for a wide range of health outcomes, but rapid urbanization adds the need to develop supportive community structures (McKenzie, 2008). Although Mexico nearly reached universal health coverage by 2012 (Knaul et al., 2012) official figures report that 22% of the urban population lacked effective access to health services (CONEVAL, 2014). Therefore, important health gaps by socioeconomic status continue: poor places are still associated with poorer health (Juárez-Ramírez & Márquez-Serrano, 2014). As in high-income countries (Diez Roux & Mair, 2010), in Mexico socioeconomic status highly determines living conditions and individual behavior creating important health gradients and a poverty trap (Murayama & Cordera, 2012). Notably, these trends have a geographic element. In spite of the fact that Mexico is regionally converging into better health outcomes, there is a growing health inequality within states (Godinez & Burns, 2012). Associations between place, poverty and health suggest a complex milieu that requires a careful examination of its determinants (Gutiérrez & García-Saisó, 2016).

Evidence shows that progress in life expectancy is accompanied by a stagnant reduction of the social gradient on health; and cardiovascular disease (CVD) is an important contributor to health inequality (Brunner, 2016). Accounting for almost 25% of total deaths, CVD is the main

cause of morbidity and mortality in low and middle-income countries (Kreatsoulas & Anand, 2010). In spite of regional and sociodemographic variations, ischaemic heart disease is also the leading cause of disability-adjusted life-years (DALYs) among high-income countries and in Latin America comes right after violence (G.B.D., 2015b). Along with the epidemiological transition, the mortality risk factors shifted to behavioral and metabolic risks (G.B.D., 2015a). By 2013, the leading global risk factors for DALYs in both sexes combined were high blood pressure, smoking, and high Body Mass Index (BMI). Globally, "high systolic blood pressure accounts for 9.6% of all DALYs, up from 5.6% in 1990, making high systolic blood pressure larger than ischaemic heart disease and three times larger than HIV/AIDS in terms of DALYS" (G.B.D., 2015a, p. 2310). Recent efforts to predict CVD acknowledge that mean risk factor levels substantially differ between populations and over time due to access and quality of health care, and because of differences in environmental, genetic, and psychosocial factors (Hajifathalian et al., 2015).

Mexico is at an advanced stage in the epidemiologic transition where, nationally, 75% of total deaths were from non-communicable diseases (Stevens et al., 2008). The leading causes of death in the country are ischemic disease, diabetes mellitus, and cerebrovascular disease (Stevens et al., 2008). Among men, the main causes of DALYs are violence, ischemic disease, and traffic accidents while in women are diabetes, chronic renal disease, and ischemic disease (Lozano et al., 2013). A feature of Mexico's epidemiological transition is that the highest mortality rates and years with the burden of disease are attributable to high blood glucose, a high BMI, and alcohol-use risk factors (Stevens et al., 2008). In addition to high cholesterol, low fruit and vegetable intake, physical inactivity, and tobacco smoking, all these risk factors jointly account for an estimated 36% of deaths and 16% of disease burden (Stevens et al., 2008).

The previous figures indicate Mexico has a severe problem with CVD risk. Recent studies with the Mexican population of 20 years and above estimate the prevalence of overweight and obesity at 71.3% (39% and 32% in men and women, respectively), which represents a 15% increase in the period from 2000 to 2012, although the increase since 2006 was 2.3% only (Barquera, Campos-Nonato, Hernández-Barrera, Pedroza-Tobías, & Rivera-Dommarco, 2012). Likewise, the prevalence for central adiposity was 74%, representing a yearly increase of 1.3% since the year 2000 (Barquera et al., 2012). Interestingly, urban localities present a higher BMI prevalence but lower values were observed in the low socioeconomic tertile when compared to the medium and high segments. Such prevalence estimates contradict the social gradient on health that has been observed widely across countries (Marmot, 2015). Moreover, the prevalence of hypertension is 31.5% although its levels were stable in the period from 2000 to 2012 (Campos-Nonato et al., 2013). Following a social gradient, hypertension is 19% higher in urban than in the rural localities and 34% higher in the medium socioeconomic tertile when compared with the low one (Campos-Nonato et al., 2013). Therefore, the social gradient on CVD risk factors is not as consistent in Mexico as in other countries.

In sum, the dissertation addresses these research gaps by offering a more nuanced examination of the link between poverty, violence, and health in a context of rapid urbanization. In addition, it presents evidence for the need to consider violence as an additional vulnerability that does not always overlap with poverty. Cardiovascular risk measures are sensitive to stress, so they are appropriate to test the indirect effects of poverty and crime. Exposure to violence disrupts the social gradient of health and thus the dissertation will suggest some of the mechanisms involved by identifying the municipality structure that causes more health damage. Moreover, the singularity of the Mexican context will help advance ecosocial theory and could serve as a cautionary tale against importing policies based on foreign contexts, such as the ones in

the U.S.A. Lastly, the dissertation will constitute a historical record of the indirect but objective consequences on health of one of the deadliest periods of Mexican history.

1.3 Research Questions and Specific Aims

The research questions for the dissertation are summarized as follows:

- What are the indirect effects on health due to the cumulative exposure to contexts characterized by high levels of violence in Mexico?
- Are these effects independent from poverty or do they operate as an interaction?
- Do these contexts have further interactions with individual characteristics?
- Do indices and single biomarkers of cardio vascular disease risk are adequate measures to assess contextual stress in Mexico?

The hypothesis of the dissertation is that, in urban environments, exposure to higher levels of contextual violence –in isolation and as an interaction with poverty– works as a stressor that wears down the body, increasing the levels of cardiovascular risk. The aim of the investigation is to assess the specific impact of violence and poverty beyond subjective perceptions of vulnerability and direct victimization and to show its contextual effect.

The empirical objectives are:

1- To validate individual indices and single biomarkers of cardiometabolic risk in Mexico by comparing its correlations with conceptually associated measures at the individual and municipality level.

2- To examine whether contexts of violence and poverty have an effect –over and above individual SES, personal victimization, and fear of crime– on the cross-sectional and longitudinal association with cardiometabolic risk in Mexican urban municipalities.

3- To test cross-level interactions between context characteristics and individual attributes as means to locate the heterogeneity of effects on cardiometabolic risk in Mexican urban municipalities.

Chapter II. Literature Review

2.1 Context effects

The definition of context effects is deceptively simple; it is the independent effect of social or environmental factors on human thinking, behavior and health, over and above individual characteristics (Oakes, Andrade, Biyoow, & Cowan, 2015). The conceptual history of this idea can be narrated from several disciplines and perspectives. Regardless of the viewpoint, however, it is a story fraught with bold conceptual assertions, methodological obstacles, and heated debates. Sociology has a long tradition theorizing how society influences individual attitudes and behaviors, but it is probably the branch of Social Ecology that actively seeks to empirically evaluate the mechanisms responsible for these forces. Even though examining context effects are more recent in Public Health, the discipline has vigorously implemented them into the fruitful area of Social Epidemiology. Social Epidemiology integrates several approaches ranging from ecosocial theories of biological embedding, to syndemics and the life-course approach. Notably both perspectives are inextricably related to the understanding of the effects of poverty on health and behavior. As the following section aims to demonstrate, these two disciplinary paths interrelate and reinforce themselves in an effort to appraise the fascinating and useful concept of context effects.

2.1.1 Origins in Sociology

The notion of "context effects" is embedded in the origin of Sociology. Durkheim defends that society is more than a collection of individuals and should be studied as a "thing" in itself (Durkheim, 1982). Society, in the form of a collective conscience but originally disguised as religion, makes people stronger (Durkheim, 1965) and its enclosing force as either moral or anomie is capable of shaping intimate decisions such as suicide (Durkheim, 1952). Durkheim considers that the society is as an antecedent entity for the individual whose force surpasses his or her will and, for him, its contextual effects are the extent and multiple ways it regulates individual behavior. His insights endure at the core of sociological thinking, although the theorization of these forces changed when it was paired to the advent of the city; they came to be called meso-level effects (Bronfenbrenner, 1989) or neighborhood effects (Sampson, Morenoff, & Gannon-Rowley, 2002).

A "neighborhood", in this literature, is understood as a spatially delimited segment of a city whose character is defined by its people and infrastructure; it is composed by "a variably interacting population of people and institutions in a common place" (Sampson, 2011, p. 228). Since the first cities, neighborhoods were spatially differentiated on the basis of power, wealth, occupation, religion, and ethnicity and thus created residential segregation (Massey & Brodmann, 2014). In the United States, Robert Park developed these ideas to defend that societies behave as physical and cultural units of inter-dependent like-minded individuals that engage in corporate or collective action (Park, 1921). Even though urban life weakens the influence of traditional institutions –like the family or the church– Park believes that cities are ideal, *natural*, sites to observe these forces. Neighborhoods in particular need to be studied as "moral regions" i.e. geographical localities with sentiments, traditions, and a history of its own, which by a process of segregation and contagion establish moral distances; "little worlds which touch but do not

interpenetrate" (Park, 1915, p. 608). Proximity and neighborly contact are the most elementary forms of association in the organization of city life because they lend a "moral support" for the traits of its members (Park, 1915). Park's ideas are the founding blocks of the ecological theory, which was cemented in the Chicago School of Sociology.

The next milestone for ecological theories, also stemming from the Chicago School of Sociology, is Shaw and McKay's study of "Juvenile Delinquency and Urban Areas" (1942). Their key finding is that economic composition of neighborhoods is negatively associated with delinquency rates (Bursik, 1988). They argue delinquency is not an isolated disadvantage and rather deprived neighborhoods are a cluster of social problems that includes high rates of infant mortality, low birth weight, and physical abuse (Sampson, 2012). Moreover, these clusters of social problems persist over time despite of the movement of its residents, thus asserting that neighborhoods possess enduring features and properties that transcend their temporary inhabitants (Sampson, 2012).

These ideas are further refined when William Julius Wilson acknowledges that, due to the dramatic retreat of the US government from the Great Society programs during the 60s, in the 70s poverty became increasingly concentrated within inner-city neighborhoods creating a new type of underclass (Wilson & Aponte, 1985). In *The Truly Disadvantaged,* he shows how the decline of manufacturing, the suburbanization of blue-collar employment, and the rise of the service sector tended to cluster and interact in urban ghettos generating concentration effects that increased poverty and inequality while weakened familial bonds, eroded informal social controls, and diminished trust in institutions (Wilson, 2012). The "underclass" is characterized for its geographic concentration, its social isolation from the middle class, and the lack of jobs (Small & Newman, 2001). Wilson proves his claims with the widely used measure of concentrated disadvantage, a multidimensional cluster of traits that comprises neighborhoods with a majority

of people living below the poverty line, mostly African American population, female-headed families, with high children density, and an elevated percentage of people living from public assistance. Based on Bronfenbrenner´s ecological developmental perspective (1989), Wilson considers neighborhoods as a transactional setting influencing individual behavior both directly and indirectly and thus embraces a multilevel explanation of how neighborhood structure supports or undermines socialization processes linked to individual-level outcomes (Elliott et al., 1996). Worried about the mechanisms and buffers driving and mitigating the concentration effects, he seeks to assess how the organizational structure and culture of a neighborhood mediates the effect of poverty on individual development in a reciprocal relationship with individual selection effects and generative neighborhood effects (Elliott et al., 1996). He and his colleagues identify that some of the mechanisms that explain the concentration effects of poverty were "limited institutional resources, low levels of family integration and neighborhood cohesion, weak informal social controls, limited consensus on norms and values, and the presence of illegitimate opportunity structures" (Elliott et al., 1996, p. 395). Importantly, in an early example of cross-level interactions, these effects are hypothesized to be stronger in poor neighborhoods than in rich ones:

> Families in affluent neighborhoods may not be as dependent on their physical neighborhood for social support, resources, and informal social supports as are those living in disadvantaged neighborhoods; their functional neighborhood is more likely to transcend the physical boundaries of the neighborhood, whereas in more disadvantaged neighborhoods, the physical and functional neighborhoods are more likely to coincide (Elliott et al., 1996, p. 417)

Douglas Massey (1990) supplements Wilson's thesis by showing that the interaction of rising poverty and high levels of segregation (caused by the housing market discriminatory policies) creates the urban underclass because exogenous economic shocks spread unevenly: "the

greater the segregation, the smaller number of minority neighborhoods absorbing the shock, and the more severe the concentration of poverty" (Massey, 1990, p. 337). Some consequences that accompany poverty are reduced buying power, increased welfare dependence, family disruption, elevated crime rates, household deterioration, elevated mortality rates, and decreased educational quality; a constellation that has profound implications for the well-being of the neighborhood's residents (Massey, 1990). Massey underscores that such conditions of concentrated disadvantage are cumulative and mutually reinforcing and thus create the "structural niche within which a self-perpetuating cycle of minority poverty and deprivation can survive and flourish" (1990, p. 350).

Whether the underclass was increasing because of the departure of skilled-jobs or due to residential segregation, there is little disagreement about the concentration of poverty and eventually both explanations ceased to be considered as mutually exclusive (Small & Newman, 2001).

The notion of cumulative effects suggests that interactions between several types of disadvantage may not only be additive but multiplicative (Massey & Brodmann, 2014). The possibility of interactions is akin to the concept of intersectionality (Watkins-Hayes, 2014) i.e. the double disadvantage hypothesis suggests that adults who hold more than one adversity or stigmatized status may experience worse health outcomes (depression, physical health, and functional limitations) than their singly disadvantaged and privileged counterparts (Grollman, 2014). For example, a recent review concludes that HIV/AIDS is an epidemic of intersectional inequality that is fueled by racial, gender, class, and sexual inequities at the macro, meso, and interpersonal levels that significantly shape the likelihood of exposure to the virus, the access to care, and the daily realities of living with the virus (Watkins-Hayes, 2014).

Following these findings, Sampson, Raudenbush and Earls (1997) are concerned by the fact that poverty and crime usually appear in the same neighborhoods year after year, regardless

of the population's mobility. They believe that the residents' characteristics –such as effective informal mechanisms of control– can mediate crime incidence. For example, they argue that the adult monitoring of children playing or the willingness to intervene and stop youth's acts of truancy constitute important community prevention actions. Rather than police efficacy, they are interested in the communities' abilities to regulate its own antisocial behavior and collectively work to solve their own problems. They claim that a condition for these informal controls is mutual trust and solidarity. In places where the rules are unclear and fear or mistrust pervade the social relationships, informal mechanisms will not appear. Therefore, extrapolating Bandura's concept of individual self-efficacy, they argue that the sum of trust and informal social controls yields a willingness to act for the common good of the community. They are trying to assess a situated neighborhood attribute called collective efficacy that varies when the community faces particular tasks. Their hypothesis is that the "collective efficacy of residents is a critical means by which urban neighborhoods inhibit the occurrence of personal violence, without regard to the demographic composition of the population" (Sampson et al., 1997, p. 919). In the same vein, Bursik argues that their key theoretical advance is the specification of disorganization as "the capacity of a neighborhood to regulate itself through formal and informal processes of social control" (Bursik, 1988, p. 527). Collective efficacy serves as a clear mediating variable between poverty and crime that accounts for meso-level neighborhood effects. Empirically, it was easier to account for variability within poor neighborhoods and show that not all poor places are disorganized. Moreover, with the conceptualization, measurement, and modeling of collective efficacy these authors explicitly open the possibility of *ecometrics* i.e. the adequate assessment of ecological settings (Raudenbush & Sampson, 1999b).

2.1.2 Origins in Public Health

Epidemiologists study the distribution and determinants of states of health in populations (Berkman & Kawachi, 2014) but they have traditionally focused on the characteristics by which healthy individuals differ from sick ones (Ahern, Galea, Hubbard, & Karpati, 2008). Epidemiology's considerable success is based on the case-control and cohort studies aimed at locating risk factors that identify which individuals are more susceptible to disease and, if such factors are indeed causes, the acquired knowledge guides health prevention (Rose, 2001).

However, as Rose brilliantly argues, these methods address the causes of individual cases but not the causes of *population* prevalence and incidence. The traditional approach is quite successful in answering, "Why do some individuals have hypertension?" but traditional epidemiology rarely asked, "Why do some populations have much hypertension, whilst in others it is rare?" (Rose, 2001). The question for the causes of *population* maladies seeks the determinants of population's averages because what distinguishes two groups might not have much to do with individual characteristics but on social influence acting upon a population *as a whole* (Rose, 2001). Therefore, the answers to these questions are a matter of whether exposure varies similarly within and between populations over a period of time (Rose, 2001).

For example, the laboratory ranges used to interpret "normal" levels in blood samples are defined for what is common within a local population. But Rose asks what would happen if, in that population, *everyone* smokes 20 cigarettes a day. Most likely, an experimental, a case-control, or a cohort study with that population would conclude that lung cancer is a genetic disease, not a behavioral one. And the conclusion would be sound in traditional epidemiology because, if everyone is exposed, then the distribution of cases is completely determined by individual susceptibility (Rose, 2001). The gist of Rose´s example is that only a comparison between populations with sufficient exposure differences could show otherwise. However, a key

difficulty for his approach is that, in order to study the determinants of the distribution of a disease, "the more widespread is a particular cause, the less it explains the distribution of cases" (Rose, 2001, p. 428). Since genetic heterogeneity seems to have greater variation within populations while the opposite occurs with environmental factors (Rose, 2001), then population effects are easy to overlook. Nonetheless, "there is hardly a disease whose incidence rate does not vary widely either over time or between populations at the same time" (Rose, 2001, p. 429). Rose's key insights are closely related to Durkheim's discovery that there are multiple reasons why someone commits suicide: those individuals come and go while the social rate of suicide remains constant and predictable due to its link with other social forces (Berkman & Kawachi, 2014).

The emphasis on the causes of individual health leads traditional epidemiology to hone in the proximal causes of disease (i.e. diet, cholesterol level, exercise, or life-style) and makes the web of multiple causation the theoretical canon (Krieger, 1994). A more critical Epidemiology emerges by acknowledging that the distal causes of population health have to include at least two "spiders", the biological and the social, in an ecosocial framework that considers history and agency (Krieger, 1994). The emerging epidemiological approach tries to substitute the metaphor of the "web of causation" for the "nested Chinese boxes", which represent the pursuit to investigate the hierarchical ecology of systems all from the molecular to the societal levels (Earls & Carlson, 2001).

Nancy Krieger (2001; 2011b, 2012) soundly outlines the core propositions of an ecosocial theory in Epidemiology. Foremost, "people literally embody, biologically, their lived experience in social and ecologic contexts, thereby creating population patterns of health and disease" (Krieger, 2011b, p. 215). The determinants of the social patterning of disease distribution are exogenous to people's bodies and manifest at different levels in multiple spatiotemporal scales,

therefore disease distribution cannot be reduced solely to explanations of disease mechanisms. Rather, the pathways of embodiment involve adverse exposures such as social and economic deprivations, social trauma, or inadequate health care, yielding the fact that impoverished populations disproportionately bear the burden of disease. Moreover, there is a cumulative interplay of exposure, susceptibility, and resistance across the life-course that stress the importance of timing and accumulation when evaluating these impacts (Krieger, 2011b).

Consequently, ecological approaches in epidemiology frequently link low socioeconomic status with poor health. Social conditions are considered the fundamental causes of disease that affect overall health because the lack of resources – money, power, knowledge, or social networks- is the key driver to prevent or avoid disease (Link & Phelan, 1995). Therefore, individual risk factors need to be contextualized as means to understand what puts people "at risk of risks". Moreover, considering that socioeconomic status is transportable from one situation to another, it maintains its link with disease even when intervening mechanisms change (i.e. proximal causes) and so the impact of individually tailored interventions is reduced (Link & Phelan, 1995). These insights follow Hart's inverse care law in which the "accumulation of health hazards tends to vary inversely with the power and resources of the populations affected" (Krieger et al., 2008). Accordingly, these new tenets guided the investigations on the changing social patterning of health, especially regarding social inequalities and the cumulative effects of exposure to discrimination (Gee, 2008; Krieger, 1999).

The UN Commission on the Social Determinants of Health, headed by Michael Marmot, helps advance the "causes of causes" agenda worldwide (Marmot, Friel, Bell, Houweling, & Taylor, 2008). Poverty, however, was not the sole emphasis of the ecosocial approach because Marmot added a key element: the social gradient in health. In a previous text, he was persuasive in demonstrating how, when taking the subway in downtown Washington, when heading towards

Montgomery County in Maryland, life expectancy increased by a year and a half for every mile travelled. He highlights how "subtle differences in neighborhood, or more importantly in other conditions affecting the people who live there, have great import for health and length of life" (Marmot, 2015, p. 27). He argues that the major health problem worldwide is health inequalities. These are clearly appreciated within countries, where the social gradient means that "from top to bottom, the lower our social position the worse our health" (Marmot, 2015, p. 29). However, the pervasive gradient appears between countries as well. Therefore, health inequity is not a problem of 'them', the poor, and 'us', the non-poor, but a collective effort in which everyone aims to achieve higher health standards: "improving society, improving everyone's health up to that of the best off, does not preclude extra effort on improving health for the poor" (Marmot, 2015, p. 29).

Just as sociologists are concerned with inequality, intersectionality and concentration effects, a novel population-based approach called *Syndemics*[1] studies why certain diseases cluster, interact, and co-vary with macro social forces that facilitate them (Tsai, Mendenhall, Trostle, & Kawachi, 2017). It examines how social conditions contribute to the formation and clustering of disease by increasing susceptibility and reducing immune function and thus contributing to progression (Singer et al., 2017). Syndemics eschew the traditional assumption of comorbidity in which independent diseases simply occur in tandem and it rather understands it as the adverse interaction of diseases that most likely emerge in contexts characterized by poverty, stigmatization, stress, or structural violence (Singer et al., 2017). Syndemics theory thus poses

[1] Syndemics are defined as the "aggregation of two or more diseases or other health conditions in a population in which there is some level of deleterious biological or behavior interface that exacerbates the negative health effects of any or all of the diseases involved" (M. Singer, Bulled, Ostrach, & Mendenhall, 2017, p. 941). A syndemic involves the population-level clustering of social and health problems that increases morbidity and mortality as a result of clustering and adverse interaction within a certain context.

that “co-occurring epidemics interact at the population and individual levels with mutually enhancing deleterious consequences for health” (Tsai et al., 2017, p. 978).

One of the most investigated syndemics is SAVA i.e. the intersection of substance abuse, violence, and AIDS. It refers to the interdependent conditions of individuals that mostly live in low-income urban environments. Unemployment, homelessness, overcrowding, substandard nutrition, disruption of social support networks, and ethnic inequalities characterize these contexts, but they also have higher rates of tuberculosis, sexually transmitted infections, hepatitis, cirrhosis, drug abuse, suicide, and homicide (Singer et al., 2017). Syndemics are not universal but shaped by local circumstances. For instance, diabetes and poverty frequently cluster together, but they interact with depression, tuberculosis, and HIV in countries that vary by income, health system, and cultural values (Mendenhall, Kohrt, Norris, Ndetei, & Prabhakaran, 2017). An important consequence of both syndemics is reduced treatment efficacies and increased treatment costs (Singer et al., 2017). The key importance of syndemics is how these interactions amplify disease burden and how it reduces the effectiveness of common interventions (Tsai et al., 2017). Nonetheless, research is still mixed and future studies need to incorporate a multilevel and longitudinal framework to understand how syndemics evolve across space and time (Tsai et al., 2017).

Space clustering, as studied by syndemics, is worse when its effects are examined as cumulative exposures. Complimentary to the ecosocial theorization –as well as in opposition to dominant frameworks in epidemiology– the life-course approach seeks to study, interdisciplinarily, the long-term and inter-generational effects of physical and social exposures through life (Ben-Shlomo & Kuh, 2002). The life-course perspective emphasizes that, as individuals age, they exit some social systems and enter new ones, which provide additional contexts for potential exposure, as with the transition from education to work (Gee, Walsemann,

& Brondolo, 2012). These transitional exposures may change in nature, importance, and intensity and thus shape the latency periods between exposure and disease appearance, the critical periods where it has its more profound effects, and the additional stressors that the exposure entails (Gee et al., 2012).

Associated with syndemics and the life-course approach is the notion of susceptibility. It refers to the assertion that social factors influence disease processes by creating a susceptibility to disease in general; the offset of a specific one depends on the environmental exposures as well as on the biological and genetic makeup (Berkman & Kawachi, 2014). In this vein, recent research shows how the cumulative effects of stress accelerate aging and alter neuroendocrine-mediated biological pathways that lead to disorders that range from cardiovascular disease to cancer (Berkman & Kawachi, 2014).

The ecosocial approach, paired with a life course perspective and syndemics, requires shunning the linear causal thinking based on paradigms of infectious agents and toxins (Hertzman & Boyce, 2010). In contrast, Hartzman and Boyce argue that the social causation of disease is nonlinear and disease outcomes are nested within complex and dynamic systems of exposures over time. In addition, social causation is nonspecific because "exposures to stressors and rearing in disadvantageous socioeconomic circumstances appear to augment risk for multiple categories of disorder by generating a generalized susceptibility within multiple causal paths" (2010, p. 331). Conversely, salutary contexts diminish liabilities to different types of disease. Biological embedding is caused by iterative, recursive, and amplifying exposures over time. Importantly, social causation involves "mundane rather than exceptional exposures i.e. the repeated, cumulative effects of wearing but otherwise unremarkable events, as opposed, for example, to a singular, transformative encounter with a highly virulent organism such as HIV" (Hertzman & Boyce, 2010, p. 331). In the dissertation, this translates into a concern for the subtle but constant

weathering of the body in violent contexts, rather than the trauma caused by victimization events. This paradigm shift also implies that social causation involves symbolic processes by which psychosocial determinants of disease deal with the meaning and affective valences of life experience, such as self-worth, social respectability, disregard, subordination, and helplessness (Hertzman & Boyce, 2010).

Since the nineteenth and early twentieth centuries, public health in the US and Britain called attention to the increased risk of disease among the poor and several attempts to improve their physical environments were broadly implemented (Berkman & Kawachi, 2014). Nonetheless, ecological fallacy reduced the credibility of this approach because it was considered impossible to rule out reverse causation i.e. that illness influences residential location. However, it was with multilevel analyses and improved designs that the assessment of exposures at the community level could separate individual traits and thus allowed for the understanding of how social determinants of health are more than the sum of individual-level measures (Berkman & Kawachi, 2014). Contrary to the long tradition in Sociology of thinking about the importance of context, the outlines of Social Epidemiology are quite recent. Studies attempting to explain neighborhood effects on health and the social sciences started in the late 1980s and grew exponentially over the next 15 years (Diez Roux & Mair, 2010). But early reviews already praise the importance of their findings in spite of the small effect sizes and the methodological challenges posed by design and multilevel interpretations (Pickett & Pearl, 2001). But currently "there is substantial evidence that health is spatially patterned" (Diez Roux & Mair, 2010, p. 138).

Under the novel perspective of individual behaviors nested in specific contexts, social and economic level interventions gained momentum and efforts to enhance the understanding of complex social and economic dynamics illuminated what "seems to be, but is not, individual

choice" (Berkman & Kawachi, 2014, p. 9). Interventions to change behaviors shifted its focus from the individual, proximate causes of disease, to attempts to shape norms, enforce patterns of social control, provide environmental opportunities to engage in certain behaviors, and reduce stress for which behaviors could be effective coping strategies (Berkman & Kawachi, 2014). Even though clinical trials aim at individual behavioral risk factors, "by and large the most successful have been those which incorporated elements of social organizational changes into interventions" (Berkman & Kawachi, 2014, p. 8).

The breakthrough of Social Epidemiology in general, and of context effects in particular, is possible due to the successful articulation of five trends (Berkman & Kawachi, 2014; Diez Roux & Mair, 2010; Diez-Roux, 2001). First, a more nuanced understanding of populations' distributions and a focus on exposures, interaction, and clustering rather than on specific diseases. This amounts to a growing interest in explaining the social determinants of health (Braveman, Egerter, & Williams, 2011). Second, the realization that not all health determinants are best conceptualized as individual-level attributes and that neighborhood contexts are related to health independently from them (Galea, Ahern, & Karpati, 2005). It creates an interest in how social experiences influence physiologic stress responses, particularly trough specific phenomena such as socioeconomic stratification. Third, the assessment of how social and economic policies could negatively impact health outcomes was combined with an awareness that policies aimed at populations could affect health trough their impact on the contexts in which individuals live and thus have substantially larger effects by moving the overall incidence mean (Rose, 2001). Fourth, the convergence with the life-course perspective provide a rationale to assess multiple patterns of change and heterogeneity of effects over time (Wodtke et al., 2011). Finally, there is an increasing availability of methods to assess neighborhood effects, most notably Hierarchical Linear models or HLM (Raudenbush & Bryk, 2002; Raudenbush & Sampson, 1999b). The

development of multilevel analytic approaches facilitated the empirical integration of the nested Chinese boxes.

Social epidemiology is thus now conceptualized as the branch of epidemiology that studies the social distributions and social determinants of states of health; it specifically, addresses "the way that social structures, institutions, and relationships influence health […] and how our social world powerfully shapes patterns and distributions of health" (Berkman & Kawachi, 2014, p. 2).

2.2 Mechanisms of context effects

Asserting the importance of context effects is insufficient to make them theoretically relevant or even useful. There is a need to specify how do they operate. The question "Do neighborhoods matter?" has had a disproportionate influence on the literature over the past two decades yielding the temptation to answer it with a crude yes or no response that trivializes theory and findings (Sharkey & Faber, 2014). Posing the question as a simple dichotomy obscures the ways in which neighborhood effects may have policy relevance (Ellen & Turner, 2003).

An influential article by Mayer and Jencks (1989) raised some serious doubts about the alleged "neighborhood" and "school" effects over the life chances of children. In particular, they highlighted how these studies "seldom tried to distinguish the effects of neighborhoods from the effects of neighbors" (Mayer & Jencks, 1989, p. 1442). They argued that it wasn't sufficient to show that places matter but it was fundamental to demonstrate how they matter as means to separate them from possible confounders. Most studies were based on a "black box" model with no assertions on how social composition affects individual behavior. Moreover, they certified that

neighborhood effects tend to be small, but more importantly that "the more we learn about a given outcome, the smaller the effects of mean SES look" (Mayer & Jencks, 1989, p. 1444). They called for a more nuanced analysis of the social mechanisms involved because it is not enough to explain how much would an individual's behavior change by moving to a more affluent neighborhood, it is needed to explain *why* it has such effect (Mayer & Jencks, 1989).

A first step toward this direction is to show which contextual characteristics exert an influence and on what. These characteristics may be truly contextual as they reflect collective attributes with no individual correlate, as inequality, or compositional characteristics made up of aggregate individual traits, like mean municipality income. Either way, "contextual measures such as mean friendship ties aim to describe a collective property (akin to a network) and not individual attributes" (Sampson, 2011, p. 237). In the last twenty years, the most popular contextual characteristics have been the poverty and inequality levels of a specific place, but other traits of interest have been: crime, social cohesion, racial composition, walkability, or food environment, among others (Diez Roux & Mair, 2010). A more recent public health review in the U.S.A found similar results, being poverty and the built environment the most common contextual predictors and obesity and mental health the most frequently studied individual outcomes (Arcaya et al., 2016).

After the proper definition of key contextual characteristics, the second theoretical challenge is to identify their multilevel linkages i.e. defend how are they operating. A fundamental theoretical challenge for context effects is to disentangle social interactions from the built environment and from institutional effects, especially if the intention of a study is to help design policy interventions (Ellen & Turner, 2003). The *how* question in context effects reminisces sociology's debate between the linkage between the macro and the micro analytic levels (Coleman, 1990). The notion of "mechanism" is usually invoked as a solution because it

refers to the "set of elements and their causal links that regularly lead from an initial social state to a subsequent one" (Demeulenaere, 2011, p. 12). From a perspective of Analytic Sociology, it refers to the explanation from the macro-social variables to the micro-social action and it implies an effort to identify the process by which a specific input leads to a particular outcome. Although the mere use of this notion already takes a stance towards a methodological individualism –which frequently disregards the macro-macro associations–, contemporary approaches now recognize that an actor is embedded in an environment shaped by previously defined norms (Demeulenaere, 2011). This means that beliefs and motives of individual actors are founded upon knowledge and norms, which are not of their own making, and therefore their objectives and representations are formulated in terms of their social environment. The efforts to elucidate the mechanisms of context effects usually occur under this epistemological framework.

George Galster analyzed the empirical literature on neighborhood effects and found that, although there is a broad theoretical agreement between social scientists and epidemiologists, "unfortunately there are few tentative conclusions, let alone consensus, about which mechanisms demonstrate the strongest empirical support" (Galster, 2012, p. 23). Nonetheless, he identified several "potential" pathways linking context with individual behaviors and health outcomes. Even though he was not the first or only one to compile these types of social mechanisms (Elliott et al., 1996; Mayer & Jencks, 1989; Sampson et al., 2002; Small & Newman, 2001; Wodtke et al., 2011), Galster's groupings[2] are comprehensive enough to illustrate them.

Social Interactive Mechanisms

This group of interrelated mechanisms reflects social processes within neighborhoods. Its theorization began after establishing the broad features of concentrated disadvantage, when the

[2] Galster's original taxonomy (2012) was modified. The "environmental" and "geographical" sets were merged into one. In addition, the number of mechanisms was reduced from 15 to 10 because they were somewhat redundant. Importantly, "exposure to violence" was an environmental mechanism but it was left out so it can be treated in depth on the following section.

analysis shifted to moderating variables between macro-social characteristics and individual outcomes (Sampson & Groves, 1989).

1. **Social Contagion** refers to how contacts with neighbors may change behaviors, aspirations and attitudes (Galster, 2012). The rationale of peer influences is that "like begets like" so if children grow-up in neighborhoods where other kids engage in antisocial behavior, it is likely that they will too. Conversely, if other teens value school, they may do as well (Jencks & Mayer, 1990). However, these models fail to acknowledge other individual differences and do not explain why neighborhoods are rarely homogeneous but tend to have some adolescents who steal cars and some who finish school (Jencks & Mayer, 1990). Their value resides in the key insight that exposure to a given behavior increases the odds of engaging in that behavior (Galster, 2012).

2. Similarly, adult influences focus on **collective socialization** models where different types of adults (i.e. role models) have different effects on kids (Jencks & Mayer, 1990). Nonetheless, it requires a minimum threshold or critical mass before a norm produces noticeable consequences in the neighborhood (Galster, 2012). For instance, adults who promote hard work and discipline will model these behaviors on the kids of the neighborhood. Likewise, this kind of behavior will make them "enforcers" of discipline for kids in the neighborhood, even if they are not their own (Jencks & Mayer, 1990). A good example of collective socialization is the role of informal social control within neighborhoods (Sampson, 2012). Parental mediation of physical and mental health, as well as overall stress, coping skills, efficacy and material resources may also affect the environment where children are raised (Galster, 2012). Moreover, parental mediation can be observed in how kids may absorb the "Black English vernacular" by which they do poorly in

school and get rejected in job interviews, thus creating linguistic isolation (Small & Newman, 2001).

3. The influential social experiment 'Moving to Opportunity' had the underlying assumption that advantaged neighbors cause individual advantages (Leventhal & Brooks-Gunn, 2003; Sampson, 2008). However, advantaged neighbors can also bring disadvantages, especially when the advantage is socioeconomic status (Jencks & Mayer, 1990). For instance, mechanisms based on **relative deprivation** propose that people assess their success or failure by comparing themselves with their neighbors (Jencks & Mayer, 1990). This means that, keeping income constant, someone may feel poorer with rich neighbors than with poor neighbors. The same rationale may apply with a child's ability in a high and low socioeconomic level school (Jencks & Mayer, 1990). When individuals evaluate their own situation relative to others, it is inequality rather than absolute poverty what matters (Kawachi & Subramanian, 2014). In this scenario, moving a poor family to a high-income neighborhood could have deleterious effects. For example, a recent study finds that adolescent's consumption of symbolic status goods is significantly associated with their blood pressure dependent upon parental economic resources (Sweet, 2010). In addition, if there is competition for scarce resources (i.e. teenagers compete for jobs), a more affluent neighborhood could hamper the chances to thrive if between two persons only one has a better education (Ellen & Turner, 1997) and more privileged social capital (Bourdieu, 2010); "in both cases a big frog in a small pond is probably better off than a small frog in a big pond" (Jencks & Mayer, 1990, p. 118).

4. Social networks or **social capital** stands for the ability to secure benefits through collective membership (Kawachi, Takao, & Subramanian, 2013; Putnam, 2001). These networks influence

interpersonal communication of valuable information and resources of various types through networks characterized by weak and strong ties (Granovetter, 1973; Smith & Scrivens, 2013). A notable example is how Tokyo experienced different recovery rates by neighborhood after the 1923 earthquake and longitudinal analyses revealed that social capital –more than actual damage, density, education or economic capital- was its best predictor (Aldrich, 2012). While positive externalities are frequently highlighted, there is a negative or dark side to social capital; especially exclusion of outsiders, excess claims on group members, restrictions on individual freedoms, and downward leveling norms (Portes, 1998). The strong ties that enable group members to get privileged access to limited resources are the same that exclude others from getting them (Ahn & Ostrom, 2002). Social capital is neutral, therefore it's difficult to control when it is used to harm, exclude or dominate others (Svendsen & Svendsen, 2009). A key critique is whether the excessive extensions of the concept jeopardize its value, especially when authors, like Putnam (2001), argue that social capital is a trait of communities or nations rather than individuals and simultaneously treat it as a cause and as an effect (Portes, 1998). A second important critique is that the stock of friends implied with the concept is based on a bucolic imagery of socialization processes based on a communitarianism that might not be appropriate for urban anonymity based on organic solidarity (Portes & Vickstrom, 2011). Moreover, these stocks of friends might activate only for specific problems and not others, making it an unreliable mechanism for some issues, like crime (Sampson, 2012).

5. Durkheim argued in the Division of Labor (1964) that modern societies based their **social cohesion** in organic solidarity and, for the proper functioning of modern cities, individuals didn't rely so much in personal acquaintances but in shared values and the recognition of a mutual normative order that grants individual goals. The conditions for organic solidarity were: diversity

among members of a society, a complex division of labor, and strong institutions (Portes & Vickstrom, 2011). An insightful example is how Shaw and Mckay showed that clusters of social problems turn into social disorganization i.e. the inability of a community structure to realize the common values of its residents and maintain effective social controls, which hampers social cohesion (Shaw & McKay, 1942). They found that the key intervening processes between concentrated disadvantage and antisocial behavior, at the neighborhood level, were (a) the ability of a community to supervise and control teenage peer groups to thwart antisocial behavior from becoming a criminal activity, (b) the density of informal local friendship networks as another constraint to deviant behavior and guardianship against strangers, and (c) local participation in formal and voluntary organizations which reflects the solidarity of a community to defend local interests (Sampson & Groves, 1989). Conversely, an important application of the ideas of Shaw and Mckay was Skogan and Maxfields' (1981) recognition that low-level breaches of community standards signal an erosion of conventionally accepted norms and values, which cue that the risk of victimization is higher, thus triggering fear of crime. Recent evaluations of the theory of Social Disorder show how it is deeply implicated in the dynamics of a neighborhood and has notable consequences for public health and crime, as well as feedback loops with crime (Skogan, 2015).

Environmental and geographical mechanisms

These mechanisms refer to natural and human-made attributes of a neighborhood that may affect specific behaviors. In essence, they refer to the problem of distance to other people or resources and exposure to harm (Galster, 2012). It is also important when referring to exposure to spatial clustering i.e. "zones in which there is a larger than expected concentration of some characteristic" (Logan, 2012, p. 516).

6. **Physical surroundings** such as decay of the built environment (litter, graffiti, or abandoned structures) may have psychological effects on residents, like a sense of powerlessness (Galster, 2012). Likewise, Social Disorder is also comprised by a physical dimension of decay that causes fear of crime (Brunton-Smith, 2011), even though these are shared perceptions closely associated with racial and socioeconomic stereotypes (Brunton-Smith, Jackson, & Sutherland, 2014; Sampson & Raudenbush, 2004).

7. Residents may be exposed to toxic or unhealthy elements like water-borne pollutants or even noise (Burton, Matthews, Leung, Kemp, & Takeuchi, 2011).

8. The lack of accessibility or isolation may create a **spatial mismatch** between location and economic opportunities (i.e. resources, jobs and public transportation) that may reduce the chances to get decent work in spite of personal skills (Galster, 2012). There may be substantial variation in the prevalence of private actors that encourage certain behaviors of the residents (Ellen & Turner, 2003), like the strategic location of Starbucks' coffee shops that seem to be *everywhere* (Sampson, 2012). Another good example is food desserts, where low-income urban populations have limited access to healthy foods, which leads to poor nutrition and health inequity (Widener, Metcalf, & Bar-Yam, 2011). As in many places, in Philadelphia the highest income neighborhoods had 156% more supermarkets than the lowest income neighborhoods and, since low-income households do not have access to a car, they purchase food items in nearby stores, thereby sacrificing cost and quality for convenience (Walker, Keane, & Burke, 2010).

Institutional Mechanisms

The last group involves the actions of people not residing in the neighborhood but who control key institutional resources located there and who constitute the bridge with vital markets (Galster, 2012).

9. A fundamental mechanism by which neighborhoods affect individual outcomes is by access or deprivation to **institutional resources**, that make stimulant or poor environments, especially regarding education, housing, transportation, and health care (Elliott et al., 1996; Massey, 1990). The quality, quantity, and diversity of institutions like community-based organizations that provide public goods are a key feature that explains neighborhood variability (Sampson, 2011). The importance of adults outside the community lies in actors such as teachers, policemen, and other neighborhood institutions (Jencks & Mayer, 1990). Moreover, some neighborhoods may offer inferior public services and facilities due to their limited tax-based resources, incompetence, corruption, or other operational challenges (Galster, 2012). What is sometimes described as the effect of neighbors can actually be attributed to local policies, neighborhood institutions, or school practices (Massey & Brodmann, 2014). A good example of the importance of local services and institutions is the quality of the nearby public school and its significance in individual outcomes (Ellen & Turner, 2003).

10. Neighborhoods may suffer from **stigmatization** on the basis of public stereotypes –based on history, style, type of dwelling, commercial districts, etc.– held by powerful public or private actors independently of the current residents but with the ability to reduce their opportunities and self-esteem (Galster, 2012; Sampson & Raudenbush, 2004). An eloquent example is the enduring discredit of black ghettos in the US as crime-prone, drug-infested, and impoverished (Anderson, 2012).

These potential mechanisms for social contexts are good clues about the ways in which a context influences individual behavior. However, they still lack specificity. Using a pharmacological metaphor, the problem with the state of the literature is "what about this dose of neighborhood might be causing the observed individual response?" (Galster, 2012, p. 27). In this sense, identifying broad mechanisms might not be enough because there is a need to quantitatively know their relative contribution. A stricter *how* question refers to the active processes that must intervene in a neighborhood to produce an effect. There are many more gaps about the treatment and the dose that need further research: the timing, frequency, duration, intensity and thresholds of the *dose*; the consistency, extent, interactions, buffers, generality and antidotes to the *treatment (Galster, 2012)*. These knowledge gaps indicate that the field is promising but still in its infancy.

2.3 Contextual violence

2.3.1 From individual to contextual violence

Canonical research on violence uses the individual as the unit of analysis with the objective to find 'risk factors' (Collins, 2009; Pratt & Cullen, 2005). The common association on these studies is the link between a person's social class and antisocial behavior (Tittle, 1983) and the most popular approach among criminologists was Hirschi's social bond theory (Fox & Bouffard, 2015; Pratt & Cullen, 2005). Beyond criminology, victimization has also been tested for its association with health. Following the individual approach, studies using the first two waves of the MxFLS find that victimized individuals are more likely to suffer an anxiety disorder, fear, pessimism, headaches, chest pain, increased alcohol consumption, and smoking;

and these effects are stronger in women (Cuevas, Rubalcava Peñafiel, & Teruel, 2006). Likewise, it was estimated that victimization affects sleeping habits and fosters citizens to arm themselves (Braakmann, 2012).

The first attempts to change the unit of analysis and instead examine contextual violence aimed to aggregate individual-level theories to the neighborhood. An interesting example is the effort to prove that Agnew's General Strain theory could be equated to the context i.e. if strain is mostly understood as individual poverty, then strain could also mean concentrated disadvantage in the neighborhood (Warner & Fowler, 2003). Another similar attempt takes Merton's anomie theory (Merton, 1968) to the institutional level (Messner, Thome, & Rosenfeld, 2008), which has the merit to shift excessive focus on the neighborhood.

Nonetheless, before the hype of the neighborhoods effects literature, there was already a realization that place´s characteristics –by themselves and not mere as an aggregation of individuals– were associated with violence. There were two main competing theories on this line of thought: routine activities and social disorder (Miethe, Hughes, & McDowall, 1991). In spite of being strongly cemented on rational theory, the first posits that places favoring criminal opportunities –vulnerability of victims, motivation of criminals, or lack of guardianship– lead to higher criminal rates (Felson, 2000). The second argues that crime rates flourish in places communicating that low-level antisocial behavior is allowed and thus favor the actual emergence of more severe criminal activities, in what was later simplified as 'broken windows' (Skogan, 2012). Likewise, the rediscovery of Shaw and Mckay's theories (1942) boosted the focus on the role of social networks (Papachristos, Hureau, & Braga, 2013), informal social controls and collective efficacy (Bursik, 1988; Sampson et al., 1997) and even cultural attitudes like legal cynicism (Kirk & Papachristos, 2011) within the neighborhood. At the same time, research challenged the so-called culture of violence and rather explains it as economic inequalities in

metropolitan areas (Blau & Blau, 1982; Kawachi et al., 1999; Morenoff et al., 2001), racial inequality (Shihadeh & Barranco, 2013) and with concentrated disadvantages (Wilson, 2012), which are socially corrosive on several aspects of well-being (Wilkinson, 2006; Wolf, Gray, & Fazel, 2014).

The convergence of these theories contributed to a renewed emergence of macro-social theories in Sociology to explain crime and, by doing so, they innovated in the ways that these contextual characteristics are operationalized (Bursik & Grasmick, 1993). The same effort in public health strengthened the examination of the effects of place on health (Macintyre, Ellaway, & Cummins, 2002), the ways to measure urban health (Prasad, Gray, Ross, & Kano, 2016), and violence started to be conceptualized from an ecological perspective (Krug, 2002).

An influential meta-analysis of studies conducted in the USA synthesized the results of the renewed macro-social approach and concluded that the strongest and most stable macro-level predictor of crime was contextual poverty measured as concentrated disadvantage (Pratt & Cullen, 2005). Social disorder also received strong support, followed by theories based on anomie, social support, and routine activities, which received only moderate support. Theories with the least support were based on indicators associated with the criminal system (Pratt & Cullen, 2005). Likewise, research on the historic covariates of homicide rates in the U.S.A claims that the strongest and most invariant covariate was a resource-deprivation index, which was consistent across four censuses and along cities, metropolitan areas, and states, while the next predictor in importance was population density; and the least important predictors were unemployment and percentage of population 15-29 years old (Land, McCall, & Cohen, 1990). Re-estimations of the study confirm these results (McCall, Land, & Parker, 2010, 2011). Even in Latin America, poverty, income inequality, illiteracy and alcohol consumption are the main predictors of homicide rates (Frías & Castro, 2011; Nivette, 2011).

The thrust of these findings is that violent crime is known to be concentrated in the same urban neighborhoods as poverty and associated disadvantages (Stretesky, Schuck, & Hogan, 2004). Even though from a historical perspective crime rates are dwindling (Pinker, 2011), they recently declined most in the most violent and disadvantaged neighborhoods but, they still remain concentrated in these same neighborhoods in the U.S.A, only with lower overall rates (Friedson & Sharkey, 2015). Notably, in spite of these recurrent findings over many studies, poverty only increases the likelihood of high homicide rates but that does not mean that poor places worldwide are violent, as the case of Calcutta illustrates, but other structural variables can disrupt the apparently inextricable association between crime and poverty (Sen, 2008).

2.3.2 Indirect consequences of contextual violence

Theories in criminology try to explain crime, so delinquency is frequently the dependent variable under study. Nonetheless, a different line of research aims to establish what are the consequences of violence. Direct impacts of crime are usually assessed as the effects of victimization and include both physical injuries and psychological trauma (Macmillan, 2001; Ribeiro et al., 2013). Indirect impacts of crime are harder to apprehend but they can be measured as fear of crime (Ferraro, 1995) or as vicarious victimization when a close person suffers the injuries, the trauma, or is perceived to be in danger (Boelen, van Denderen, & de Keijser, 2015; Warr & Ellison, 2000). These indirect consequences of crime can be assessed at the ecological level as the impact on well-being of living in violent contexts regardless of suffering victimization events or having other individual characteristics (Lorenc et al., 2012).

The former approach is best illustrated with the advent of research on community violence. Interest in community violence began when, during the 1980's and the beginning of the 1990s, several cities in the U.S.A faced an important increase in the homicide rate (Selner-

O'Hagan, Kindlon, Buka, Raudenbush, & Earls, 1998). The first researchers sought to create an epidemiological measurement to understand the size and intensity of the violent waves by means of measuring the extent of *exposure* to violence and victimization (Richters & Martinez, 1993). The original 15-item scale of collective violence assesses the frequency in which the respondent heard, witnessed or was victim of: shootings, stabbings, sexual assault, muggings, physical threats, drug trade, drug use, arrest, punches, use of illegal weapon, forced entry (own or other), dead body, murder, and suicide. As expected, the results showed that witnessing community violence was more prevalent than experiencing it (Richters & Martinez, 1993).

The next step was a conceptual refinement of the concept (Steinbrenner, 2010). Importantly, the term "violence" is used in a restricted sense, in opposition to structural violence (DeVerteuil, 2015), to reflect a bundle of predefined criminal acts, which are most common in poor urban neighborhoods in the USA and England (Eisner, 2009). In this context, violence is usually a short cut to describe violent crime, an activity based on two instrumental behaviors: harm-doing and rule-breaking (Felson, 2009). The term *community violence* denotes acts of interpersonal violence that occur in community settings, including neighborhoods, streets, schools, shops, playgrounds, or other community locales and these acts may include such incidents as gang violence, rapes, shootings, knifings, beatings, or muggings (Guterman & Muahhmmad, 2008). The concept distinguishes private from public violence, especially domestic abuse, but is oftentimes confounded with perceptions of insecurity and fails to account on whether the source of violence is socially or politically motivated (Guterman, Cameron, & Staller, 2000; Martin, McCarthy, & McPhail, 2009). According to the emerging taxonomy, the

violence in Mexico would be considered as social because of the alleged links to organized crime[3] (Krug, 2002).

An important implication of the study of collective violence is the number of studies that establish some of the negative consequences of exposure to community violence. A meta-analysis identifies that the most common outcomes are mental health problems, especially post-traumatic stress disorder (Fowler, Tompsett, Braciszewski, Jacques-Tiura, & Baltes, 2009). The intensity of the exposure to violence is moderated by the proximity to the event (first as victim, then as ocular witness, and finally when only heard about), the frequency and the chronicity of the exposure, and it has a higher impact with adolescents than with children. The authors conclude that the exposure to community violence presents a unique chronic and detrimental type of trauma that includes victims and witnesses (Fowler et al., 2009). From a public health perspective, exposure to diverse types of community violence increases the rates of infant mortality, communicable diseases and disability in a community and reduces access to food (Krug, 2002). Moreover, it impacts specific populations by displacement, impoverishment, and further human rights abuses (Krug, 2002). Community violence has been tested for additional purposes; the outcomes range from more aggressive parenting practices (Zhang & Anderson, 2010), lower academic achievement (Margolin, Vickerman, Oliver, & Gordis, 2010), and higher prevalence of substance abuse (Wright, Fagan, & Pinchevsky, 2013) to higher rates of violent youth (Wilkinson & Carr, 2008), the reproduction of violence (Patchin, Huebner, McCluskey, Varano, & Bynum, 2006) and a higher prevalence of suicides (Lambert, Copeland-Linder, & Ialongo, 2008).

[3] Therefore, all the direct and indirect consequences of war and political conflicts are not considered in the present literature review. Even though Mexico's complex characteristics oftentimes blur with war scenarios (Schedler, 2015), the dissertation narrows down contextual violence to violent crime only. Nevertheless, when examining the consequences, both types considerably overlap on its effects (Miller & Rasmussen, 2010; Panter-Brick, 2010).

2.3.3 Widening the assessment of the consequences of contextual violence

Even though good measures of community violence are available (Buka, Stichick, Birdthistle, & Earls, 2001; Suglia, Ryan, & Wright, 2008), due to data availability and the need to aggregate from the individual, the preferred measure to assess contextual violence is homicide rates as a contextual trait that is not based on subjective perceptions (Eisner, 2009; Galster, 2012; Sampson et al., 2002). The homicide rate also favors comparability between states and regions and thus facilitates examination of unequal distributions of violence (Briceño-León, Villaveces, & Concha-Eastman, 2008). Currently, the homicide rate is the most widely used indicator of contextual violence even though it fails to distinguish between individual experiences of victimization and perceived vulnerability (UNODOC, 2014).

A key improvement in the assessment of contextual violence was the use of multilevel models because they can control personal and contextual variables at the same time (Sampson et al., 2002). Using this methodological framework, health and well-being have been strongly associated with social characteristics of neighborhoods (Earls & Carlson, 2001; Helliwell, 2003; OECD, 2011; Sampson, 2003; Sarracino, 2013). For example, a cross-sectional study in Baltimore finds that, after controlling for age, education, and marital status, mothers with high exposure to neighborhood violence were twice as likely to express poor health, smoking, lack of exercise, and poor sleeping habits than the unexposed (Johnson et al., 2009). A similar analysis reveals significant effects on mother's depressive symptoms and general aggression, which in turn shapes their disciplinary practices (Mitchell et al., 2010). Likewise, higher levels of neighborhood violent crime rates and perceptions of neighborhood insecurity are associated with higher levels of depressive symptoms on older adults, controlling for sex, age, and household income (Wilson-Genderson & Pruchno, 2013). Crime rates also were found to impact social networks and reduce collective efficacy (Uchida, Swatt, Solomon, & Varano, 2014). A different

study noticed that crime rates affected levels of fear of crime, but not subjective well-being (Hanslmaier, 2013).

Longitudinal studies confirm prior research indicating that exposure to different forms of violence across multiple domains of life has negative impacts on adolescents' outcomes; in particular, exposure to violence in a one-year period increases the frequency of illegal substance use three years later but it has cross-level interactions with victimization and previous use of alcohol and marijuana (Wright et al., 2013). Other negative effects of exposure among youth include lower high-school graduation rates (Wodtke et al., 2011).

A limitation in many of these studies is the lack of empirical support for the mechanisms linking contextual violence with these outcomes. Exceptions are a multilevel study that tests neighborhoods' stress-buffering mechanisms –like households' occupancy and churches per capita- and shows they have a lower likelihood of alcohol, drug, and mental health disorders, and, conversely, individuals exposed to violence in high crime neighborhoods are vulnerable to depressive and anxiety disorders (Stockdale et al., 2007). More nuanced studies exploring cross-level interactions identify how neighborhood disadvantage increases exposure to peer violence for men and women, but peer violence has a stronger effect on women, reducing the gender gap in criminal behavior (Zimmerman & Messner, 2010). An important study discovers that children have worse performances on cognitive skills assessments if these are administered four to seven days after a local homicide occurred near their home and the mechanisms involved are most likely stress, shock, trauma, and fear (Sharkey, 2010).

In spite of the expanding literature on context effects enough empirical evidence is still lacking to support the theoretical claims on the mechanisms (Sharkey & Faber, 2014). Nevertheless, one of the most robust mediators between concentrated disadvantages, violence and health outcomes is stress (Burgard & Kalousova, 2015). The ways in which stress yields

deleterious health outcomes is described later. Nonetheless, a fundamental challenge in the field is to separate the effects of concentrated disadvantages and identify distinct mechanisms, as the ones for contextual violence independently from poverty.

2.4 Theoretical challenges for the context effects

The major theoretical problem that the context effects literature is trying to address is the often-contested issue of the micro-macro relations between agency and structure (Entwisle, 2007). While some have theorized that only micro-macro relationships are worth studying (Coleman, 1990; Homans, 1958, 1964) others believe there is a macro-micro interaction (Bourdieu, 1984; Durkheim, 1982; Massey & Brodmann, 2014). The first ones take social interactions as the independent variable to figure out the social structures they produce, while the second group takes the structures, once created and maintained, as the independent variable and see its consequences on individuals (Demeulenaere, 2011). Regardless of the epistemological team, both positions face great challenges to explain the linkages between levels; these difficulties involve solving, at least, the next intractable questions.

2.4.1 Object of study of the context effects

The great influence of the Chicago School of Sociology and particularly Park's theorizing of urbanism as a specific way of life –also Burgess, but he was more focused on urbanization developments– led to an incipient urban sociology (Castells, 1977). However, the specific object of study, the "urban", was not clearly differentiated from other social processes. Park´s type of urban analysis –characterized by the loosening of family ties, diversity, role segmentation, individualistic competition, anonymity, isolation, instrumental relations, and the absence of direct social control– is unveiled as the process of integration or acculturation to capitalist

industrialization (Castells, 1976). In this sense, the study of "deviant urban behavior" was actually the analysis of the differential opportunities and difficulties to integrate into (American) modern cities. The unsolved question for an urban sociology was then its own object of study. Is the urban life the integration to American capitalism or to Modernity itself? This uncertainty has direct implications in the definition of the independent variables, the levels of analysis, and the area of a "neighborhood".

2.4.2 Direction of causality

Another sensitive issue is the need to disentangle if spatial processes shape social interactions or if the process goes the other way around. This is the classic sociological problem between the agent and the structure: places, as well as institutions, are built through human practices and then these same places and institutions guide and shape future human practices (Giddens, 1984). For the urban case, industrialization processes yielded the modern city but, for current inhabitants, the city is an external fact that guides their interactions (Castells, 1976). Do we create our neighborhoods or do they create us? Castells argues that the key issue is not if urban contexts have specific effects but which *types* of urban contexts have such effects. Moreover, he claims that it is necessary to distinguish how "urban" is spatially coded and at what level of analysis specific social processes or "contexts" (social systems, systems of actions, or systems of signs) are analyzed (Castells, 1976). The problem is well illustrated when Shaw and Mckay do not differentiate between cause and effects and thus social disorganization was simultaneously considered as a cause *and* as an effect of crime (Bursik, 1988). The effort to define the social mechanisms involved in the context effects intends to solve this issue.

2.4.3 Possible mismatch between social and spatial units

In addition to such criticisms, a poignant question for the "context effects" literature is how social and spatial units coincide, if they do (Castells, 1976). Are moral regions geographically situated? Do Park's "little worlds" have clear boundaries? Do relevant social networks reflect at the neighborhood level? Most sociologists conceptualize neighborhoods in terms of informal relationships or networks that take place in a geographic space, and thus confound the term "neighborhood" with "community", even when these are separate and distinct attributes that may have different effects on individuals (Small & Newman, 2001). It is unclear if Sociology necessarily required the spatial delimitation (Castells, 1977); for instance, Max Weber didn't need the notion of place and defined the city in strictly institutional terms, as a political-administrative autonomy within a given economic base and spatial location (Castells, 1976; Weber, 1958). Therefore, when contexts are defined as neighborhoods, the assumption is that community processes mirror a spatial area, an assumption rarely proven. For some authors, however, the distinction may be futile because it is the "intersection of practices and social meanings with spatial context that is at the root of neighborhood effects" (Sampson, 2011, p. 230).

2.4.4 Assumption of within neighborhood homogeneity

Detailed analyses aiming to demonstrate if theorized social processes actually do occur in delimited neighborhoods tend to show how geographic communities are not homogeneous (Jencks & Mayer, 1990). There are at least two notable examples. First, the analysis of urban life heavily relied in the contrast with rural life. Currently, the mechanic inclusion of "urban residence" as a statistical control for unobserved variables –as if both types of people are

significantly different– is becoming problematic because there is a blurring and shifting of rural-urban spatial boundaries in a strong inter-dependence and there is a hardening of *aspatial* boundaries (like race and class), at least in the United States (Lichter & Brown, 2011). Moreover, these contrasts have often yielded conflicting results, as with mental health comparisons (Galea, Freudenberg, et al., 2005). Second, there is a strong conception of the *ghetto* as a particular type of neighborhood with a cohesive set of characteristics like deteriorating housing, crime, depopulation, and social isolation but it's a stereotyped image based on unacknowledged heterogeneity grounded in the idea of the sole-entity state and the involuntary segregation (Small, 2008).

2.4.5 A social or a spatial definition of a context

In spite of the fact that "the city" or "the urban" are probably dubious objects of study by themselves and that "neighborhoods" have considerable within heterogeneity, there is a growing agreement that *place* is not only a setting for social action but a force with identifiable and independent effects on social life (Burton et al., 2011). Since everything happens somewhere, all action is embedded and could be affected by its placement, especially in relation to others (Logan, 2012). *Places* –characterized by location, material form, and invested with meaning and value– have been found to stabilize social categories, reinforce social differences and hierarchies, arrange patterns of interaction and network formations, promote or hamper collective action, and embody cultural norms, identities, memories, and values (Gieryn, 2000). *Place* contributes to health processes by constraining action, limiting resources, and imposing norms and behaviors (Leung & Takeuchi, 2011). The notion of "neighborhood" is not a predefined arrangement of streets and houses but an ongoing practical and discursive production and imagining of people

(Certeau, 1984). Neighborhoods emerge when common people transform a continuous and abstract space into an identified, bounded, significant, and named place through daily practices, experiences, and histories (Gieryn, 2000).

From a psychological perspective, the "sense of place" indicates that individuals require an environment where needs are satisfied and people experience emotional ties developing attachment, familiarity and identity (Leung & Takeuchi, 2011). Displacement constitutes a traumatic shock that destroys emotional and social ecosystems and clearly evidences the deleterious consequences of dismantling social support networks (Leung & Takeuchi, 2011).

Unfortunately, however, most sociological studies tend to study neighborhoods in terms of its economic and demographic features and boundaries (i.e. poverty rates, racial proportions, or homicide rates by census tracts or municipalities) thus neglecting key features of places (Gieryn, 2000). On the contrary, when researchers use socially defined neighborhoods to geographically locate them (Sampson, 2012), the disadvantage is that the more accurately they reflect local perceptions of boundaries, the more costly and time-consuming it is to draw them (Small & Newman, 2001).

Nonetheless, ecological metrics have their own logic, besides individual assessments. Neighborhoods are valid units in themselves (Sampson et al., 2002); they are ecological units nested within successively larger communities but they are frequently operationalized with administrative boundaries as census tracks (Gieryn, 2000). Neighborhoods are sites, perceptions, networks and cultures, and therefore should be considered as several complementary dimensions, such as a social space; a set of relationships, a set of institutions, and a symbolic unit (Small & Newman, 2001).

A similar problem haunts epidemiology because there is no clear definition of what constitutes a "population", who defines it and under which criteria (Krieger, 2011b). Frequently

the rarely theorized solution has been to equate populations with geographically defined locations as neighborhoods (Ahern et al., 2008).

In sum, the literature on neighborhood effects has always struggled to define its core concept, the neighborhood, because there are discrepancies derived from theoretical debates, from the inhabitants own perceptions of what their neighborhood is and where its boundaries lie, and from definitions based on data availability, oftentimes from administrative demarcations (Raudenbush & Sampson, 1999b).

2.5 Methodological challenges to context effects

A neighborhood effect is the independent causal effect of a residential community on any social or health outcome (Jencks & Mayer, 1990). Putting people into place means explaining individual outcomes in association to a potentially changing social and spatial context (Entwisle, 2007). Notably, proving such an effect –the *potentially* changing context- represents enormous challenges for a researcher. Besides the black box problem (Mayer & Jencks, 1989), "much of the literature on neighborhood effects has been methodological, and with good reason" (Small & Newman, 2001, p. 30). In spite of the statistical advances with HLM, it is considerably difficult in terms of research design to test that, *ceteribus paribus*, an individual living under some neighborhood condition is worse or better off in the absence of that condition (Small & Newman, 2001).

Many researchers have pointed out these big methodological challenges. As an example, in the sociological literature, Sampson and colleagues (Sampson et al., 2002) highlight a few of them: ecological and selection bias; excessive focus on place of residence when people move in multiple neighborhoods; account for spatial interdependence; common use of static cross-sectional designs when experimental or life-course designs could account for dynamics of

change; and absence of benchmark data collection on neighborhood social processes. A similar account in the public health literature appears in Diez-Roux & Mair (2010) who claim for: improved casual inferences from observational studies and attention for natural experiments; definition and measurement of relevant spatial and dynamic contexts; inclusion of life-course processes and synergistic effects; and consideration of stress-related mechanisms besides behaviors. In addition, methodological threats to observational studies posed by social stratification and endogeneity of effects challenge the independence of observations and thus impedes causality claims unless randomized community trials are conducted (Oakes, 2004).

Since these methodological challenges continue to be serious concerns (Sharkey & Faber, 2014), a brief account of them aims to explain the methodological problem and possible ways to solve them.

2.5.1 Ecological bias and endogeneity of effects

A fundamental reason why the theories of Park or Shaw and Mckay were not fully developed until recently was Robinson's critique (1950) of the ecological fallacy i.e. the problematic results of making individual-level inferences on the basis of aggregate data (Bursik, 1988). This type of bias reconnected with the epistemological difficulty to make macro-micro inferences and led to a continued emphasis on individual-level analysis (Coleman, 1990). After Robinson, theoretical development focused on the individual level, like Merton's strain theory of crime using the famous "opportunity models" (Merton, 1968), but also Hirschi's control theory (Bursik, 1988), and more recently the widely used Felson's routine activity theory for crime prevention (Felson, 2000).

Ecological bias can be observed in several other ways. One of the biggest problems when estimating context effects is distinguishing between individual, family and neighborhood effects. It is particularly hard to decide which parental characteristics are exogenous to where they live and which are endogenous (Mayer & Jencks, 1989). Ecologic studies with aggregate data cannot directly differentiate whether differences across areas are due to characteristics of the areas themselves or to the differences between the types of individuals living in different areas (Diez-Roux, 2001). Therefore, it is important to distinguish between the effects of "context" (area or group properties) and of "composition" (characteristics of individuals living in different areas) as well as the heterogeneous impact they may have in different populations (Duncan, Jones, & Moon, 1998). Therefore, there is a great need to control for individual and family characteristics such as parental income, size of household, and family composition (Diez-Roux, 2001).

The theoretical landscape dramatically changed with the advent of HLM techniques – particularly through the influential explanations of Stephen W. Raudenbush and Bryk (2002)– and powerful statistical software (Oakes et al., 2015; Raudenbush & Sampson, 1999a). The possibility to model nested data provided three distinct advantages over traditional econometric techniques: 1) the use of multiple data sources at different levels with improved estimation within units; 2) the possibility to partition the variance across levels and thus model predictors and controls at their corresponding levels; and 3) test cross-level interactions to examine social mechanisms i.e. the way person-level associations with the outcome vary by contextual characteristics (Raudenbush & Bryk, 2002). It was the second point that helped dispel the threat of ecological bias. Before the HLM techniques, it was difficult to discern whether it was a poor school or a poor family that cause a kid's deficient school performance. Now multilevel models control for individual characteristics, as family income, and focus on school's traits variability. Therefore, the HLM model now poses that two equivalent kids on family income differ in

performance due to the school's characteristics. In a nutshell, these techniques opened the possibility of testing the macro-micro relationships and interactions with sound statistical techniques that help control for individual confounders.

The switch from individualistic research to the context effects literature fostered a move to "social research away of the view of people as fully rational actors in control of the world around them, and toward a perspective that views human beings embedded in and responding to a set of environmental influences that shape them" (Massey & Brodmann, 2014, p. 330). This view considers human actions and decisions as subject to constraints that limit the range of information and experience accessible and determine the quantity of resources available for growth and development which in turn guide the attributes, meanings, and scripts that help make sense of the world (Massey & Brodmann, 2014). This new perspective eschews an excessive focus on the individual-level, which neglects the effects of context, and points out to the need to complement psychometrics with *ecometrics* (Raudenbush & Sampson, 1999b). Moreover, this literature was able to flip the ecological bias and in turn show the problems with an "individualistic fallacy" that conflates context effects with individual effects (Sampson, 2012).

This type of bias is controlled in the dissertation with the inclusion of individual-level confounders when assessing the effects of context. The key compositional controls are victimization for contextual violence and household expenditure for contextual poverty. In addition, cross-level interactions help to identify the linkages between the macro and micro levels.

2.5.2 Isolation of relevant effects

The aforementioned concern with the black-box problem can be posed as the difficulty to identify, capture, and isolate the neighborhood characteristics that may be most critical to

individual outcomes, potentially leading to attenuation bias i.e. unduly weak estimates (Jencks & Mayer, 1990; Small & Newman, 2001). The issue is that the context effects are frequently defined and operationalized as neighborhoods rather than how different dimensions of residential context affect individuals and families (Sharkey & Faber, 2014). Most studies use proxies to represent neighborhood conditions, such as the poverty rate, but they might not accurately reflect the attributes that really matter and where policy interventions should be targeted (Ellen & Turner, 1997). Since many symptoms of neighborhood distress are highly correlated, it's hard to empirically differentiate their effects and might disguise different within social dynamics like social capital or civic infrastructure (Ellen & Turner, 2003). Crucial for the dissertation, the fact that context effects are bundled together complicates their isolation, for instance, between poverty, crime, and poor health.

One of the main objectives of the dissertation is to isolate the context effects and differentiate between the effects of poverty and violence. Therefore, descriptive statistics show the degree of association between them, and then the modeling strategy tests them together, in interaction, and with dummy variables that clearly differentiate their presence and absence at the municipality.

2.5.3 Selection bias

Along with ecological bias, another key methodological threat is selection bias. Mostly based on the Gautreaux project and the MTO experiment (Massey, 2013), the issue of selectivity became highly contested (Clampet-Lundquist & Massey, 2008; Sampson, 2008). The gist is that people are not randomly distributed across neighborhoods but choose them as a result of observable and unobservable characteristics that, independently of neighborhoods, may affect individual outcomes. However, failing to account for these characteristics may overstate the influence of

neighborhoods on individual outcomes (Small & Newman, 2001). There is a difficulty in distinguishing whether households choose the neighborhoods they live in and the same reasons that lead them to choose certain places may also lead them to particular individual outcomes (Sharkey & Faber, 2014). For instance, parents with low education are more likely to live in poor neighborhoods and they are also more likely to have children who drop out of school (Small & Newman, 2001).

The debate is on how to model selectivity, not as a validity threat, but as a key socio-ecological process that requires an explanation (Sampson, 2008). People make choices about the neighborhoods in which they live; neighborhoods of both origin and destination change in composition and structure due to mobility and segregation but also because people, as agents, directly change their neighborhoods, and, finally, people may be selective in relating to a local socio-spatial context (Entwisle, 2007). Residential stratification in the U.S.A is greatly determined by racial/ethnic lines and socioeconomic position measured by income and education (Sharkey & Sampson, 2010). Longitudinal research on residential selection in Chicago finds that the most common outcome was to stay in the original address and this decision is a crucial element in the reproduction of residential stratification (Sampson & Sharkey, 2008). Notably, the second most frequent outcome is people moving to the same type of neighborhood i.e. families in segregated, poor black neighborhoods changed address to a neighborhood with the same characteristics (Sampson & Sharkey, 2008). Residential selection is thus an individual decision heavily influenced by contextual characteristics. Self-selection bias is thus tricky to control with statistical variables at either level. Even experimental designs could not successfully rule out selection bias (Sampson, 2008). Randomization would have to be done at the neighborhood level as well in order to account for the processes of residential selection (Sampson, 2011).

An important methodological decision of the dissertation was to exclude from the analyses people who changed municipality between waves as to focus on the cumulative exposure to specific contexts. This decision opened the threat of selection bias, which is addressed in Appendix 1. A way to control it was first assessing its size by knowing how many people changed municipality. Then to examine what were the motives of such a movement and if they were related to the context characteristics under study. Finally, comparisons based on attrition and residential stability against the analytic sample helped to identify the profile of the subgroups that may be driving the bias.

2.5.4 Uncertain Geographic Problem

It is important to distinguish neighborhood/ community from political/ administrative demarcation and discuss if the latter is an appropriate proxy because their boundaries do not necessarily overlap (Diez-Roux, 2001). Availability of data have made neighborhoods epidemiology's first choice for "contexts" and the census tracts have been the preferred way to operationalize them (Diez-Roux, 2001). The same happens with the sociological studies (Sharkey & Faber, 2014). This decision is problematic in two ways. Geographically, social and administrative areas do not always coincide. This is why Sampson and colleagues decided to use meaningful neighborhoods for the residents of Chicago rather than politically predefined areas (Sampson, 2012). The second is that the level of aggregation is also politically predefined; the social processes within a block are different from the ones at the census track, county, or state (Hipp, 2007). The uncertain geographic problem highlights the fact that area units are arbitrarily determined and thus can be aggregated to units of different sizes or arrangements, thus potentially confounding the results (Arcaya et al., 2016). The challenge to assess the optimum level of context leads to the problem of spatial dependence across nearby places and questions the degree

up to which clustering is still relevant (Logan, 2012). For example, violence and collective efficacy in a given neighborhood are positively correlated with the same rates in the surrounding neighborhoods, finding that suggest a diffusion mechanism in which adjoining neighborhoods condition both processes in a metropolitan system (Morenoff et al., 2001). Therefore, small areas matter for some mechanisms but the macro context needs to be properly considered as well.

Defining the area and the proper level of aggregation is important as it is related with the proposed social mechanisms involved. Hipp examined the effects of using contextual predictors on individual perceptions of crime and disorder, but changed the level of aggregation from block to census track and then to county. His results show that the decision matters for the strength of the associations. Racial heterogeneity and crime rates were similar across levels of aggregation, but the effect of average income was stronger at the block level (Hipp, 2007). Nevertheless, some effects tend to have consistent effects across levels (Sampson et al., 2002); for instance, "poverty at the state, county, city, and neighborhood levels has been linked to poor health status" (Galea, Ahern, et al., 2005, p. 2420).

The use of secondary data precludes the researcher from testing the appropriate area and level of aggregation. The dissertation has to use the municipality as a proxy for neighborhood because it is the smallest area publicly accessible in the MxFLS. Moreover, most of the context variables were only available for the municipality. Even though municipalities are the smallest political entity in Mexico, they are much broader than a neighborhood that would be best characterized as a "colonia". Therefore, this problem is a limitation of the dissertation.

2.5.5 Spatial Polygamy

The challenge to define meaningful boundaries is paired with a more complex problem: the analytic dependence to a neighborhood of residence when people are frequently exposed to a

network of neighborhoods that can be studied from an egocentric perspective (Graif et al., 2014). The analytic dependence to the residence assumes that neighborhoods are like islands that contain the residents' lives and interactions and thus have constant contextual exposures. However, "the biggest critique of neighborhood effects research is the simple fact that neighborhoods are themselves penetrated by a host of external forces and contexts" (Sampson, 2011, p. 235). A more realistic assumption is that people live, work, and play in different neighborhoods (Leung & Takeuchi, 2011) but may be affected by the same municipal public policies (Galea, Freudenberg, et al., 2005).

Recent advances in HLM modeling would allow testing the effects of multiple exposures. If more detailed data were available by "colonia", cross-classified random effects models would be adequate. However, having few people changing residence from one municipality to another, fixating residence at the municipality is a tenable assumption.

2.5.6 Measurement problems

There is a need to distinguish which attributes belong to persons, networks, and neighborhoods (Portes & Vickstrom, 2011). Therefore, measures that work for persons do not directly describe ecological settings and specific measurements need to be developed in what has been called "ecometrics" (Raudenbush & Sampson, 1999b). It is crucial to create measurements that describe the full range of ecological attributes (Entwisle, 2007; Schaefer-McDaniel, Caughy, O'Campo, & Gearey, 2010).

Another source of nonrandom error is in how key variables are measured. For example, the link between SES and health outcomes may be attenuated if self-reported measures of health are used because low SES individuals are less likely to have regular contact with the medical system and thus receive fewer diagnostic tests (Matthews et al., 2010). Furthermore, even among

people with health-care, there is still a link between SES and health literacy that may confound self-reported measures (Matthews et al., 2010).

Two worrisome limitations of the dissertation are measurement problems. The first one is the elevated number of missing values in the MxFLS, especially with the biomarkers. This issue is addressed in Appendix 2. The second problem is the low-quality data on the contextual measures. The poverty and inequality indices are reliable, although the measurement of income is limited at both ends of the distribution (Teruel, 2014). However, the violence measures are not as reliable. Recent research found persuasive evidence that about one fourth of Mexico's municipalities tend to underreport and/or misclassify the intentional homicide statistics (David, Furszyfer, & Gallegos, 2017). Measurement error at both the dependent and independent variables can lead to type II statistical errors i.e. to incorrectly retain the null hypothesis and thus fail to identify an hypothesized effect (Singleton & Straits, 2010).

2.5.7 A need for longitudinal analysis

It is feasible to expect that neighborhood effects accumulate slowly, thus measuring at a single point may lead to serious measurement errors (Ellen & Turner, 2003; Massey & Brodmann, 2014). Moreover, neighborhood conditions probably impact individuals in different ways at different life-stages; for instance, the environment might exert little influence on kids because their family is the most important institution and they might be less exposed to the neighborhood effects, but this will change as they grow older with the school of choice and as other adults (teachers) increase their influence, particularly their peers during the teen years (Ellen & Turner, 2003). Albeit rare (Oakes et al., 2015), only longitudinal analyses could test a central tenet of social ecology, i.e. that neighborhood effects are lagged and cumulative exposures (Wodtke et al., 2011).

The dissertation tackles this gap in the literature by exploiting the three waves of the MxFLS by comparing between and within person changes.

2.5.8 Nonlinear and heterogeneity of effects

Many studies assume that the effect of mean SES is linear. However, these effects have different intensities along the SES continuum, with stronger effects at the low-end and nearly non-significant at the middle (Jencks & Mayer, 1990; Small & Newman, 2001). It is well-known that the influence of families over individual outcomes might be larger than neighborhood effects (Ellen & Turner, 1997), but this might conceal that the average effects of neighborhood conditions could appear small even if the effects on some subgroups are large (Jencks & Mayer, 1990). Neighborhood characteristics may be more influential for families with low socioeconomic status because they can't supplement what the environment is lacking (i.e. attending a private school). In addition, specific aspects of the neighborhood might matter more for some people than for others; for example, "girls are more influenced than boys by high rates of teen child bearing in their immediate surroundings, while boys are more likely to be influenced by neighborhood crime rates" (Ellen & Turner, 1997, p. 857). Likewise, the interactions between race and class demonstrate that the effects are not homogenous over everyone (Massey & Brodmann, 2014).

One of the main objectives in the dissertation is to examine the heterogeneity of effects in multiple populations. Interactions among the independent variables also shed some light and the different intensities of the context effects.

2.5.9 Experimental designs

Previous research on context effects has mostly relied in observational studies and a few experimental designs, most notably the Moving to Opportunity Study, but both types of designs

have yielded mixed findings (Clampet-Lundquist & Massey, 2008; Sampson, 2008). Static-models and the problem with selection bias to the neighborhood has favored an experiments-only position (Sharkey & Faber, 2014) even though it does not solve several issues as the relevant unit of analysis, generalizability to other populations, the frequent short-term measures, or the "black-box" problem (Wodtke et al., 2011). A systematic review concluded that, in spite of an ever-increasing HLM articles after the year 2000 on several health outcomes, studies based on observational designs typically have small significant effects, but fundamental design flaws (Oakes et al., 2015). Randomized and natural experiments do a little better: "Overall, despite methodological advances and the great advantage of the design to assess neighborhood effects, the effect of neighborhood interventions on health remain modest, if not nonreplicable, at best" (Oakes et al., 2015, p. 80).

Population health interventions are not always amenable to experimental designs, so natural experiments with quasi-experimental designs are attractive options (Craig, Katikireddi, Leyland, & Popham, 2017). Among them, difference-in difference models, as the one in the dissertation, are a common approach. Its major strength is that it controls for observed and unobserved differences in time-invariant attributes and is therefore less prone to omitted variable bias (Craig et al., 2017).

The methodological issues discussed above are far from solved and clearly exceed the objectives of the dissertation. However, intermediate solutions, as the ones offered by Sharkey and Faber (2014) to advance the field, include the use of a flexible conception and measurement of residential contexts because there is not a one-size-fits-all solution. They also argue for a relevant timeframe that adopts a life-course perspective that acknowledges the duration, accumulation, and persistence of exposure to disadvantaged and advantaged environments over time. Finally, they advocate for research that assumes heterogeneous responses to neighborhood

effects because there are different levels of exposures and differential vulnerabilities among population subgroups (e.g. age, race, or gender). The analyses of the dissertation incorporate the flexible approach. The rationale is that, rather than wait for ideal methodological conditions, it is preferable to advance the debate with preliminary results.

Michael Oakes is one of the public health researchers with the harshest methodological critiques to the context effects literature. At the end of his last review, he rhetorically asks if epidemiologists should devote more time and resources to context effects. He answers: "perhaps ironically, our answer is a resounding yes". He claims that estimating the impact of biosocial contexts on health is a worthwhile endeavor. However, he argues that the problem lies elsewhere, with the very definition of a neighborhood effect and its alleged threats to identify it:

> The basic idea that biosocial contexts affect health is patently obvious. It is the idea of an independent, separate, neighborhood effect that seems foolish. The error lies in efforts to disentangle people from places. Paraphrasing Macintyre and Ellaway, the fact is that neighborhoods make people, and people make neighborhoods. It is not composition or context, but composition and contexts. Efforts to disentangle mutually constitutive, inextricably linked, synergistic, and coevolving elements are doomed to fail (Oakes et al., 2015, p. 85).

Chapter III. Towards an integrated theory of context effects and health

3.1 Integrated Health Factors

3.1.1 Proximal Risk factors of Cardiovascular Disease

A key development in cardiovascular research was the identification of risk factors associated with CVD that help shape treatments to abate it (Kreatsoulas & Anand, 2010). Experts in CVD already expressed reluctance in placing exclusive emphasis on short-term risk estimates when in 10 years people may actually be at a higher risk because any untreated single factor can produce cumulative damage (Lloyd-Jones et al., 2004). This type of research found that key risk factors for developing CVD are blood pressure, cholesterol (TC, HDL-C, LDL-C), obesity, and cigarette smoking, as well as sociodemographic characteristics like age, gender (males), and having a family history of CVD (Wilson et al., 1998).

Another valuable finding for the identification of CVD and type-2 diabetes was the co-occurrence of these risk factors. The risk factors present themselves together and tend to cluster, suggesting that they are not independent of one another but may share underlying causes and mechanisms (Huang, 2009). These clusters are fundamental to identify at-risk patients and to design preventive treatments, but also to understand the mechanisms linking them together and with CVD (Alberti, Zimmet, & Shaw, 2006; Huang, 2009).

Two fundamental cohort-prospective investigations that greatly contributed to finding the clustering of proximal and distal CVD risk factors are, respectively, the Framingham study in the United States (Kannel, Feinleib, McNamara, Garrison, & Castelli, 1979) and the Whitehall study in England (Marmot, Shipley, & Rose, 1984).

A successful outcome from the Framingham study was the identification of a cluster of biomarkers for CVD risk that was easily transformed into a calibrated index or score. The score is comprised by age, total cholesterol, HDL cholesterol, blood pressure, diabetes, and smoking

(Wilson et al., 1998). The authors' approach sought to emphasize the "established, powerful, independent, and biologically important factors" involved in CVD, so they left out family history for heart disease, physical activity, and obesity because "these factors work to a large extent through the major risk factors, and their unique contribution to CHD prediction can be difficult to quantify" (Wilson et al., 1998, p. 1837). The Framingham score proved very effective in predicting long-term individual cumulative risk of CVD even in the context of competing risk of death from non-coronary causes (Lloyd-Jones et al., 2004). Overall, the Framingham score is well calibrated to predict first coronary events in populations from the United States, Australia, and New Zealand (Eichler, Puhan, Steurer, & Bachmann, 2007).

The Framingham score served as a blueprint for similar indices that aimed to use fewer biomarkers but yield more accurate predictions. After a systematic review of multiple risk scores of cardiovascular diseases developed from healthy adult populations, a research group defined a new risk equation that can be used in several countries: the Globorisk. They chose the factors that were more easily measured and with less cost, and thus the more common in low-income countries. The most predictive risk-factors were systolic blood pressure, total cholesterol, diabetes, and smoking, while age and sex are also weighted (Hajifathalian et al., 2015). Following the between country social gradient of health, low and middle-income countries had increased prevalence of people with high 10-year risk of fatal CVD when compared with high-income countries. Notably, Mexico was the second country with the highest risk prevalence: 15% for men and 11% for women (Hajifathalian et al., 2015).

The Framingham score and similar indices are oftentimes considered as a syndrome. A syndrome is a "recognizable complex of symptoms and physical or biochemical findings for which a direct cause is not understood" (Alberti et al., 2006, p. 473). In the Framingham score, the frequent clustering of risk factors suggests shared underlying causes. However, since causal

mechanisms were not yet identified, it was not understood as a disease. A similar cluster of risk factors was identified in the metabolic syndrome (MetS) or syndrome X, which also directly promotes CVD and type 2 diabetes (Grundy, 2005).

The main risk factors of MetS are abdominal obesity and insulin resistance, but the underlying cause was also elusive. Several measures and thresholds to measure MetS have been proposed. However, the Adult Treatment Panel III (ATP III), a consensus group, defined MetS as abdominal obesity measured with waist circumference (or BMI>30), and *any two* of the following: raised tryglicerides, reduced HDL-cholesterol, raised blood pressure (systolic or diastolic), raised fasting plasma glucose or previously diagnosed type-2 diabetes (Alberti et al., 2006). Since insulin resistance presents important challenges for an accurate measurement, it is considered that abdominal obesity already incorporates it and thus is the only required element for the diagnosis of MetS. An updated classification indicated increased waist circumference may not be necessary if 3 out of the 5 other risk factor criteria are present (Grundy, 2005). Nonetheless, while blood pressure is the common factor in all the previous indices, abdominal obesity measured with waist circumference is the form of obesity most strongly associated with the metabolic syndrome (Grundy et al., 2004).

In Mexico, MetS is considered a major health problem due to its association with ischemic disease and diabetes mellitus. The prevalence for MetS was 26.6% but it was more common in men and varies between 10% and 30% among an age range of 20-69 years old (Aguilar-Salinas et al., 2004). Moreover, with MetS the relative risk of having a cardiovascular event is 2.7 (CI 1.2-6.2) (Aguilar-Salinas et al., 2004).

3.1.2. Distal risk factors of Cardiovascular Disease

While the Framingham study was most useful to identify proximate risk factors, the Whitehall study was better at locating distal predictors of CVD, *the causes of causes,* because it used socioeconomic status as a risk factor in its own right and not only as a confounding variable (Brunner, 2016). Rose and Marmot (1981) argue there is a social class gradient of coronary heart disease by showing how the prevalence was lower in the highest employment grade when compared with the lowest grade. In addition, they noted how "a man's employment status was a stronger predictor of his risk of dying from coronary heart disease than any of the more familiar risk factors" (Geoffrey Rose & Marmot, 1981). Notably, 10 years after the first examination, the Whitehall II study found no decrease in the social class difference between employment grade and prevalence of angina or ischaemia (Marmot et al., 1991). Further research on the biological basis of social inequalities regarding coronary risk found that central obesity, components of the metabolic syndrome, and plasma fibrinogen are inversely associated with socioeconomic status (Brunner et al., 1997). When tested longitudinally, metabolic syndrome variables, inflammatory markers, and health behaviors accounted for half of the social gradient (Marmot, Shipley, Hemingway, Head, & Brunner, 2008). Moreover, a review confirmed with strong evidence that low socioeconomic position is associated with a poorer biomarker profile of CVD and diabetes, especially for women (Kavanagh et al., 2010).

Marmot and colleagues (2008) specify three types of influences over the biological pathways to the metabolic syndrome: health behaviors, early life, and psychosocial factors. The first ones are the well-known proximal risk factors such as diet, physical activity, smoking, and alcohol intake, although the metabolic syndrome variables mediated the effect of social position independently of these behaviors. Adverse socioeconomic circumstances in childhood are also important determinants of CVD risk (Galobardes, Smith, & Lynch, 2006). These circumstances

range from childhood illness to fetal exposures and to poor maternal nutrition (Kavanagh et al., 2010).

Marmot's team also advanced the idea that psychosocial stress activates the hypothalamic-pituitary-adrenal axis, which is linked to central obesity, metabolic syndrome, and CVD risk (Marmot et al., 2008). Psychological stress happens when an "individual perceives that environmental demands tax or exceeds his or her adaptive capacity" (Cohen, Janicki-Deverts, & Miller, 2007, p. 1685). A review in *The Lancet* found that "stress is clearly an important –and potentially modifiable– risk factor for acute and chronic adverse cardiovascular disorders" (Brotman, Golden, & Wittstein, 2007, p. 1096). The cardiovascular effects of stress have been assayed as acute physical stressors such as trauma or physical exertion. Nonetheless, emotional stressors like anger, fear, surprise, and severe grief are also precipitants of cardiovascular events (Brotman et al., 2007). Another review found that stress, anger, and depressed mood act as important triggers of major cardiac events: "the pooled relative risk of acute coronary syndrome onset being preceded by stress is 2.5 (1.8-3.5)" (Steptoe & Kivimaki, 2013, p. 337). Notably, as with the subsequent fear to 9-11, "major life changes associated with psychological and emotional adjustment are associated with an increased risk of cardiac events, in many cases with a magnitude of association similar to traditional cardiovascular risk factors" (Brotman et al., 2007, p. 1091). In addition to studies of animal response, laboratory experiments and prospective investigations with healthy adults provide strong support for considering stress as a "causative factor" of disease onset and progression, particularly for depression and CVD (Cohen et al., 2007).

Acute stressors –such as earthquakes– increase cardiovascular risk, but chronic stressors like job stress, marital discord, or perceptions of discriminations are also associated with worsening health behaviors and in terms of risk biomarkers (Dimsdale, 2008). Besides the

episodic negative affective states that exert direct effects on biological processes, exposures to chronic stress are considered "the most toxic because they are most likely to result in long-term or permanent changes in the emotional, physiological, and behavioral responses that influence susceptibility to and course of disease" (Cohen et al., 2007, p. 1685). Even though individuals are not equally susceptible to stress-mediated cardiovascular events, stress of daily living can also increase its risk.

Cumulative stress associated with social circumstances exerts an important toll in CVD. Early-life stressors like childhood abuse or poverty are linked to higher cardiovascular morbidity in adulthood (Steptoe & Kivimaki, 2013). Likewise, components of the metabolic syndrome have been associated with chronic psychological stress, including depression, chronic anxiety, and sleep deprivation, and with conditions such as low employment grade, low socioeconomic status, and persistent stress at work (Brotman et al., 2007). For example, the demand-control model explained differential work experiences where low-income work characterized by high demands but low control meant high strain or stressful work (Brunner, 2016). When a variation of the Framingham risk score was used as a measure of stress, it was demonstrated that job strains were strongly related to employees' CVD risks and sleep deprivation (Berkman, Buxton, Ertel, & Okechukwu, 2010). In this important study, the chosen categories for the CVD risk index were: current smoking (self-reported), obesity (self-reported BMI), high blood pressure for hypertension (wrist blood pressure monitor), high total cholesterol (blood samples), and high glycosylated hemoglobin for diabetes (blood samples). In contrast with the original Framingham risk score, Berkman and colleagues only used the presence of two or more CVD risk factors; omit age and HDL cholesterol; use glycosylated hemoglobin in lieu of self-reported diabetes; and add obesity as BMI. This index is used in the dissertation with the "CVDR" acronym.

As with job stress, poverty began to be conceptualized as a condition of chronic stress and was considered to have both indirect effects on illness through behaviors, and direct effects via biological changes; it's "the idea that chronic stress may change short term reactivity and baseline resting set points in autonomic, neuroendocrine or metabolic pathways" (Brunner, 2016, p. 8). Regardless of the evidence supporting the link between SES and risks of CVD (Aiello & Kaplan, 2009), not all studies found this association (Dowd & Goldman, 2006). Nonetheless, the "link between social adversity and stress has been strongest in relation to cardiometabolic diseases. Thus, studies have frequently considered social (e.g. SES) and psychological (e.g., depression) exposures in relation to major cardiovascular risk factors" (Kubzansky, Seeman, & Glymour, 2014, p. 527). A recent review found that an important mechanism for intergenerational poverty was how early-childhood adversities associated with the effects of social structures impact children's bodily systems and brain development through recurrent stress (McEwen & McEwen, 2017).

Once the agreement on the link between cumulative stress due to poverty and poor health reached a consensus, two debates arose: the timing of exposure and the causality. At least two sides differed on the type and timing of the poverty event, on whether it is a cumulative exposure or the exposure on a sensitive period; in other words, between the "accumulation of physiologic damage over time and those that focus on developmental timing on when exposures occur, positing a special importance of exposures during a particular (typically early) life stage" (Kubzansky et al., 2014, p. 516). While the distinction is not mutually exclusive it does matter for policy interventions. If social disadvantage gets under the skin during a critical period in early life, then resources on these stages may have better long-term returns; but accumulation models can still support valuable interventions along the life-course (Kubzansky et al., 2014).

In addition, there is an interesting debate on whether health predicts social mobility or socioeconomic status influences health. The association between environments, behaviors, and affective processes is characterized by feedback loops (Kubzansky et al., 2014). However, a longitudinal analysis suggests that the health to poverty hypothesis works at younger ages and the poverty to health hypothesis contributes to socioeconomic differences in cardiometabolic health in midlife (Elovainio et al., 2011). Nonetheless, "while existing evidence is largely observational (and much is cross-sectional), findings are consistent with the hypothesis that there is indeed a causal relationship *from* adversity *to* health, rather than from health to social conditions" (Kubzansky et al., 2014, p. 548).

The most recent innovative research on this field connects with the social epidemiology approach since adverse contexts are starting to be considered as important psychosocial stressors (Leal & Chaix, 2011). A fascinating study found that stress, trauma, and starvation during the Leningrad siege (1941-44) raised systolic and diastolic blood pressure even three decades after the event, and the survivors had increased mortality from cardiovascular disease when compared with Russians from other cities (Sparen et al., 2004). The recent approach assesses how much of individual-level health disparities are conditioned by neighborhood contexts (King, Morenoff, & House, 2011). Following this line of research, several studies find an association between contextual poverty and individual risk factors of CVD (Bird et al., 2010; Chaikiat, Li, Bennet, & Sundquist, 2012; Diez Roux & Mair, 2010; Diez Roux et al., 2001; Merkin et al., 2009), especially with hypertension (Mujahid, Roux, Cooper, Shea, & Williams, 2011; Subramanyam et al., 2013). The impacts of violence receive less attention than poverty but exposure to it has been associated with heart disease (Sundquist et al., 2006), lower self-rated health (Boynton-Jarrett, Ryan, Berkman, & Wright, 2008), higher rates of depression (Curry, Latkin, & Davey-Rothwell, 2008; Zinzow et al., 2009), and cumulative biological risk factors (King et al., 2011). For

instance, in Sweden, in neighborhoods with the highest rates of violent crime, the odds ratios for the risk of coronary heart disease were 1.75 for women and 1.39 for men. Likewise, in the neighborhoods with the highest unemployment rates, the odds ratios were 2.05 and 1.5, respectively (Sundquist et al., 2006).

An associated line research has included other environmental stressors. Notable examples considered as protective contexts examined social capital in improving health outcomes (Kawachi et al., 2013; Lochner, Kawachi, Brennan, & Buka, 2003) and collective efficacy was an important contextual protective factor for CVD risk (Cohen, Finch, Bower, & Sastry, 2006; Leal & Chaix, 2011). In spite of these improvements, currently the "evidence base is largely insufficient to distinguish underlying pathophysiologic processes of one type of social adversity from another" (Kubzansky et al., 2014, p. 513).

3.1.3 Mechanisms from stress to cardiovascular disease: Allostatic load

A broader and refined conceptualization of these risk clusters led to a multisystem perspective for the "wear and tear" of the body due to stress exposure. Allostatic Load (AL) is a way to conceptualize the biological response to stress. AL refers to a multi-system view of the cumulative physiological toll exacted on the body over the course of a lifetime (Seeman, Epel, Gruenewald, Karlamangla, & McEwen, 2010). Allostasis is the capacity to achieve stability through change, it is the regulatory process required to cope with the environment and preserve physiological stability (McEwen & Wingfield, 2003). The premise of AL is that these short-term fluctuations are necessary for the body to work properly, but excessive fluctuations in terms of the extent, duration, or frequency (i.e. perceived danger everywhere) may result in a wear and tear on the body's regulatory systems (Seeman et al., 2010). The body thus responds to its

environment “as a living organism so that changes in one system trigger disruptions in other systems, which either singly or in combination may initiate physiological processes” (Kubzansky et al., 2014, p. 513).

Biological embedding of social adversity occurs trough pathways on candidate systems. These systems have physiological functions that work as transducers between social environments and human biology with the capacity to embed and influence the life course development (Hertzman & Boyce, 2010). Candidate systems have four characteristics: 1) they are influenced by daily experiences, especially early in life; 2) the systems respond to these influences throughout the life course; 3) if dysfunctional, the systems influence health, well-being, learning and behavior; and 4) there is a differential functioning of the systems across the life course (Hertzman & Boyce, 2010). Seeman and colleagues (2010) have demonstrated that the major biological regulatory systems that react to stress are: cardiovascular, metabolic, inflammatory, hypothalamic-pituitary-adrenal (HPA) axis, and the autonomic, sympathetic, and parasympathetic nervous systems.

One of the strengths of AL lies in the fact that single indicators of single systems are not strong predictors of poor health outcomes but they matter more when aggregated as an index (Seeman et al., 2010; Seeman, Singer, Rowe, Horwitz, & McEwen, 1997). However, a key difficulty to operationalize AL is choosing which indicators in which systems should be included in the index (Juster, McEwen, & Lupien, 2010). Notably, however, not even the original index of AL includes measures for all the systems. Based on the MacArthur Studies of Successful Aging (age 70-79 years), the contents of the original index were blood pressure, waist-hip ratio, total cholesterol-HDL ratio, urinary cortisol, urinary epinephrine, HDL cholesterol, and DHEA-S (Seeman et al., 1997; Seeman, McEwen, Rowe, & Singer, 2001).

The original index has several limitations. It is based on a sample of older adults that does not represent other population groups and all items are assumed to weight equally in a monolithic reaction against stress. Moreover, it includes several indicators for some systems, as the metabolic system, but it omits the inflammatory system altogether. Even though the original conceptualization of AL encompasses four types of biological reactions to stress, "our measure of AL reflects only one of the two aspects of physiological activity postulated to contribute to AL, namely, higher, chronic, steady-state levels of activity related to the diurnal variation as well as any residual activity reflecting chronic stress or failure to shut off responses to acute stressors" (McEwen & Seeman, 1999, p. 37). Therefore, the original index is not a gold standard and partially measures the concept of AL. Due to these limitations, Seeman and colleagues admit that "it is important to note that this original set of 10 parameters was not meant to be comprehensive nor was offered as a fixed/standard measure of AL, rather it was an initial attempt to operationalize AL using available data" (2010, p. 228). Instead, they favor an inclusive approach in which AL reflects the "overall total cumulative burden of physiological dysregulation in allostatic processes across as many regulatory systems as possible" (2010, p. 229). Recent attempts to operationalize AL include 24 biomarkers from 7 different physiological systems (Gruenewald et al., 2012).

In spite of the multiple ways to operationalize, AL has been a powerful measure to assess stress impact. It is a concept that reflects the detrimental consequences of chronic or repeated stress exposure and can result in weathering of the body (Karlamangla, Singer, McEwen, Rowe, & Seeman, 2002), telomere shortening (Epel et al., 2004), and premature aging (Juster et al., 2010). A few studies deserve special attention because they link cumulative risk contextual exposures, among them poverty and violence, with AL (Evans, 2003; Evans & Kim, 2012). AL is thus a pre-disease indicator of physical and mental health and is associated with lower

socioeconomic status, lifestyle, and stress exposure (Beckie, 2012). Due to known system interconnections, AL offers a more realistic assessment of stress impacts because single biomarkers may suggest low levels of risk but cumulated across multiple physiological systems may have significant impacts on health risks (Seeman et al., 2010).

3.1.4 Allostatic load: a cascade of events

There has been an important effort to identify the biological pathways that link social stressors with cardiovascular disease (Seeman et al., 2001). However, the way AL is usually measured implies that the components were not organized to reflect its place or importance in the causal chain that leads from allostasis to allostatic load (McEwen & Seeman, 1999). A key feature of AL is that multiple mediators of adaptation are involved and interconnected in a non-linear network, which regulates each mediator system, often in reciprocal fashion (Juster et al., 2010). Further explanations deepened into the mechanisms involved as described by McEwen. The cascade of events then suggests that primary mediators between environment and disease lead to primary effects and then to secondary outcomes, which lead, finally, to tertiary outcomes that represent actual diseases. Figure 2 synthesizes the cascade of events leading to disease; it flows in the following fashion (McEwen & Seeman, 1999):

1. When facing real or perceived stress or allostatic challenges, the first systems involved are the neuroendocrine and inflammatory. The primary mediators are the chemical messengers, the hormonal component of the AL index. Prolonged secretion of the stress hormones epinephrine, norepinephrine, and cortisol (antagonized by dehydroepiandosterone, DHEA) can fail to protect a distressed individual and damage the brain. Synergistic effects of these molecules exert primary effects on cellular activities that

compromise the physiological integrity of allostatic mechanisms (Juster et al., 2010). Particularly catecholamines (adrenalin and noradrenalin) produce widespread effects throughout the body, like the acceleration of the heart rate or the enhancement of fear-related memory formation (McEwen & Seeman, 1999). Thus, stress hormones and their antagonists, as well as inflammatory cytokines, represent primary mediators of AL. The primary effects are cellular events and structural proteins targeting specific organs but these processes remain understudied (McEwen & Seeman, 1999).

2. The secondary outcomes are integrated processes that reflect the cumulative outcome of the primary effects, often reflecting the actions of more than one primary mediator (McEwen & Seeman, 1999). Over time, subsidiary biological systems compensate for the over or under protection of primary mediators and in turn shift their operating ranges to maintain chemical, tissue, and organ functions (Juster et al., 2010). In this level, the secondary outcomes appear when parameters of three systems reach subclinical levels: the metabolic system (i.e. insulin, glucose, TC, HDL, triglycerides, and visceral fat depositing), the cardiovascular system (i.e. blood pressure), and the immune system (i.e. fibrinogen, c-reactive protein). Blood pressure is particularly interesting because it may be a primary mediator or a secondary outcome; it is part of "the pathophysiological pathway of the metabolic syndrome, but it is also a more primary indication of the allostatic load that can lead to accelerated atherosclerosis as well as insulin resistance" (McEwen & Seeman, 1999, p. 40).

3. The final stage is allostatic overload. It is the culmination of physiological dysregulation, which leads to disease, also referred to as tertiary outcomes (Juster et al., 2010). The idea

behind AL is to have a pre-disease indicator of tertiary outcomes, such as CVD, decreased physical capacity, cognitive decline, and all-cause mortality (Beckie, 2012).

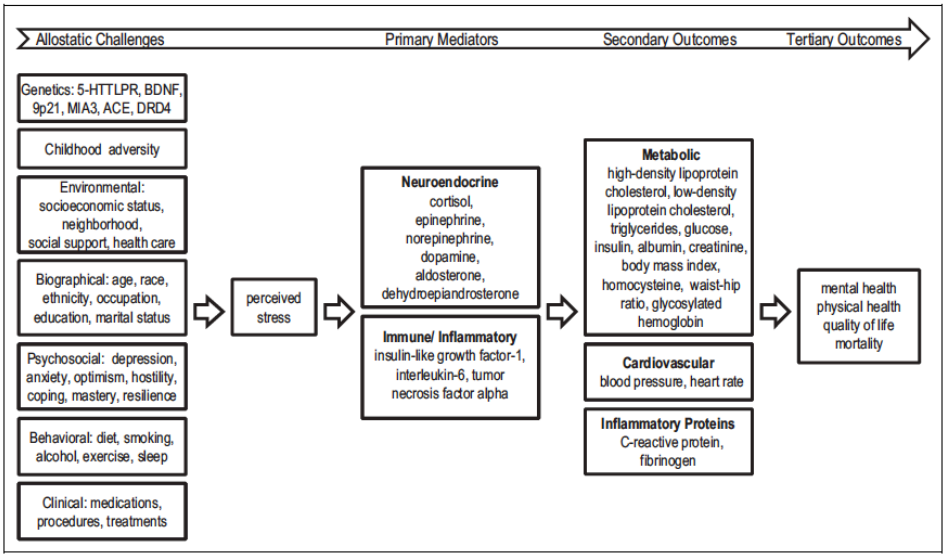


Figure 1. Heuristic model: allostatic load, health and health disparities.

Figure 2. Cascade of events mediating between stressful events or allostatic challenges and disease or deleterious outcomes. The source is Beckie (2012, p. 312).

The cascade of events is still preliminary and several challenges remain (McEwen & McEwen, 2017). For instance, most researchers have yet to clarify if the cascade of events can be measured separately or if they need to be bundled together (McEwen & Seeman, 1999, p. 41). Nevertheless, these mechanisms help to frame what processes is the Framingham score tapping into. The Framingham score, as well as the single biomarkers, are secondary outcomes of the cardiovascular and metabolic systems. The Framingham score and associated measures are thus partial pre-disease indicators of the tertiary outcomes. The unobserved but inferred reactions are the primary mediators occurring in the immune and neuroendocrine systems.

This framework also suggests the limitations of the measures in the dissertation. Single biomarkers or partial indices fail to capture the multisystem nature of AL and may not reflect the wear and tear of the body. AL is partly confounded by CVDR factors that also constitute MetS

(Seeman et al., 2001). However, in essence, AL seeks to move beyond CVDR factors in an effort to include more systems and indicators. A review of 58 studies using AL found great heterogeneity in the operationalization of the index although most included some indicator of primary mediators and secondary outcomes (Juster et al., 2010). Likewise, Beckie (2012) highlights that measurement issues are still the weakest link in the AL framework but, despite the lack of consensus on which indices of AL are necessary and sufficient for its measurement, she notices the centrality of neuroendocrine functioning to the operationalization of AL. Isolating CVDR factors, as in the Framingham risk score or even worse, with single biomarkers, would not reflect the multisystem view of AL. The dominance of CVDR factors over the AL summary measure is only due to initial data availability but is still far from the ideal measure. As a literature review on the topic concluded: "none of the individual biomarkers comprising the AL index significantly explained these tertiary outcomes, supporting the utility of a multi-systemic composite approach to predicting tertiary outcomes in geriatric populations" (Juster et al., 2010, p. 4).

Consequently, a common cautionary practice to avoid overuse of the term AL has become to name such indexes as cumulative biological risk profiles and use comprehensive measures or sub-indices for either cardiovascular, metabolic and inflammatory risks (Bird et al., 2010; King et al., 2011; Seeman et al., 2008). The available data in the MxFLS is not enough for an index of AL. Following this guideline, the dissertation uses CVDR indices and single biomarkers. These indices highlight the use of indirect indicators of the underlying processes guiding AL. The rationale was to assess secondary outcomes of AL and examine the intermediate step in the "cascade of events". At the same time, the use of a measure of CVDR may be interpreted as a proxy for secondary outcomes of AL and a (limited) measure of stress on its own right. While acknowledging the considerable overlap of AL with these risk profiles, the available secondary

outcomes “reflect adaptive physiological responses to stress and other adverse stimuli but which also can arise from other etiologies” (King et al., 2011, p. 572).

3.2 Integrated theoretical framework

3.2.1 Social Ecology: lessons learned

In contemporary societies, the fundamental social settings for human growth and development are family, school, peer group, and neighborhood (Bronfenbrenner, 1989). For humans to reach full adult potential, these four social spheres must provide access to key material, symbolic and emotional resources (Massey & Brodmann, 2014). Whereas critical material resources such as wealth and income determine access to food, shelter, medicine, or education, and symbolic resources such as status and prestige garner esteem and social standing, it is emotional resources like affection and safety that are crucial for wellbeing and healthy social maturation. Unfortunately, none of these resources is equally distributed and, thus, development and overall wellbeing is stratified along the life cycle (Massey & Brodmann, 2014).

The places or contexts where people strive are shaped by the distribution of these resources and therefore have been an important object of study for the social sciences (Giddens, 1984). This branch within Sociology is best described as “Social Ecology” which studies the interaction between human beings and their environment and examines how exposure to different social contexts influences human development and action (Wikström, 2009). In this literature, a context is frequently understood as a neighborhood: a spatially delimited segment of the city of which its character is defined by its people and infrastructure and such segments are spatially differentiated on the basis of power, wealth, religion, and ethnicity (Massey & Brodmann, 2014). Moreover, a neighborhood effect can be defined as the independent causal effect of a residential community on any social or health outcome (Jencks & Mayer, 1990). Key tenets of Social Ecology are that: 1) there is considerable social inequality between neighborhoods in terms of socioeconomic and racial segregation (Wilson, 2012); 2) a number of social

problems tend to come bundled together at the neighborhood level and have cumulative and mutually reinforcing effects on the wellbeing of its residents (Massey, 1990) but the concentration of disadvantage makes it difficult to discern which neighborhood characteristics affect people's life chances and which do not (Small & Newman, 2001); and 3) social dynamics within neighborhoods can mediate the relationship between concentrated disadvantage and individual level outcomes and thus cross-level interactions require careful examination (Sampson et al., 1997; Sharkey & Faber, 2014). These insights matter because "if numerous and seemingly disparate outcomes are linked together empirically across neighborhoods and are predicted by similar structural characteristics, there may be common underlying causes" (Sampson et al., 2002, p. 447). This is the rationale by which the clustering by neighborhood of social disadvantages and health risks merits examination.

Ecological models in Sociology have a corresponding history in Public Health. Social epidemiology focuses on exposures rather than in specific diseases and studies social phenomena, as socioeconomic stratification, and outcomes as functional status and well-being (Berkman & Kawachi, 2014). This branch of epidemiology stems from the convergence of a more nuanced understanding of population distributions of health rather than individual risk factors (Rose, 2001) and from an emphasis on the distal or fundamental causes of disease, particularly the lack of resources as money, power, knowledge, or social networks (Link & Phelan, 1995). Likewise, it favors the life-course approach as means to understand the long-term outcomes of social exposures to assess cumulative risks (Ben-Shlomo & Kuh, 2002). It emphasizes how exposures may change in nature, importance, and intensity and thus shape the sensitive or critical periods where it has its more profound results (Gee et al., 2012). Importantly, ecosocial models try to explain how people literally embody their lived experience in social contexts (Krieger, 2011b). These contexts tend to concentrate disadvantages and its interactions create different and more pernicious expressions of disease, in what has been called syndemics (Singer et al., 2017). Even though the biosocial approach requires a new way to understand causality

(Hertzman & Boyce, 2010), ecological health interventions have shifted focus accordingly from efforts to change individuals to attempts to shape norms or provide opportunities to engage in certain behaviors as effective coping strategies (Berkman & Kawachi, 2014).

Two fundamental reasons why ecological theories were not fully developed until recently was the black-box problem where macro-micro mechanisms were not clearly specified (Mayer & Jencks, 1989) and the ecological fallacy i.e. the problematic results of making individual-level inferences on the basis of aggregate data (Bursik, 1988). In other words, there is a need to disentangle the effects of neighbors (composition) from the neighborhood (context) and to identify the differences between the effects of social interactions, the built environment, and institutions, as well as the heterogeneous impact they may have in different populations (Duncan et al., 1998; Ellen & Turner, 2003). A key feature for the development of ecological models was the increasing availability of statistical methods, most notably HLM, which helps to control for attributes at both levels (Diez Roux & Mair, 2010; Raudenbush & Bryk, 2002). Such a possibility fueled efforts to specify the social mechanisms involved in ecological theories by way of assessing cross-level interactions (Galster, 2012; Mayer & Jencks, 1989; Sampson et al., 2002).

Improvements on assessing context effects are not accompanied by corresponding evidence supporting the theoretical claims on the social mechanisms that explain specific outcomes (Sharkey & Faber, 2014). Nevertheless, there is important consensus that stress is a fundamental mediator in the process by which concentrated disadvantages get under the skin (Burgard & Kalousova, 2015; Hertzman & Boyce, 2010). A remaining dispute in both fields is the possibility to isolate the effects of concentrated disadvantages and identify distinct mechanisms, for instance, the ones that differentiate exposure to violence or poverty.

3.2.2 Contexts as vulnerabilities and capacities

An important trend in Social Epidemiology is to understand how cities influence health and well-being, especially because cities have both positive (i.e. access to social and health care) and negative (i.e. pollution) effects (Galea, Freudenberg, et al., 2005). Sandro Galea and colleagues propose a framework to appreciate the specificity of urban contexts that allows for the identification of pathways by which changes in the urban context affect health. Their underlying assumption is that the urban environment in its broadest sense (physical, social, economic, and political) affects all residents either directly or indirectly (Galea, Freudenberg, et al., 2005). It posits that urban populations are defined by size, density, diversity, and complexity and urban population health is a function of the living conditions shaped by global, national, and municipal determinants (Galea, Freudenberg, et al., 2005). While nested on enduring social structures (economic systems and types of governments) examples of the major global and national trends that affect urban health are migration and suburbanization. More proximal influences of health are the municipal level determinants, particularly the local governments my means of their policies (urban development and housing) and the provision of services (transportation and health care services). In addition, municipal determinants are linked to markets that allocate employment, food, or medical care, and also to the role of civil society that may provide social support, for example, through community-based organizations or even churches. Finally, the urban living conditions are the population's demographic characteristics (individual SES, race, attitudes, behaviors), the built environment, the social environment made up by social networks, and formal or informal health and social services (Galea, Freudenberg, et al., 2005).

This framework suggests unique patterns of health and disease that may be expressed in terms of differential capacities and vulnerabilities of populations (Ahern et al., 2008; Galea, Ahern, et al., 2005). In a similar claim as Hertzman and Boyce (2010), the authors argue that human populations are complex systems and its study implies that a single exposure (like poverty) may cause several

effects and that changes in exposures do not always yield monotonic changes in disease, as with the mental health consequences of a disaster, that remain beyond the removal of the exposure (Galea, Ahern, et al., 2005). The framework then posits that the health of populations reflects the interrelationship between underlying vulnerabilities and capacities and the intermittent stressors and protective events (Ahern et al., 2008). Vulnerabilities are considered conditions with the potential to harm an individual or system, for example, the lack of resources or poverty in a given population. Conversely, capacities are characteristics that confer protections on individuals or populations, such as abundant resources or social capital (Galea, Ahern, et al., 2005). On the other hand, intermittent stressors are destabilizing events like a natural disaster or the closure of a large employer while a protective event is an intermittent stabilizing occurrence as the opening of a school.

"Importantly, the intermittent influences interact with the underlying conditions to shape health at any particular moment" (Galea, Ahern, et al., 2005, p. 2418). This means that disease variability reflects the impact of external stressors in an attempt to maintain homeostasis because systems at the boundary of their tolerance are more vulnerable to small differences in circumstances than systems without such stress. In more concrete terms, intermittent stressors will result in greater variability in outcomes among vulnerable populations and they will be more stable in more resilient populations: "a geographically isolated community characterized by limited employment opportunities may not cope as well with the sudden departure of a major employer as a community where employment opportunities are abundant" (Galea, Ahern, et al., 2005, p. 2419). A good example is how these authors found much higher variance in homicide rates among low-income neighborhoods than in more affluent ones (Ahern et al., 2008).

3.2.3 Heterogeneity of contextual effects

Frameworks of this sort have moved the public health field from investigating the association of poverty and health to its mediators and, among them, an important pathway is through emotions and the physiological, cognitive, and behavioral responses they evoke (Kubzansky, Winning, & Kawachi, 2014). Ecological perspectives prove that individuals are embedded within social structures that determine exposure to stress, the available stress mediators, and shape the perceptions or appraisals of stress (Stockdale et al., 2007). These new models suggest reinforcing mechanisms between neighborhoods and health inequalities that are mediated by personal behaviors and subjective perceptions with differential sensitivities to stress (Diez Roux & Mair, 2010).

Among these, the reserve-capacity model proposes that a leading pathway between poverty and health is the intensity of exposure to stress, where poor environments are associated with greater exposure to frequent and intense harmful or threatening situations in the form of negative events and fewer beneficial events (Matthews et al., 2010). These authors theorize that individuals with low socioeconomic status have a smaller stock of resources (tangible, interpersonal, and intrapersonal) to deal with stressful events and their reserve capacity to cope with the negative events may be diminished because they face more situations that deplete their resources and also have fewer resources to replenish them (Matthews et al., 2010). From a life-course perspective, the cumulative effects imply that "once an individual has been exposed to stress, resources tend to deteriorate, leaving individuals more vulnerable to future strains" (Matthews et al., 2010, p. 147). Likewise, other models show that neighborhood disadvantage functions as a stressor that interacts with several individual-level stressors and buffers. For instance, Stockdale et al (2007) found cross-level interactions in which violence-exposed individuals were vulnerable to depressive/anxiety disorders while individuals with low social support, nested in neighborhoods with high social isolation, had a higher likelihood of alcohol, drug, and mental health disorders.

3.2.4 Empirical model of context effects on cardiometabolic risk biomarkers

The previous framework guides the empirical examination of the dissertation´s hypothesis. As figure 3 displays, the framework follows a multilevel structure that aims to represent the "Chinese boxes" paradigm in which biological outcomes are nested in diverse types of individuals, which in turn are nested in municipalities with different social structures.

At the municipality level the analysis centers on poverty and violence as the two main contextual characteristics that reflect cumulative stress while controlling for urbanization. Contextual poverty is conceptualized as a constant vulnerability because –as will be shown in the next chapter– in Mexico poverty at the municipality-level is very stable. Violence, however, is sometimes characterized as a stable vulnerability for municipalities that are constantly violent but as an intermittent stressor if the homicide rate was low in 2006 and then spiked by 2012. Urbanization was categorized as a capacity although it may be also a stressor. Therefore, a limitation of the dissertation is that few contextual capacities are tested and the focus is mostly on vulnerabilities and stressors i.e. the interest is placed on violent municipalities rather than on peaceful ones.

The key person-level predictor of stress was monthly household expenditure as individual SES, which theoretically operates as vulnerability in the lowest economic quintiles. Conversely, collective efficacy served as an individual-level predictor of capacities when available. Intermittent stressors were personal and property victimization. Fear of crime was an independent variable that may affect the appraisal of an event and thus shape the intermittent stressors. These individual level predictors had their equivalence in the contextual measures of poverty and the homicide rates and these are the variables that may explain some of the mechanisms linking context with CVDR. In addition, the sociodemographic controls were gender, age, ethnicity, disabilities, household size,

marital status, and indicators of chronic diseases. These controls help to assess different sensitivities to stress.

When this diagram is examined over time, there are several simultaneous within and between unit comparisons: 1) within-person changes in the biomarkers as when waist circumference has yearly increases in an individual; 2) within-person changes on individual characteristics like an adolescent that increases income wave after wave in the survey; 3) within-municipality change as rapid urbanization or the increment in the homicide rates; 4) between-person differences in the biomarkers as people with wide or narrow waist circumference at baseline; 5) between-person differences that reflect varying sensitivities like between men and women or young and old individuals; 6) between municipality differences such as comparisons between poor and affluent municipalities. In order to estimate these comparisons the variance has to be accounted for at the three levels and over the three waves of the survey.

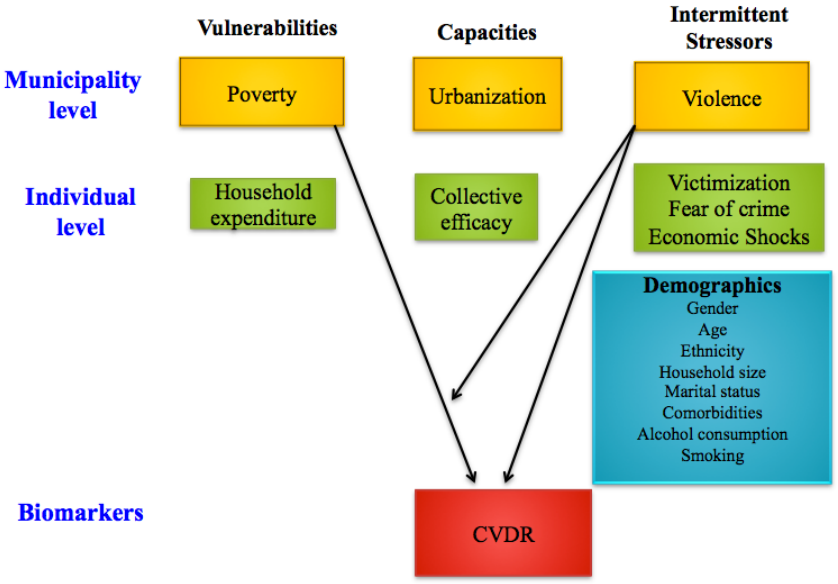


Figure 3. The diagram shows the multilevel framework for the dissertation´s empirical models. Source: author.

The empirical models test two types of associations in cross-sectional and longitudinal analyses. The main effects examine the direct impact of violence and poverty on CVDR; first in isolation as independent effects and then as an interaction i.e. violent and poor municipalities or peaceful and affluent municipalities. The indirect effects on CVDR are tested with cross-level interactions and tests for heterogeneity of effects, which help to identify the mechanisms leading to CVDR; arrows not displayed in figure 3. Cross-level interactions assess how person-level associations vary by context and the heterogeneity of effects examines the strength of association by subgroups. The next chapter delves into the details of each analysis.

Chapter IV. Methodology

4.1 Overview of the Study Design

The dissertation had two quantitative approaches: cross-sectional and longitudinal. An important reason to use both perspectives was to balance their limitations and triangulate the findings. A common thread for the two approaches was the emphasis on an ecological perspective. In terms of design, the ecological perspective intended to explain individual cardiovascular risk with municipality-level variables theorized as stressful while controlling for individual characteristics. An advantage of modeling the two levels simultaneously was to avoid the ecological fallacy. A second common thread was the examination of the data as a natural experiment. The overarching objective of the modeling strategies was to evaluate the differences in cardiovascular risk between people exposed to high homicide rates at the municipality level that drastically changed after 2006 with people unexposed to these levels of violence. Poverty is a constant confounder and catalyst of the association so it was always present when testing the effect of the violence exposure. Therefore, the level of scientific inquiry for the second approach is explanatory since risk variations were explained as cumulative exposures. An important design feature was that the analysis excludes people who moved to a different municipality in the period under study as a means to assess the effect of cumulative exposures to the same context. This raises a concern for self-selection into peaceful or violent municipalities, so the matter was addressed in Appendix 1. Another design hallmark of the dissertation was that most of the analyses highlight the importance of partitioning the variance between the two levels as well as between and within individuals. As is explained with each statistical analysis, a correct identification of the sources of error yields precise estimates to answer the hypotheses under study.

The analysis was divided in three sections: 1) the cross-sectional approach examined the between-person associations with HLM models; 2) the first longitudinal approach focused on within-person change; and 3) a second longitudinal approach integrated within and between person associations in a multilevel framework. The three sections began with descriptive statistics and exploratory analyses relevant to the methodological approach. Statistical nuances that delve into important issues but are indirectly related with the objectives of the dissertation were developed as stand-alone appendices.

The cross-sectional approach had two parts. The first sought to validate the multiple ways the cardio vascular risk was operationalized. The validation was cross-sectional because only the last wave of the survey contained all the measures. Since there was no gold-standard to validate the indices and single biomarkers, the analyses aimed to establish construct validity, specifically convergent validity (Kaplan & Saccuzzo, 2012). To validate the dependent variables, the risk measures were correlated with theoretically meaningful constructs. These constructs were grouped in sociodemographic, health, economic and violence sets of variables. While the health variables were more directly linked with the dependent variables, the economic and violence variables more closely resembled stressful situations. This grouping continued throughout the dissertation to ease the interpretation of results. Validation analyses are usually done at the first level, but from an ecological perspective it was important to assess the sensitivity to specific contexts. Thus, the validation process was first done at the person level and then at the municipality level.

The second part of the cross-sectional approach examined the between-person and the between-municipality associations in a full HLM model. The objective was to identify key predictors at both levels on the most robust dependent variable: the Framingham score. The main comparisons were at the municipality-level when person-level predictors were accounted for. In addition, cross-

level interactions between independent variables were tested. The variance was separated by level in both parts of the cross-sectional analyses.

The second and third sections focused on the longitudinal analyses. The approach had an ecological observational design (Aschengrau & Seage, 2008). This was a retrospective cohort study with an open population using the longitudinal data of the survey. The second section assessed within-person change between 2006 and 2012. Difference-in-difference models were used to test whether different "treatments" or types of contexts accounted for the mean individual differences. The "treatments" were violence, change in violence, and poverty. The "treatments" were tested at different thresholds, with and without covariates, and in interactions among them. Once the most adequate thresholds were identified, mean differences at baseline were compared. The dependent variable for these analyses was the Cardio Vascular Disease Risk index (CVDR).

The third section aimed to integrate the within and between variance at the person and the municipality levels. The best way to separate the variance in this way are the growth curve models (Raudenbush & Bryk, 2002). However, these models require at least three data points. The information needed to build the indices was not available for the three waves of the survey, so these models were conducted with three biomarkers: waist circumference, systolic blood pressure and diastolic blood pressure. Cross-level interactions between independent variables were also tested. The main outcomes of these analyses were the comparisons of growth trajectories of the single biomarkers based on whether municipalities were violent, poor, or both.

4.2 Sampling strategy

Secondary data for the analyses was used. Datasets were obtained from the publicly available Mexican Family Life Survey (MXFLS) (Rubalcava & Teruel, 2013). The MXFLS is a

longitudinal, multi-thematic, survey developed to assess the well-being of the Mexican population. The MXFLS gathered information for a ten-year period in three unbalanced waves: 2002, 2005-2006, and 2009-2012. To ease the communication of results on the rest of the dissertation, these waves are referred to simply as from 2002, 2006, and 2012. The MXFLS is representative of the Mexican population at the national, urban, rural, and regional level. The original sample comprised 35,000 individuals of ages 0 to 98 years from 8,400 households in 150 localities throughout the country. The second and third waves relocated and re-interviewed almost 90% of the original sampled households. The survey collected a wide array of social indicators at the individual, household and community levels, such as education, labor, income, marriage, participation in social programs, and victimization. The survey also gathered multiple health indicators like biomarkers of physical health, subjective assessments of health, a scale of depression, and a history of the current health condition. The analytical sample from the MXFLS discarded people under 15 years old because no biomarkers were collected.

The project used secondary data with de-identified information. No linkages can be made from the responses in the dataset to specific individuals. Accordingly, the project has, by definition, no human subjects.

Mexico has a population of 122.3 million people, residing in 2,473 municipalities within 32 states. Municipalities are the smallest political unit, with its own local government and police force, but most importantly it is the "place" where many social programs are targeted, managed, and evaluated. Public institutions offering municipality-level data are INEGI[4], for the homicide

[4] INEGI is Mexico's National Institute of Geography and Statistics. It generates several high quality, publicly available, nationally representative surveys relevant for the dissertation, such as the Census and the Victimization Survey (ENVIPE).

rates, and CONEVAL[5], for the poverty measures. Contextual measures came from these two sources and were matched to the sampled municipalities. The sample in the MXFLS comprised 16 states and 308 municipalities representing Mexico. The urban subsample reduced the number of municipalities to 130 based on its level of urbanization, defined by an average population above 2,500. A comparison between these municipalities indicated that the excluded had an average population density in the year 2000 of 404 people per square kilometer while the included had 801.

Statistical power to detect direct and cross-level interactions for Hierarchical Linear Models (HLM) suggested a relative premium of 3:2 in the average level 1 to level 2 samples. Since a simulation study found an optimal power of .80 in a sample of 18 individuals nested in 115 groups, the analytical sample of the dissertation should suffice to find the expected effects (Mathieu, Aguinis, Culpepper, & Chen, 2012) .

Once the youngest subpopulation was excluded, the original total sample size was 37,597. This figure includes 13, 061 cases with an ID but without data. In 2002, the sample comprised 24,536 individuals older than 15 years old. By 2006, the original sample had an attrition of 3,901 individuals and, in addition, by 2012 the lost to follow-up was of 2,485 individuals. Therefore, the complete sample for the three waves is 18,150. However, the survey design included a natural mechanism of replacement of the sample as to avoid losing representativeness due to attrition. Whenever a household added a new member –marriage or newborns- they were automatically included in the sample. 1,620 people were included in 2005 and 2,112 in 2009.

Importantly for the present study, 1,784 individuals changed municipality between 2002 and 2006 and 3,130 moved to a different municipality between 2006 and 2012. Therefore, 19,788

[5] CONEVAL is Mexico's National Evaluation Council, who evaluates public policies and publishes the poverty measures for the dissertation.

individuals, 80.56% of the sample, kept living in the same municipality during the three waves of the study. Among them, for 16,183 (81.8%) there is data for the three waves. Appendix 1 shows an analysis on key variables comparing the people who moved from those who stayed.

The study uses two samples. The first sample includes people from the second and third waves: 2006 and 2012. Most of the analyses came from this sample; they comprised the complete cross-sectional approach and the first longitudinal section, up to the difference-in-difference models. The second sample constituted the three waves and was used exclusively for the third section, with the growth curve models. Both samples had plenty of missing values, especially in the biomarkers. A discussion on the strategy to deal with this problem by using multiple imputations is in Appendix 2.

4.2.1 Sample 1

The final sample for the first analyses comprised 16,679 people nested in 130 municipalities. The average cluster size was 187 persons per municipality with a minimum of 25 and a maximum of 613. Therefore, the sample had enough statistical power. The following description shows how the final analytic sample was derived.

Starting with the original sample of 37,597. First 13,061 observations were deleted because they had missing values in all variables. Next, 3,426 observations were excluded because of the 2012 attrition. Then, 3,163 persons who had missing observations in all of the biomarkers that compose the risk indices score were omitted. These respondents refused to provide a blood sample and had missing values for several other reasons. Afterwards, 1,194 observations were left out because they lived in rural municipalities. 5 more persons were eliminated because they were 100 years old or more. Moreover, 18 observations were omitted because they have a missing value in the sampling weight variable designed for the health cross-sectional analyses.

A few observations in each biomarker had outliers when compared with average levels. For example, 31 persons have systolic blood pressure above 210 and 62 have a diastolic pressure above 120. Some of these levels may be implausible[6] but there was no clear empirical reason to exclude them, especially if they were going to be dichotomized and then included in a composite index. Thus, only 8 observations with diastolic pressure above 140 were discarded. After removing observations due to missing values and attrition, a comparison between the included and the excluded observations revealed several differences. The excluded –mostly from rural localities and people who changed municipalities– were 11 years younger, household incomes were lower, and the proportion of single individuals was higher. A higher proportion of the excluded subsample was female, with an indigenous background, and experienced more personal victimization. The excluded also had less chronic diseases, less comorbidities and a smaller waist circumference than those included. The final sample for the first analyses comprised 16,679 people nested in 130 municipalities.

4.2.2 Sample 2

The final analytic sample for the last analyses encompassed the three measures of single biomarkers from 2002, 2006, and 2012, nested in 14,862 people, which in turn were nested in 130 municipalities. The average cluster size was 174 persons per municipality with a minimum of 21 and a maximum of 667.

The procedure to reach the analytic sample was as follows. Starting in the original sample with 37, 597 observations of people older than 15 years old, the first step was to delete the 13,061 observations with missing values in all variables. The complete sample comprised 24,536.

[6] The inclusion criteria was broader than the used by (Kaplan & Saccuzzo, 2012) as to respect the available data, which goes smoothly and continually up (i.e. not as a single extreme value; see graphs in results section) and because Latin America, especially Mexico, has elevated averages in blood pressure and diabetes (M. Marmot, 2015) so these could be legitimate values. The used thresholds were: systolic blood pressure >60 & <230; diastolic blood pressure >30 & <140; total cholesterol 150-300 mg/dl; and HDL- cholesterol >20 & <100.

Among them, 20,803 (84.8%) joined the survey in 2002; 1,620 (6.6%) entered in 2006, and 2,112 (8.6%) refreshed the sample in 2009. Only the original members of the survey were kept. The attrition from 2002 to 2005 was of 1,780 (8.6%) individuals and the attrition for the period between 2006 and 2009 was 2,101 (11%). One observation was deleted because the respondent was older than 100 years in 2002. Therefore, the sample with data for the three waves was 16, 913. Then 6.4% (1,083) of the observations from rural municipalities were removed. Lastly, 249 more cases (0.016%) were deleted because they changed municipality at some time between 2002 and 2009.

The three dependent variables for these analyses were systolic blood pressure (SBP), diastolic blood pressure (DBP), and waist circumference. Even though only 48.95% have complete information on the three variables for the three waves (55.2% complete for the three waves on SBP, 55.1% on DBP[7], and 55.2% on waist circumference), all the remaining observations have at least one datum on these variables. A more rigorous standard was used to assess the implausible values of the dependent variables; the reference ranges to consider them came from the most recent ENSANUT survey (Gutierrez et al., 2012). Valid data was between the following ranges: 80-200 for SBP, 50-110 for DPB, and 50-180 centimeters for waist circumference. The implausible data for SBP comprised 181 observations overall (0.011%); observations below the lower bound were 50 in 2005 and 12 in 2009, while in the upper bound there were 45 in 2002, 5 for 2005, and 69 for 2009. DBP had more implausible data than SBP, with a total of 549 (0.034%); in the lower bound, there were 184 in 2002, 24 in 2005, and 1 in

[7] The mismatch in missing values between SBP and DBP tellingly concentrated exclusively in 2002: 151 observations have a value for SBP but no DBP and, conversely, 118 observations have a value for DBP but no SBP. It is worth remembering that in this wave, the data collection on BP was of a lower quality because it relied on a single measure, while in the following waves the measures were taken twice.

2009, and in the upper bound the number of invalid data was 172 in 2002, 23 in 2005, and 145 in 2009. Waist circumference had no implausible data.

As with sample 1, after removing missing values in every variable and after excluding attrition, the comparison between the excluded and the included showed several differences. The excluded were slightly older, with similar household expenditures, but lower education. The proportion of females and from indigenous backgrounds was higher in the excluded subsample. Personal victimization was lower in the omitted subsample. They had similar chronic diseases, higher comorbidities, and equal waist circumferences than those included in the sample. The final sample for the last analyses with single biomarkers consisted of 14,862 people nested in 130 municipalities.

4.3 Measurement

4.3.1 Dependent variables

The dissertation used three interrelated dependent variables: the Framingham score, Berkman and colleagues' CVDR index (2010), blood pressure, and waist circumference. They were all based on objective biomarkers.

Trained staff in the three waves collected medical samples; but personnel from the National Institute of Health made the data collection in the last two. In 2002, the MxFLS collected weight in kilograms, and height and waist circumference in centimeters. Unfortunately, blood pressure was taken only once. By 2005, the same biometric measures were collected, except for hip circumference. A blood sample added total cholesterol and hemoglobin. This time blood pressure was taken twice and then averaged. In 2012 blood pressure was also taken twice

and averaged. The other biomarkers were collected in the same way. In addition to total cholesterol and hemoglobin, HDL- cholesterol, and Glycosylated hemoglobin were added.

These biomarkers served as the base for the indices used as dependent variables. The Framingham score is a robust index conformed by the biomarkers available for 2012 and, therefore, was used exclusively for the cross-sectional analyses. The CVDR index is similar but requires fewer biomarkers and thus was employed for the two-wave longitudinal analyses. The preferred dependent variables for the growth curve models were the single biomarkers available for the three waves: waist circumference, systolic blood pressure, and diastolic blood pressure.

The most complex dependent variable was the Framingham score (Wilson et al., 1998). The original score was designed to assess cardiovascular disease risk in the next 10 years in people without cardiovascular problems. It is an additive score based on continuous and categorical variables. These variables are sex, age, total cholesterol, HDL-cholesterol, blood pressure, having diabetes and a smoking habit. Ranges on these measures are transformed into discrete points and then added as a score. For this study, a self-reported diagnosis of heart disease or hypertension granted a point even when cholesterol or blood pressure was below the risk cut points. The cut points greatly coincided with the Mexican consensus for the metabolic syndrome so the use of the measure seemed granted (Gonzalez-Chavez, 2002). Table 1 shows how the index was built. A fundamental predictor in the original Framingham study was age. For example, the original score provided adults (45+ years old) and elders (+60 years old) additional points regardless of the levels of the biomarkers and subtracted points to the youngest subpopulations. However, age was not as relevant for the purposes of the present research. Therefore, in the version for this study, a difference with the original score is that the present index does not *decrease* points due to age but leaves it in zero. A second difference with the original score is in the cut point for women´s blood pressure. According to the Mexican

consensus for the metabolic syndrome, the range between 130-139 & 85-89 should be considered with a point, whereas in the original it corresponded to a zero because it was below the risk threshold (Gonzalez-Chavez, 2002).

Table 1
Point Conversions to Build the Additive Framingham Score for the Present Study

	Framingham Score	
	Men	Women
Age	15-34: 0 35-39: 0 40-44: 1 45-49: 2 50-54: 3 55-59: 4 60-64: 5 65-69: 6 70+ : 7	15-34: 0 35-39: 0 40-44: 1 45-49: 3 50-54: 6 55-59: 7 60-64: 8 65-69: 8 70+ : 8
Total Cholesterol	<160: -3 160-199: 0 200-239:1 240-279:2 ≥280:3	<160: -2 160-199: 0 200-239:1 240-279:2 ≥280:3
HDL-Cholesterol	≥60: -1 50-59: 0 45-49: 0 35-44: 1 <35: 2	≥60: -3 50-59: 0 45-49: 1 35-44: 2 <35: 5
Systolic BP - Diastolic BP	<120 & <80: 0 120-129 & 80-84: 0 130-139 & 85-89:1 140- 159 & 90-99:2 ≥160 & ≥100: 3	<120 & <80: 0 120-129 & 80-84: 0 130-139 & 85-89:1 140- 159 & 90-99:2 ≥160 & ≥100: 3
Diabetes	Yes: 4	Yes: 4
Smoke	Yes: 2	Yes: 2

Note: BP= Blood Pressure

A limitation of the Framingham score was the difficulty to model the interactions between age, sex and the biomarkers because they were already included and weighted in the index. When these indices were used to measure stress rather than cardio vascular disease, these two variables may have lost some dominance. For instance, stress felt by a twenty-year old woman might have

been obscured in these two indices. Berkman and colleagues (Berkman et al., 2010) developed a dichotomous variable as a version of the Framingham score that uses only five of the relevant risk factors: smoking; obesity (BMI>30); high blood pressure (Systolic >140 and Diastolic > 90 or a self-reported hypertension diagnosis); high levels of cholesterol (Total cholesterol ≥ 200 or a self-reported heart disease diagnosis); and elevated levels of glycosylated hemoglobin (≥ 6% or a self-reported diabetes diagnosis). They proved with this measure that people with two or more risk factors were suffering work-related stress. A second advantage of Berkman's index is that almost all of these biomarkers are available for the 2006 MXFLS sample and thus it was ideal for the two-wave longitudinal analyses. The exception was glycosylated hemoglobin, so in 2006 only the self-reported diabetes diagnosis was used. In the present study, the CVDR index was not dichotomized so the variance could be modeled with greater precision.

Unfortunately, there was no available data in the 2002 wave to build these indices. Therefore, the dependent variables for the second sample were single biomarkers: waist circumference, systolic blood pressure, and diastolic blood pressure. The ranges of the implausible values have already been explained in the sample section.

4.3.2 Person-level independent variables

The following variables were used selectively in different models. Throughout the dissertation, the person-level independent variables were used in four thematic groups: Sociodemographic, Health, Economic, and Health variables. At the end of the list, a brief theoretical rationale of the variable selection is presented. The variables were built as follows:

<u>Sociodemographic variables</u>

Male: Dichotomous variable coded 1 for males and 0 for females.

Age: Continuous variable measures age in years.

Self-identification as indigenous: Dichotomous variable coded 1 for a person who self-recognizes as part of an indigenous group and speaks an indigenous language.

Disability: Dichotomous variable where the survey's interviewer labeled 1 if he or she considered that the respondent had a paralysis, was blind, deaf, or had a mental disability.

Household size: Continuous variable for the number of people living in the household.

Marital status: Dichotomous variable coded 1 if the respondent is married or living in cohabitation.

Health variables

Chronic diseases: Dichotomous variable coded 1 for an affirmative answer to the question: "Have you ever been diagnosed with [cancer, arthritis, gastric ulcer, migraine]?"

Comorbidities: Dichotomous variable coded 1 for an affirmative answer to the question: "Have you ever been diagnosed with [diabetes, hypertension, heart disease]?"

Smokes cigarettes: The original question was "Currently, how many cigarettes do you smoke a week?". Answers with one or above were coded as 1 in a dummy variable.

Alcohol consumption: Two questions were combined into a dummy variable. The first asked "When you are at a parties, reunions, or celebrations of any kind, what do you like to drink?" The second was: "In your house, with what beverage do you usually take your food with?". Items with alcoholic beverages like beer, tequila, rum, and "other alcoholic beverages" were tagged with a one. Any positive response to an alcoholic beverage was coded as 1.

Body Mass Index (BMI): It was calculated as weight in kilograms divided by the squared height in meters.

Subjective health rate: Ordinal variable with five response options for the question: "Currently, do you consider your health to be?" The options were: Very good/ Good/ Regular/ Bad/Very bad. It was coded so the maximum value, 5, reflects the worst health.

Depression: 20-item depression scale with four response options per item. The original instrument had 21 items (Calderon Narvaez, 1997). However, a reliability analysis showed that item 8 ("In the last 4 weeks, has your sexual interest diminished?") had a very low item-rest correlation of 0.2. Thus, it was deleted and the scale was rescaled to 0 from 1 as the minimum value. The scale had an alpha statistic of 0.93 in the three waves.

Hours of sleep: Continuous variable for the self-reported number of hours the respondent sleeps.

Economic variables

Education: Five-category ordinal variable: No-school and pre-school are labeled as "No instruction"; "Primary" reflects the first six years of education after pre-school (not mandatory in Mexico); "Secondary" corresponds to the next three years of education and includes the *open* system[8]; "High-school" stands for the next three years of education and comprises the high school *open* system as well as the *basic normal* system; and "College or higher" reflects the first year of college and above.

Household expenditure: Continuous variable for the household's monthly total spending in Mexican pesos.

Unemployment: Dichotomous variable coded 1 for not having worked in the last 12 months in a productive activity that helps with the household expenditure.

Household Economic shocks: An ordinal variable built from a summative score to four possible affirmative answers to the question: "In the last 5 years, did this happen in your household". The items were: a) death of a member of the household; b) sickness or serious accident from a member of the household that had required hospitalization; c) unemployment or commercial failure of a member of the household; d) loss of household or business due to an earthquake,

[8] In Mexico there are several ways to get a middle and high school diploma. The open system is a way to get it without going to regular classes and rather doing homeschool with standardized exams. It can be finished in less time than the regular diplomas so it is not an exact correspondence with the number of years. The basic normal system is a special school design to teach at public schools in basic levels and it oftentimes does not require high qualifications.

flood or any other natural disaster. The responses were transformed into a dummy variable coded 1 for any positive answer.

Violence variables

Personal victimization: Count variable for the number of times a respondent provided an affirmative answer to the question: "Have you been robbed, assaulted, or been victim of a violent event that happened outside your house, turf, or business from 2005 until today?" The year changes from wave to wave to specify the period between waves. In 2009, the range went from 0 to 5; 91.6% said no and 6.6% said 1. Since low percentages hardly reflected intensity, to facilitate the interpretation, it was dichotomized to 1 for any number of personal victimization events.

Property victimization: Count variable at the household level for the number of times a respondent provided an affirmative answer to two questions: "Has anyone trespassed by force on this [house/business] without permission or to rob after 2005?" Again, the year changes from wave to wave to specify the period between waves. Answers referring to house and business were added. For the same reasons that personal victimization, the variable was dichotomized to 1 for any number of personal victimization events.

Fear of crime: Two questions with four response options were combined into one: "During the [day/night] do you feel [Very scared/ Somewhat scared/ Little scared/ Not scared at all] of being a victim of an aggression or an assault?" The responses were then added into an ordinal variable with a range from 0 to 6, in which "not scared at all" was coded as 0.

Violence increase: Ordinal variable for the question: "In comparison to the present level of violence: how much violence and crime do you believe Mexico will face in the next three years?" The response options were: Much less/ Less/ Same/ More/ Much more. The responses were centered at 0 in the "Same" option. These questions were only available in 2006 and 2012.

Collective efficacy: A 9-item summative scale and each item with 4 response options. The questions were grouped in two themes. First around the trust on the neighbors (1-4): united neighborhood / people are willing to help neighbors/ share the same values/ people are trustworthy. The response options are: Very much agree/ agree/ disagree/ very much disagree. The second group referred to informal social controls in the locality or neighborhood (5-9): if a kid skips school, neighbors do something/ if kids spray with graffiti, neighbors do something/ if a kid disrespects an adult, neighbors scold/ if there is a fight, neighbors stop it/ if the police station is closing, neighbors organize to prevent it. The response options were: Very likely/ likely/ unlikely/ very unlikely. Responses had a range of 0 to 27. The scale with the two groups had adequate reliability with an alpha statistic of 0.84 in 2009 and 0.85 in 2005; it was not available for 2002.

4.3.3 Municipality-level independent variables

The two main independent variables (or exposures) at the municipality levels were violence and poverty; both controlled for inequality and the level of urbanization.

Violence was measured as the municipality-level homicide rate per 100,000 people. The measures were taken from the official mortality statistics (INEGI-SIMBAD) rather than police records because they are more reliable (David et al., 2017). The total population data to build the rates came from the 2000, 2005, and 2010 census; estimates by CONAPO. However, the sampling methodology of the MXFLS complicated the choice of years for the rates. A first option was to pick only the homicide rates for 2002, 2006, and 2012. The main problem was that some people would have been sampled in the MXFLS a year or two *before* the exposure. A second option was to use unbalanced averages i.e. the mean of total homicides from 2000-2002, 2003-2006, and 2007-2012. In this way, the exposure would always precede the individual sampling, missing data would be less harmful, and the grouping of years

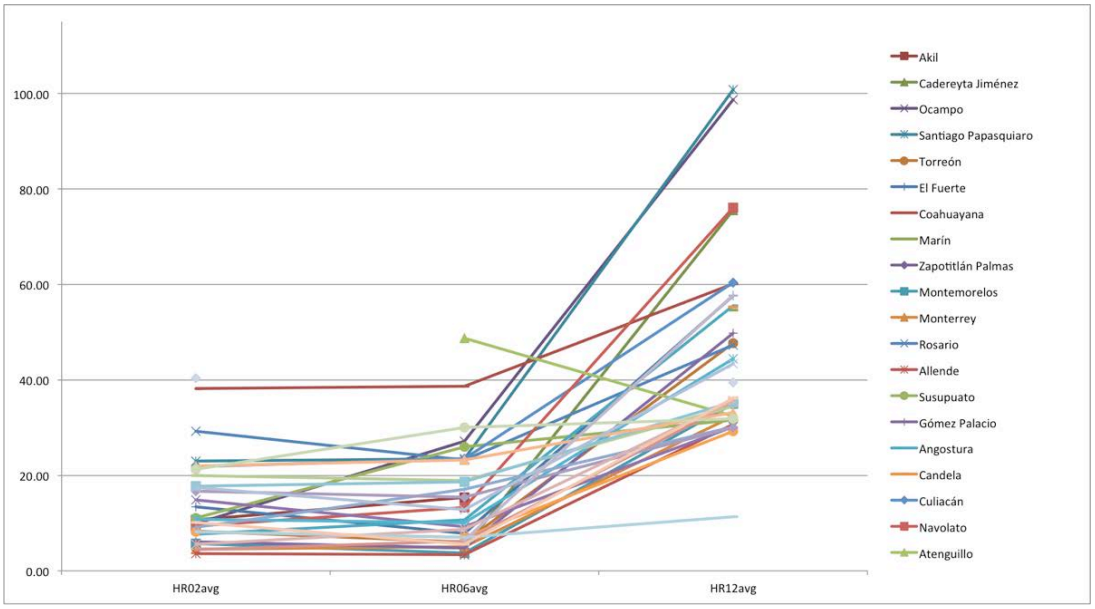
coincided with the increase of violence in Mexico. These reasons led to the second option for the dissertation[9]. Higher rates reflect more violence. For ease of interpretation, from now on, the word average will be omitted and these measures are referred to simply as from 2002, 2006, and 2012.

An initial exploration of the homicide rates in the sampled municipalities showed a similar pattern as the one observed at the national level. Graph A in Figure 4 depicted the trajectory of the homicide rates in those municipalities in the highest quartile for the averaged homicide rates in 2012. Likewise, graph B showed the trajectories for the municipalities in the lowest quartile for the averaged homicide rates in 2012. These two graphs revealed that each group of municipalities had its own story with violence. While graph A exhibited a stable trajectory from 2002 to 2006, it had a notable spike by 2012, as the national trend suggested, indicating important within change. Nonetheless, graph B showed it was not a uniform spike because many municipalities remained stable throughout the decade. This was provisional evidence supporting the feasibility of considering homicides as an external shock in some municipalities but not others. In other words, these graphs suggested that these differences might be considered as a natural experiment.

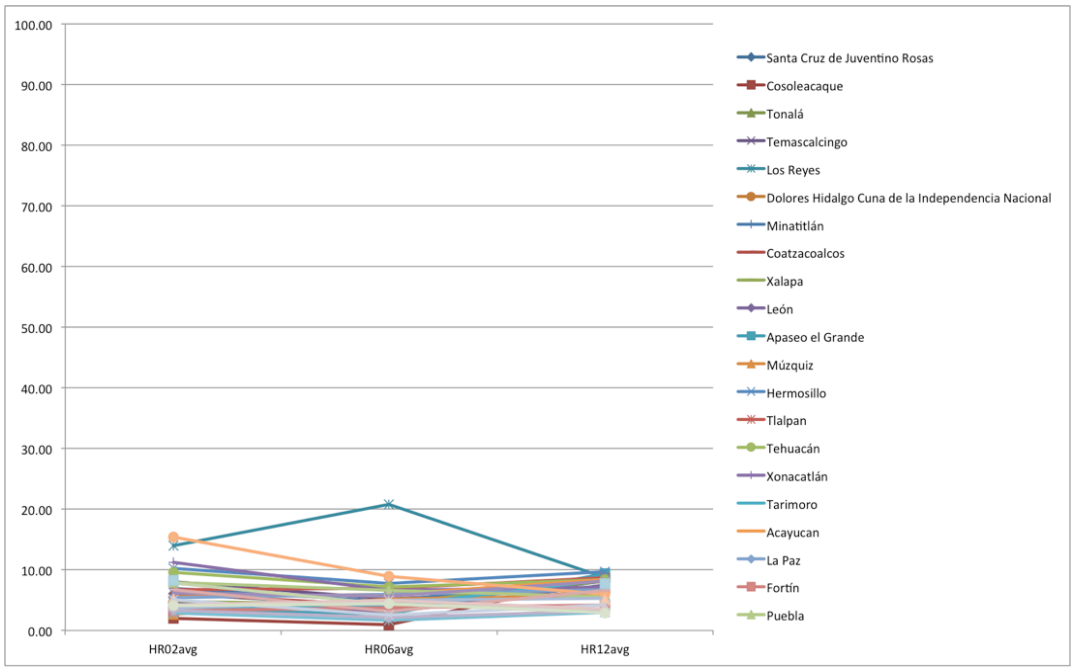
These graphs also indicated that the simple homicide rates might not be enough to describe the external shock. Municipalities that already had high homicide rates may be confounded in 2012 with the ones that were initially low but became violent after 2006. Therefore, a *change*[10] variable is warranted. It was built as the difference between the 2012 averages rates and the 2006 averaged rates. Higher levels indicate that the change from one year to the next was more drastic.

[9] An analysis not shown evidences a disadvantage of the approach. By averaging the rates of several years, the single number obscures sharp fluctuations. This estimation assuages the spikes in violence in some years and in certain municipalities.

[10] From now on, when *change* appears in italics it will refer to the variable and, without italics, refers to the word change in its regular sense.



A



B

Figure 4. Comparison of the homicide rate trajectories between municipalities by top and bottom quartiles of the 2012 homicide rates. Graph A depicts the trajectories of the municipalities in the highest quartile in 2012 on homicide rates. It shows low change from 2002 to 2006 and then a sudden spike from 2006 to 2012. Graph B shows a different path in the trajectories of the municipalities in the lowest quartile in 2012 on homicide rates. These municipalities had few changes along the decade.

There were several available measures for poverty using the 2000 and 2010 census, as well as the 2005 inter-censal sample. The best measure at the municipality level was multidimensional poverty. In 2010, CONEVAL designed it as an index, which showed the percentage of people that do not enact at least one social right (education, health, social security, housing, and food security) and that his or her income was insufficient to acquire goods and services that satisfy basic needs. Higher percentages mean higher levels of multidimensional poverty. In comparison with income-based measures, for the purposes of the dissertation, these dimensions better reflect the multiple sources of stress and may give a better account of the lack of institutional resources that constitute the basic mechanisms of the context effects. However, a major disadvantage was that the index is only available for 2012 and there are no previous measures.

The second-best measure used a traditional poverty line based on a minimum income. CONEVAL estimated three different poverty lines. Nonetheless, the "assets" poverty line more closely resembled multidimensional poverty because it comprised the percentage of people without enough income to spend in health, education, clothes, housing, and transportation. Actually, in the sampled municipalities, the correlation between the two poverty measures was 0.856 ($p<0.001$). The advantage of the assets poverty line was its availability since the year 2000. Notably, as will be discussed in the next chapter, the assets-poverty measures were stubbornly constant along these years, with a correlation between them of 0.902 ($p<0.001$). This means that there was very little within-municipality change and that poor municipalities remained poor and the wealthy were still wealthy. The fact that poverty was stable along the last decade, but the homicide rates had a sudden increase in a few municipalities, suggests that Mexico is an ideal site to disentangle the effects of poverty and violence.

The homicide rates and the poverty measures were transformed into dummy variables with different thresholds for different analyses. All these transformations and combinations are described with the statistical analyses in the next section.

Income inequality, measured with the GINI coefficient, was included to control for confounding poverty with relative rather than absolute deprivation. The second control was urbanization; operationalized as population density. The measure was the municipalities' population divided by its squared kilometers. The poverty, inequality, and urbanization measures have a two-year lag from the samples of the MXFLS: 2000, 2005, and 2010.

4.4 Statistical analyses

The analytic rationale for the exploratory analyses and model-building strategy to test the hypotheses followed two axes: 1) from person to municipality, and then to cross-level interactions; and 2) from cross-sectional to longitudinal associations, and then to an integrated approach. Each analysis emphasized the need to partition the variance in two ways according to the structure of the data: multilevel, to separate persons from municipalities, and longitudinal, to distinguish within-person from between-person variation (Hoffman, 2015).

4.4.1. Exploratory analysis

The exploratory analysis provided an initial grasp of the fundamental themes in the dissertation. It was distributed in three parts, at the beginning of each section. Descriptive statistics, box-plots, and histograms depicted the size and distributions of the key variables. In addition, correlations, mean comparisons, and box plots, offered a clear illustration of the main relationships with the dependent variables and among some of the independent variables. Most of the bivariate associations at the person level are in Appendix 3 as they are part of the validation process. The analyses started with person-level variables, then the municipality level variables, and end with

cross-level associations. The graphs were located at the end of each section. All descriptive analyses were conducted in STATA 13.

4.4.2 Between-person multilevel associations: cross-sectional HLM models

The between-person associations were examined with multilevel models using HLM software. A key advantage of these models was that they partition the variance for the person and for the municipality separately, so predictors may be assessed at their corresponding levels (Raudenbush & Bryk, 2002). The null models provided the amount of variance that municipality-level predictors can explain. HLM models helped test two types of hypothesis. Models with random intercepts and slopes examined how the adjusted means of the indices at the municipality level varied according to the homicide and poverty rates. These models also tested if person-level predictors had varying associations with the dependent variable; the random slopes assessed how these municipality-level predictors may have had cross-level interactions. For instance, the association between Fear of Crime and the Framingham score could be stronger in violent municipalities and weak or non-existent in peaceful ones.

Random-intercept models tested a different kind of interaction, the one between two independent variables, as is the case when contrasting the effects on the Framingham score of poor and violent municipalities against affluent and violent municipalities. These interaction variables were first built by multiplying the homicide rate and the *change* variable with multidimensional poverty. To ease the interpretation of results with point estimates, the three independent variables were transformed into four dummy variables that contrasted with an average municipality. The thresholds were the 25^{th} and 75^{th} percentiles for each variable (see Table 2). These dummy variables had as reference category the average municipalities within these ranges i.e. poverty and violence above their 25^{th} percentile and below their 75^{th} percentile.

Differences could be obscured by low statistical power because each combination only includes 8-11 municipalities, while the reference category has 92. The same strategy was used to compare the effect of poverty and the variable for *change* in the homicide rates (see Table 2). Low statistical power was an even bigger concern: only 2 municipalities had low change and low multidimensional poverty and just 4 had high change and high multidimensional poverty; this, itself, was already an interesting finding. Therefore, the most reliable comparison was contrasting municipalities with high poverty and low change against municipalities with low poverty and high change (n=13, each).

Table 2.

Thresholds for Dummy Variables for the Cross-sectional HLM Models

	Low 25^{th} percentile	**High** 75^{th} percentile
Homicide rate 2012	≤ 10	≥25
Multidimensional Poverty percentage	≤ 35	≥64
Homicide rate change 2006-2012	<1	>11

Theory suggests that municipalities with high violence and high poverty would be the most stressful. In a descending gradient of stress, they are followed by either high violence or high poverty, and the lowest average in the Framingham score should be in the municipalities with low poverty and low violence. The same gradient was hypothesized for the combination of *change* in homicide rates with multidimensional poverty.

HLM models with robust standard errors were fitted to test the between-person and the between-municipality associations with the Framingham score. Person level predictors and controls were included as to adjust the means. In order to estimate the contextual effects by focusing on municipality level variation, person-level predictors were group-mean centered when the zero was not meaningful and dummy variables were left uncentered. Conversely, the

municipality-level variables were grand-mean centered when they did not have a meaningful zero (Enders & Tofighi, 2007). The slopes of the person-level independent variables were allowed to vary randomly to test cross-level interactions. Whenever the variance term was significant, the municipality-level predictors were tested on how these associations vary across municipalities. When the associations were not significant these models are not shown. The analyses were conducted with 10 multiple imputation datasets in HLM software version 7.

Except for the null, the rest of the models were adjusted for several person-level and municipality-level controls; estimates not shown. Therefore, the adjusted intercept of models 2-7 corresponds to non-single people without chronic diseases, who do not self-identify as indigenous or as having a disability and that live in an average size household for their municipality. At the municipality level population density was adjusted at its national average. Likewise, the person-level predictors reflect an average household expenditure, average fear of crime, and average collective efficacy for their municipality. All the models were estimated for people that were not victims. The models sought to answer the following hypotheses:

- Model 1: There is significant variability in the Framingham score at the municipality level.
- Models 2-5: At the individual level household expenditure and collective efficacy have a negative association with the Framingham score, while personal victimization, household victimization, and fear of crime have a positive association.
- Models 2-5: At the individual level household expenditure, fear of crime, and collective efficacy have significant variability in the Framingham score at the municipality level and the context variables significantly account for it by cross-level interactions.

- Model 2: At the municipality level 2012 homicide rates and 2012 multidimensional poverty have a positive and significant association with the Framingham score.
- Model 3: The previous associations are not confounded by income inequality.
- Model 4: At the municipality level the *change* in homicide rates and 2012 multidimensional poverty have a positive and significant association with the Framingham score.
- Model 5: The previous associations are not confounded by income inequality.
- Model 6: At the municipality level 2012 homicide rates, 2012 multidimensional poverty, and its interaction have positive and significant associations with the Framingham score.
- Model 7: At the municipality level *change* in the homicide rates, 2012 multidimensional poverty, and its interaction have positive and significant associations with the Framingham score.
- Model 8: Model 6 is confirmed with dummy variables.
- Model 9: Model 7 is confirmed with dummy variables

4.4.3 Within-person individual associations: difference-in-difference models

The within-person associations were estimated using fixed effects models with two-time periods; specifically, difference-in-difference models. Fixed effects models allow controlling for confounders that have not been measured and use each individual as its own control (Allison, 2009). These models, however, did not control for unobserved variables that changed over time and were of little use when the time-variant independent variables had small changes over time. These models discarded between-person differences and only used within-person differences, which reduced efficiency because they used less information and yielded substantially larger

standard errors. In spite of the trade-off between efficiency and reduced bias, fixed effects models were adequate for quasi-experimental studies (Allison, 2009).

A useful application of fixed effects models are natural experiments, which arise when an exogenous event, as the increase in the homicide rates, changes the environment in which individuals live. A natural experiment must have a control group i.e. similar environments in which the exogenous event did not occur. Unlike a true experiment, the researcher does not randomly assign to groups nor is able to manipulate the independent variable *ex ante* (Singleton & Straits, 2010). When these two groups are naturally formed and there are two time-points available, before and after the exogenous event, then a difference-in-difference model is adequate to assess the within-person effect of the event (Wooldridge, 2009). Therefore, it is critical to establish equivalence of groups at baseline as to justify the treatment as an external shock and not as a previous difference. The aim of the model was to identify the average treatment effect by estimating first the differences in averages between the treatment and the control groups at baseline and then the difference in the resulting averages over time. The average treatment effect is thus the interaction between the intervention group and time (Wooldridge, 2009).

A problem with the difference-in-difference model is the assumption of constant variance over time i.e. that participants varied just as much from each other from time1 to time 2 (Hoffman, 2015). For example, people with initial high levels in the CVDR indices may have a smaller increase over time than people with initial low levels. The problem is that the within-person model does not remove this kind of between-person variation and ignores both sources of variation. In order to account for correlated residuals, models need to partition the between-person variance from the within-person person variance by assigning a random intercept. For fixed effects models a significant between person variance could bias the results if it is not accounted for: “after including the random intercept to represent those constant person mean

differences, the residuals from the same person are then independent" (Hoffman, 2015). Therefore, two parameters in the model need to distinguish the sources of variation. Importantly, the partition does not affect the mean coefficients; it only makes the standard errors more precise. The difference-in-difference models accordingly were conducted with the mixed command in STATA 13. The estimation method was maximum likelihood with an unstructured covariance matrix and exchangeable residuals. A big disadvantage of the mixed command was that it did not allow multiple imputations. Therefore, the models were estimated with list-wise deletion.

These models followed a four-part modeling strategy. They first compared three kinds of "treatments" at the municipality level; each treatment divided the population in two. The treatments were the 2012 homicide rate, the homicide rate *change*, and the 2012 multidimensional poverty. For each treatment, the threshold where the treatment became meaningful needed to be identified. Therefore, the 2012 homicide rate divided the population, first between municipalities with a rate above 15 (45% of the municipalities and slightly above the median of 12.4), then above 25 (25% of the municipalities and the 75th percentile), and finally above 35 (23% of the municipalities). Likewise, the *change* threshold was first set above 10 (28% of the municipalities and almost the 75th percentile of 11), above 20 (19% of the municipalities), and above 30 (12% of the municipalities). The *change* thresholds started at the 75th percentile because several municipalities decreased their homicide rates and thus the median was at 4, which was previously not considered as a meaningful change. Finally, the poverty thresholds started above 45 (almost above the median of 44 with 57% of the municipalities), then above 55 (41% of the municipalities) and then above 65 (23% of the municipalities when the 75th percentile was 61). The thresholds did not go further up because of the loss of statistical power as the municipalities *receiving* the treatment decreased in number.

The second part of the modeling strategy was to identify the heterogeneity of effects. As the literature suggests, gender and age are key covariates for the CVDR index but the models of the first part were estimated without covariates. Then the models were re-estimated first for males and then for females and they included a dummy variable for respondents over 40 years old in 2006. The dummy variable makes the interpretation of the models as males/females younger than 40 years old. At the threshold in which the difference in difference coefficient was significant, personal victimization, household economic shock, and dummy variables for all the household expenditure quintiles were added; models not shown.

The third part tested equivalence at baseline in key variables for the significant thresholds. The equivalence tests consisted of means and proportion comparisons of sociodemographic, economic, health, and violence variables; all measured in 2006. The corresponding measures at the municipality level were also tested.

Lastly, the fourth part of the modeling strategy examined the significance of the interactions between homicide rates with multidimensional poverty and between *changes* in the homicide rates with multidimensional poverty. The interactions were tested as multiplicative coefficients and then as dummy indicators when both conditions were met. The thresholds of the dummy variables were established based on previous results (see Table 3). Municipalities above the median and below the "high" threshold comprised the reference group.

Table 3.

Thresholds for dummy variables for the cross/sectional HLM models

	Low	**High**
Homicide rate 2012	<=12 (median)	>=35 (85th percentile)
Multidimensional Poverty percentage	<= 44 (median)	>=65 (80th percentile)
Homicide rate *change* 2006-2012	<=4 (median)	>=10 (75th percentile)

The resulting four groups were dummy variables for low violence and low poverty (19% of municipalities), low violence and high poverty (11% of municipalities), high violence and low poverty (7% of municipalities), and high violence and high poverty (0.8% of municipalities). The last group was so small that it lacked sufficient statistical power to find meaningful results. When the homicide rate variable was substituted by *change* in homicide rates, the four new groups had a distribution that permits stronger inferences: low violence *change* and low poverty (11.5% of municipalities), low violence *change* and high poverty (15% of municipalities), high violence *change* and low poverty (15% of municipalities), and high violence *change* and high poverty (3% of municipalities). Again, the last group, with both thresholds at high levels, had the lowest sample size, thus compromising the statistical power. Even though there are no specific claims for the thresholds, the set of hypotheses is:

- There is a significant CVDR mean increase from 2006 to 2012 in all models.
- Municipalities with high 2012 homicide rates do not have a significant CVDR mean difference with the control group in 2006 but the CVDR mean is significantly higher by 2012.
- Municipalities with high levels of *change* in the homicide rates do not have a significant CVDR mean difference with the control group in 2006 but the CVDR mean is significantly higher by 2012.
- Municipalities with high 2012 multidimensional poverty do have a significant CVDR mean difference with the control group in 2006 and the CVDR mean is still significantly higher by 2012.
- There are no gender differences from the previous models in any of the three treatments.

- Age, victimization, and economic shocks reduce the CVDR mean differences in the models without covariates but differences between groups are still significant.
- The CVDR mean differences from the previous models are bigger in the poorest economic quintiles and smaller in the wealthiest economic quintiles.
- There are no significant CVDR mean differences in any of the interaction models in 2006.
- The 2012 differences with the control groups are highest in municipalities with high violence and high poverty.
- The 2012 differences with the control groups are lowest in municipalities with low violence and low poverty.
- The 2012 differences with the control groups are higher in municipalities with high violence and low poverty than in municipalities with high poverty and low violence.

4.4.4 Multilevel between and within person associations: Growth curve models

Modeling change demanded adequate conceptualization, measurement, and design (Raudenbush & Bryk, 2002) . Conceptualization under this framework referred to the type of questions a statistical model is able to answer. Singer and Willet (2003) explain that two questions tap on the core of the research of change: What is the pattern of a person's change over time? And which predictors are associated with which patterns? The average pattern can have a linear or nonlinear form, the direction could be positive or negative, and the rate of change may be fast or slow. While the first question involves a relationship exclusively among the dependent variables, the second question calls for the associations with other independent variables. Put differently, if change is conceptualized as several measures nested in individuals, then the

analysis of change proceeds in two stages (Singer & Willett, 2003). The first stage dealt with the first question, the within-individual change over time, and was illustrated by the individual growth trajectory. The second stage tackled inter-individual differences in change and examined how different people manifest different patterns of change i.e. heterogeneity of change between persons (Singer & Willett, 2003). The second stage could be extended to a third level in which people had different exposures as they are nested in places with multiple characteristics (Raudenbush & Bryk, 2002). Hierarchical linear models, especially Growth Curve Models, can examine both questions simultaneously and are thus "a powerful set of techniques for research on individual change" (Raudenbush & Bryk, 2002, p. 161). The key advantage of these models was the possibility to model within-person change *and* between-person variations while examining the context effects of municipalities on equivalent people.

Studies of change that use growth curve models require continuous measures whose values change systematically over time in a meaningful parametric form i.e. linear or quadratic (Singer & Willett, 2003). Systematic change over time means that the measures of the dependent variable have to be equivalent so the value of an outcome represents the same amount on any occasion. Therefore, the same instruments, used in the same way, should be used in every wave. A sensitive measure of time is also necessary for these studies (Singer & Willett, 2003). It must be monotonic, so it always follows the same direction, as years that pass by. The sensitivity of the time measure has to be in accordance with the theoretical rationale of the model.

A key design feature for growth curve models is the number of data points collected. Cross-sectional analyses con not measure change because there is only one data-point. However, two-data points may indicate change but not a pattern of change or growth; two is not enough to establish the rate of change, the form of the trajectory, and measurement error is harder to discard. Therefore, three-data points is the minimum to estimate these models (Singer & Willett,

2003). Spacing between waves is a theoretical decision based on the phenomena under study but Singer and Willet suggest that: “If you expect rapid nonlinear change during some time periods, you should collect more data at those times. If you expect little change during other periods, space those measurements further apart” (Singer & Willett, 2003, p. 12). Equal spacing and balanced waves (complete information per individual for all waves) is appealing for interpretation but it is not a requirement (Singer & Willett, 2003, p. 12).

Secondary data limits some decisions for the adequate modeling of change due to data availability. The research questions of the dissertation are conceptually well suited for the use of Growth Curve Models. Likewise, the dependent variables were measured in the same way and showed considerable within variation. However, some design issues posed problems. An important disadvantage of having only three data points is the restriction to a linear form so more complex processes cannot be modeled (Raudenbush & Bryk, 2002). The hypothesis of the dissertation is that individual change should be similar across municipalities between 2002 and 2006 but it accelerates by 2012 in the violent and poor municipalities. Therefore, the main hypothesis suggests a quadratic form but the available data forces a linear form. In addition to shape, data availability limits the number of random parameters to two: “the number of observations, T_i, must exceed the number of random parameters, P+1, specified in the individual growth model” (Raudenbush & Bryk, 2002, p. 176). Therefore, in a three-level model in which exposure is of key interest, the two random parameters have to be the two random intercepts: person (*e*) and municipality levels (*u*). This means that cross-level interactions cannot be tested and that heterogeneity of effects at the person level on the rate of change need to be fixed i.e. held constant.

A second limitation of both longitudinal approaches in the dissertation was the spacing between waves. Even though the dissertation examines cumulative stress, the distance between

the measures could be too large and obscure fluctuations within those five years. The third limitation due to data availability was the impossibility to build the Framingham score or the CVDR index, which have a different pattern of change from biomarkers, as theory suggests.

In spite of these limitations, the third analytic part of the dissertation estimated three-level Growth Curve Models for measures nested in persons who are also nested in municipalities. Three equivalent models were estimated, one for each dependent variable, the three single biomarkers: waist circumference, diastolic blood pressure, and systolic blood pressure. The independent variables at the municipality level were the same as the ones used to model the Framingham score: 2012 homicide rates, homicide rate *change*, and multidimensional poverty. As reference, *Change* was always modeled with the 2006 homicide rate centered at its grand-mean. Dummy variables were used to test the interactions between them. However, they were built using the thresholds of the difference-in-difference models (Table 3). The municipality-level controls were income inequality and population density. Municipality-level variables were grand-mean centered.

Several individual-level variables controlled for the effects of the exposures to these municipality characteristics. The sociodemographic controls were gender, age in quintiles, marital status, ethnicity, disability, and household size. The economic variable to control for the poverty context effect was household expenditure in quintiles. The health controls were: chronic diseases, comorbidities, smoking, and alcohol consumption. Person-level controls were group-mean centered. These controls imply that the intercept should be interpreted as the average value of an able-bodied, nonindigenous, married woman, between 36 and 44 years of age, living in an averaged-size household for her municipality. She is part of the third economic quintile according to her household expenditures. In addition, she has no chronic diseases or comorbidities, does not smoke, and does not drink alcohol frequently.

As is explained in greater detail in Appendix 2, HLM software allows multiple imputations only for the first level and uses list-wise deletion for the second and third levels. Therefore, controls at the person level with many missing values dramatically reduced sample size and threatened the validity of the model. Since the inclusion of the victimization variables would have reduced the sample by more than 20%, they were excluded.

Three groups of random-intercepts Growth Curve Models were estimated with 10 datasets from multiple imputations. The datasets were in long form. At the measure level, there was one parameter: the time or wave variable; coded zero for 2002, one for 2006, and two for 2012. Thus, the coefficient of the intercept in the null model indicates the value of the biomarker at baseline and the time variable is the slope of the growth trajectory, expressing the rate of change per wave. The error parameter of the biomarker's intercept was set as random and the error for time as fixed. The null model also showed how much variance was distributed at each of the three levels. At the person level, when the aforementioned controls were added, the intercept was then an adjusted mean. Again, the error parameter of the person's intercept was set as random and the error for time was fixed. Then the municipality-level variables, at the third level, were added in the same order as with the Framingham score, except that cross-level interactions were not tested. Population density was present in every model as a control but it was not reported in the tables to maintain parsimony.

The overall objective of these models was to examine if municipality characteristics predicted different growth trajectories. In particular, the models tested the following hypotheses:

- Null models: The rate of change is positive and significant for the three biomarkers.
- Null models: There is significant variability in the biomarkers at the person and at the municipality levels.

- Model 1: At the municipality level 2012 homicide rates has a positive and significant association with the rate of change but not with the biomarkers at baseline. And 2012 multidimensional poverty has a positive association on both coefficients.
- Model 2: The previous associations are not confounded by income inequality.
- Model 3: At the municipality level homicide rate *change* has a positive and significant association with the rate of change but not with the biomarkers at baseline. And 2012 multidimensional poverty has a positive association on both coefficients.
- Model 4: The previous associations are not confounded by income inequality.
- Model 5: At the municipality level the interaction of high 2012 homicide rates and high 2012 multidimensional poverty has a positive and significant association in the rate of change when compared to an average municipality. Conversely, the interaction of low 2012 homicide rates and low 2012 multidimensional poverty has a negative and significant association in the rate of change when compared to an average municipality.
- Model 6: At the municipality level the interaction of high homicide *change* and high 2012 multidimensional poverty has a positive and significant association in the rate of change when compared to an average municipality. Conversely, the interaction of low homicide rate *change* and low 2012 multidimensional poverty has a negative and significant association in the rate of change when compared to an average municipality.

Chapter V. Findings

The main hypothesis of the dissertation was that, in urban environments, exposure to higher levels of contextual violence works as a stressor that wears down the body by increasing the levels of cardiovascular risk. The first research question focused on the indirect effects on health due to the cumulative exposure to contexts characterized by high levels of violence in Mexico. The second sought to examine if these effects are independent from poverty or if they operate as an interaction. Finally, the third question inquired if there were further interactions with individual characteristics.

The chapter was structured in three sections that mirror the three methodological approaches. It started with cross-sectional analyses emphasizing the between person comparisons in multilevel models. The second section tackled the first longitudinal approach, focusing in the within-person differences. Finally, the third section integrated both approaches with growth curve models. Key insights were illustrated with graphs at the end of every section. Each section offers a partial response to the research questions; however, the triangulation of the results balances the methodological strengths and weaknesses to offer a more robust overall synthesis of the main findings.

5.1 Between person analyses

The first section started with an appreciation of the size and distributions of the variables corresponding to the 2012 wave. The results were presented from the person-level to the municipality level, first with univariate and then bivariate statistics. The municipality-level descriptive statistics included two and three waves to provide context to the measures. These findings helped to validate the dependent variables i.e. assess their sensitivity to health, economic, and violence independent variables at both levels; these results are in Appendix 3. At

the end of the section, a multilevel model integrated between-person and between- municipality associations with the Framingham score.

5.1.1 Person-level univariate statistics

The sociodemographic composition of the analytic sample for the most recent wave, 2012, is displayed in Table 4. The sample had 44% of males and the average age was 42.8 years. 7% of the sample self-identified as indigenous and spoke an indigenous language. This corresponded with national estimates; INEGI reported that 7% of the population speaks some indigenous language. Only 3% in the sample reported having some disability. The average household size in the sample was 5 people and 28% of the respondents were single.

The next set of variables in Table 4 showed important health indicators. The prevalence of chronic diseases in the 2012 sample was 10%. The mean BMI was 27.5. According to WHO, a person with a BMI above 25 is considered overweight and a BMI above 30 is obese. In a national sample, ENSANUT 2012[11], based on the BMI, 71% of Mexicans were labeled as overweight or obese (Gutierrez et al., 2012). Importantly, the overweight and obesity prevalence increased, for women, by 41% and 270%, respectively, between 1988 and 2006. The tendency slowed down between 2006 and 2012, when overweight decreased and the prevalence of obesity only increased by 3%. For men, in the period between 2000 and 2012, the overweight prevalence increased by 3% and obesity by 38%. As for women, the increasing rate was lower in the period between 2006 and 2012 (Gutierrez et al., 2012). Similar to the national estimates, in the 2012 sample 65.75% had a BMI above twenty-five; 37.5% were overweight and 28% obese. While men had a higher overweight prevalence (41.7% vs 33.5%), women had a higher one in obesity (29.9% vs 26.8%). Accordingly, the prevalence of comorbidities in the sample was high: 18.6%. This estimate

[11] The ENSANUT is a nationally representative survey on health and nutrition topics. It is conducted every six years by Mexico's National Institute of Health.

reflects the national prevalence of diagnosed diabetes at 9% and 13% had high cholesterol levels (Gutierrez et al., 2012). On the contrary, the prevalence of smoking in the sample was low. The ENSANUT documented that daily consumption of cigarettes was 12% in 2012 and the MxFLS registered half; although the ENSANUT did not separate by urban localities. Occasional alcohol consumption was 34% in the sample but the estimates are not comparable with the national prevalence because the items were different. The responses in the sample had a symmetric distribution for self-rated health (mean=2.5) and respondents reported relatively low levels of depression, with a mean of 6. The average hours of sleep were 7.6.

The sample reflected Mexico´s socioeconomic composition. Whereas in the sample 76% completed less than nine years of education, the 2010 national census revealed that the average level was 8.6 years; 70% of the population finished less than nine years of education. Household monthly expenditure in Mexican Pesos was 4,722 on average in the sample; at 13 pesos per dollar in 2012, it roughly equates to 363.2 US dollars. As expected, there was a positive and significant correlation between education and household expenditure (r=0.345); see graph A in figure 5. Even though INEGI reported unemployment at 5% for 2012, in the sample 44% were unemployed because it included students and retired older adults; see graph B in Figure 5. Almost a third of the sample acknowledged an economic shock (35%).

Among the violence set of variables, people in the sample recounted lower levels of victimization in the last five years (8% personal victimization and 5% property victimization) than in the ENVIPE, a nationally representative victimization survey conducted by INEGI. For instance, in the ENVIPE, in just one year, from 2011 to 2012, the incidence was the same as the sample's: personal victimization was 8% and property victimization was 5%. Similarly, people in the sample reported low levels of fear of crime (1.39 in 2012), with more than half of them expressing no fear whatsoever. In the ENVIPE survey for 2012, only 40% reported feeling safe in

their municipality. In the sample, a slight majority perceived violence would increase in the next three years. With an average score of 15, collective efficacy had a symmetric distribution.

Table 4

Person-level Descriptive Statistics for 2012

Variable	Mean/Proportion	SD	Min	Max
Sociodemographic variables				
Male	44%		0	1
Age 2012	42.85	16.7	15	100
Indigenous	7%		0	1
Disability	3%		0	1
Household size	5.3	2.68	1	23
Single	28%		0	1
Health variables	Mean/Proportion	SD	Min	Max
Chronic diseases	10%		0	1
Body Mass Index (BMI)	27.53	5.18	12.8	81.97
Comorbidities	18.6%		0	1
Smokes cigarettes	6.8%		0	1
Alcohol consumption	33.8%		0	1
Subjective health rate	2.5	0.72	1	5
Depression	6.01	7.53	0	60
Hours of sleep	7.65	1.37	1	15
Economic variables	Mean/Proportion	SD	Min	Max
Education:				
No instruction	9.35%			
Primary (6 years)	39.95%			
Secondary (3 years)	26.79%			
High school (3 years)	14.03%			
College or higher	9.87%			
Household expenditure	4,722.7	3,024.1	0	15,606
Unemployment	41%		0	1
Household Economic shocks	35%		0	1
Violence variables	Mean/Proportion	SD	Min	Max
Personal victimization	8%		0	1
Property victimization	5%		0	1
Fear of crime	1.32	1.78	0	6
Violence increase	0.74	1.04	-2	2
Collective efficacy	15.7	4.9	0	27
Dependent variables	Mean/Proportion	SD	Min	Max
Framingham score	6.8	4.3	0	20
CVDR index	1.18	1.02	0	5
Waist circumference	93.6	13.04	50	166
Systolic pressure	123.97	20.5	72.5	228
Diastolic pressure	79.34	11.35	48	140

Means and proportions were estimated with 20 datasets from multiple imputations. CVDR= Cardiovascular Disease Risk.

The Framingham score, the most robust dependent variable, had a mean of 6.8, a median value of 7, and a standard deviation of 4.3. The minimum score was 0 and the maximum was 20. Graph C in Figure 5 showed that the distribution was not symmetrical and that it was skewed towards lower scores (7% had a score of 0). The Cardiovascular Risk Disease index (CVDR) had a shorter range (0-5), with a mean of 1.18 and a median of 1; the skew was also clear, see graph D.

The single biomarkers that were used as dependent variables had symmetrical distributions; see graphs E-G in Figure 5. Waist circumference had a mean of 93.6 and a standard deviation of 13. WHO parameters consider a waist circumference above 90 as abdominal obesity. In Mexico's ENSANUT for 2012, for the adult population, 64% of men and 83% of women had abdominal obesity; in urban localities, the prevalence was 28% higher than in the rural ones (Gutierrez et al., 2012). In the sample 59% had abdominal obesity; 54.7% females and 65.1% males.

Following the Mexican medical consensus, the ENSANUT considers as hypertensive a diagnosed person or someone with a systolic blood pressure value over 140 and a diastolic blood pressure value over 90. The national prevalence of hypertension was 33% and it remained stable in the period between 2006 and 2012 (Gutierrez et al., 2012). In the sample, systolic blood pressure had a mean of 123 and a standard deviation of 20.5. In a similar distribution, diastolic blood pressure had a mean of 79 and a standard deviation of 11.35. The two blood pressure measures had a slight skew to the right. Only 12.75% of the sample reported having hypertension and, among them, 24.4% did not take medicine to control their blood pressure. The sample prevalence reached 19% once the diagnosed and those that exceeded the hypertension threshold were added; however, they were still well below the national averages.

5.1.2 Municipality level univariate statistics

Descriptive statistics from the second level were key because the main independent variables were at the municipality and thus they provided valuable clues for model interpretation. The average homicide rate for 2012 in the sample's municipalities was 20.4, with a median of 13.2, a SD of 18, a minimum value of 2.9 and a maximum value of 100.8; see table 5. In contrast, the average homicide rate for 2006 was 10.99, the median 7.25 and the maximum value was 50.7; half the one for 2012. A paired sample t-test yielded a significant difference score of 9.42 ($t=-6.17$; $p<0.000$). These rates corresponded with the national spike in homicides after 2006 and illustrated that the homicide rate was actually decreasing between 2002 and 2006 (Graph H in Figure 5). The variable for *change* in the homicide rates between 2006 and 2012 showed significant variability, with a mean of 9.4, a standard deviation of 17.39, and a considerable amount of negative values. As graph I shows, many municipalities remained unchanged or had a slight increase, but several municipalities even decreased their rates in the period from 2006 to 2012. Therefore, the ones that had a spike over 10 drove up the mean difference.

Multidimensional poverty was the best poverty measure because it captured other social needs besides income. However, it was only available for 2010. The average was 50.14, which means that 50% of the people living in a municipality reported lacking at least one dimension of poverty. It had considerable variability, with a minimum value of 13% and a maximum of 95% people living in multidimensional poverty. Likewise, the measure of assets poverty reflected the highest poverty line measured with income. It had an average of 52.2% and also showed notable variability: a standard deviation of 15.4, with a maximum value for a municipality of 92% of its inhabitants living below the income-poverty line. There were previous available measures for the assets poverty line, but they revealed no statistical change in the periods between 2000 and 2005 and with 2010 (Graph J).

The GINI coefficient was 0.40 for 2010. When compared to the value of 2005, it did have a significant decrease of 0.03, as well as when the 2005 coefficient was compared with the one from 2002 (Graph K). Conversely, population density in 2010 had an average increase of 26.6 inhabitants per square km when compared with 2005 (t=2.189, p=0.03). The difference was not significant when comparing 2000 with 2005 (Graph L).

Table 5

Municipality-level Descriptive Statistics (N=130)

Variable	Mean	SD	Min	Max
Homicide rate 2012	20.4	18.2	2.94	100.8
Homicide rate 2006	10.99**	9.14	0.95	50.7
Homicide rate 2002	11.63^{ns}	9.35	0.88	58.8
Homicide rate change 2006-2012	9.42	17.39	-25.5	77.3
Multidimensional poverty 2010	50.14	18.29	13.2	94.9
Assets poverty 2010	52.25	15.42	20.4	92.3
Assets poverty 2005	52.56^{ns}	16.28	21.8	91.22
Assets poverty 2000	53.39^{ns}	18.27	10.9	97.2
GINI 2010	0.40	0.04	0.31	0.57
GINI 2005	0.43**	0.04	0.34	0.52
GINI 2000	0.47**	0.05	0.34	0.62
Population density 2010	1,113	3,034	1.22	17,423
Population density 2005	1,087*	3,071	1.15	17,893
Population density 2000	1,060^{ns}	3,106	1.13	19,233

*=Statistical difference with a t test mean comparison of paired samples; compared with the row above. NS=No statistical difference.

5.1.3 Municipality level bivariate associations

In contrast to the assets poverty measures that were strongly correlated between periods[12] (r=0.902; p=0.001), for the homicide rates the association between 2006 and 2012 was weak but statistically significant (r=0.339; p=0.001). As graph M in figure 5 showed, municipalities with low violence in 2012 were also non-violent in 2006. Likewise, those with high violence[13] were

[12] As previously shown, the correlation is also strong between the multidimensional and assets poverty measures (0.856, p<0.001).

[13] "High violence" was defined as a municipality with a homicide rate of 25 and above and "Low violence" is a municipality with a homicide rate of 10 or below.

more violent in 2006 but the variance was greater, suggesting greater change in this group. Notably, the homicide rate *change* was not correlated with the homicide rate in 2006. Graph N revealed no differences in the homicide rates of 2006 between the municipalities that suffered sharp changes when compared to those that didn't.

Based on other countries' experiences, the theoretical expectation was that violence and poverty would be correlated. However, in Mexico, the homicide rates for 2012 were not correlated with any of the 2010 poverty and income inequality measures; see Graph O in Figure 5. The association was actually the other way around and municipalities with low violence had a higher median percentage of poverty than municipalities with high levels of violence. Nonetheless, the 2005 poverty measure was positively correlated with the average homicide rate of 2006. These results indicated that the theoretical association was present before 2006 and that a different factor, besides poverty, drove the homicide rates up. Graph P demonstrates that homicide rates were lower in non-poor municipalities in 2002 and 2006, however, by 2012, the increase was greater in the affluent municipalities and thus the difference turned the other way around. Importantly, both poverty measures had a *negative* correlation with the *change* in homicide rates. This means that wealthy municipalities experienced greater change than poor municipalities in their homicide rates. Graphs Q and R showed how municipalities with high percentages of multidimensional poverty experienced, on average, less *change* in the homicide rates and this was not due to their 2005 level of income-poverty. Another way to explain this key aspect is in graph S. The poorest municipalities that had high levels of *change* in the homicide rates were already violent in 2006. However, the non-poor municipalities (75%) did not differ much in their 2006 homicide rate and yet some had important *changes* in their violence levels but others did not.

Nor the inequality coefficient or population density variables were associated with the homicide rates or their *change*.

Table 6
Correlations Between Municipality-level Predictors

	HR 2012	**HR 06**	**Multid. poverty 2010**	**Assets poverty 2010**	**Assets poverty 2005**	**GINI 2010**	**GINI 2005**	**Pop. density 2010**	**Pop. density 2005**
HR 12	1	--	-0.056	-0.161	-0.109	-0.133	-0.071	-0.095	-0.089
HR 06	0.339*	1	0.378*	0.285*	0.268*	-0.126	-0.054	-0.009	-0.002
HR *change*	0.869*	-0.17	-0.258*	-0.319*	-0.255*	-0.072	-0.046	-0.095	-0.092

*= P<0.05. HR= Homicide rate; Multid.= Multidimensional; Pop= Population

5.1.4 Validation process

The validation process was important to asses if the dependent variables measure what they intend to measure (DeVellis, 2016). There is no gold standard to evaluate stress, so the validation process tested how sensitive the indices and the single biomarkers were to the different sets of independent variables. A thorough description of the analyses is presented in Appendix 3. These were the main results.

Validation analyses at the individual level showed that the indices and the single biomarkers were sensitive to multiple indicators of health (chronic diseases, subjective health, depression, BMI, and hours of sleep); as health worsened, the dependent variables increased. Hours of sleep had a very weak relationship with the indices and blood pressure was unrelated to depression. Results were similar with the economic set of variables. The dependent variables had a negative association with household expenditure, confirming the theoretical expectation of a socioeconomic gradient with health at the individual level (M. Marmot, 2015); graph B in Figure A3.1 in Appendix 3. However, waist circumference showed a mild gradient with expenditure in the opposite direction. Household economic shocks had very low correlations with the dependent

variable (except for waist circumference) and unemployment was not a useful variable for validation purposes.

The most problematic results came with the violence variables: personal and property victimization, fear of crime, perception of a recent violence increase, and collective efficacy. They were statistically associated with the dependent variables, but consistently in the opposite direction. For example, a person who was personally victimized had a 1.6 *lower* Framingham score and one who suffered property victimization had a 0.6 *lower* score than one who was not victimized. The same happened with the CVDR index, waist circumference and blood pressure, even when it referred to subjective appreciations as with fear of crime. The explanation was that the correlations are confounded by the economic gradient of health. The poorest individuals, first and second economic quintiles, had worse levels on these biomarkers, as the gradient showed. However, the more affluent people, fourth and fifth economic quintiles, suffered on average more personal assaults and reported higher levels of fear than those in poorer quintiles; see graph C & D in Figure A3.1 in Appendix 3. People who reported more violent events were also the ones in the population stratum with lower cardiovascular risk. Therefore, poverty and violence were confounded in the validation process and appropriate modeling to control them was needed.

Results of the validation analysis at the municipality level were mixed. While the indices did not seem to be ideal measures to capture context effects, single biomarkers mildly reacted to them. The Framingham score was not significantly associated with any of the independent variables at the municipality level. CVDR was responsive to the homicide rate *change* and, unexpectedly, was negatively associated with multidimensional poverty, suggesting that poorer municipalities were not as stressful as richer ones. Waist circumference had the expected associations with violence but also the opposite one with contextual poverty. Blood pressure was sensitive to *changes* in the homicide rates and diastolic blood pressure had a negative association

with poverty. The *change* variable was the most promising predictor and population density the least, followed by the GINI coefficient.

5.1.5 Between-person and between municipality associations with the Framingham score

The cross-sectional multilevel analysis showed the effects of the variations between people and between municipalities on the Framingham score for 2012. These models explained the differences on the dependent variable by municipality once individual differences were controlled for. The main hypothesis is that violent municipalities have higher population means on the Framingham score than peaceful municipalities after adjusting for several confounders at both levels.

The null model, number 1 in Table 7, indicated that the unadjusted average of the Framingham score, when accounting by the municipality nesting, is 7.65. The variance at the municipality level was 4.4% of the overall variance; very small but significant. This amount of variation was what municipality-level predictors aimed to explain. The significance of the variation is better illustrated in Graph T in Figure 5, where the Framingham score of each municipality was placed in ascendant order. Most municipalities were similar but the small municipality-level variance reflected the significant differences at both ends of the graph. The present analysis sought to explain the differences between these municipality-level averages in the Framingham score.

At the individual level, Models 2-7 showed a significant negative association between household expenditure and the Framingham score. Fear of crime was not significantly associated with the Framingham score in any of them. Collective efficacy had a positive association in all models. In contrast with the theoretical expectations, but in accordance with the previous empirical findings, people who were personally victimized had 1.39 lower scores in the

Framingham index than those who were not (controlling for personal and contextual socioeconomic status); estimates were similar in the rest of the models. Property victimization was not significant. These associations were consistent across models.

The variance section of Table 7 indicated that household expenditure, fear of crime and collective efficacy were tested in every model to assess if the relationship with the Framingham score was random i.e. if it varied by municipality. They were never significant. This means that the relationship between these variables and the Framingham score was statistically equal across municipalities, therefore the same slope works equally well in every context.

Each model examined different combinations of the municipality-level predictors. Model 2 showed that municipalities with higher homicide rates in 2012 scored higher in the Framingham measure; a one-unit increase in the homicide rate increased the Framingham score by 0.01 units. The association was controlled with municipalities that had an average multidimensional poverty, which was not related with the Framingham score. The context effect was that two similar persons, none of them victimized, but one living in a municipality with 10 additional points in the homicide rate, scores 0.1 higher in the Framingham index than the other i.e. a 1.5% increase; see graph U. Model 3 tested if the relationship between homicide rates and the Framingham score was confounded by income-inequality. The results showed that the association only became weaker, 0.009, but still significant, and inequality was not statistically significant.

Model 4 tested a different hypothesis i.e. if the amount of *change* in the homicide rates between 2006 and 2012 was statistically associated with the Framingham score. The 2006 homicide rate was not associated with the 2012 Framingham score. Nonetheless, for a municipality with an average homicide rate in 2006, an additional unit in the *change* homicide rate increased the Framingham index by 0.014. This means that for non-victims with average

socioeconomic status, living in a municipality with average multidimensional poverty, for every 10 additional points of homicide rate *change* there was a 7.3% increase in the Framingham score. The context effect was appreciated among victims and non-victims (Graph V). Importantly, the multidimensional poverty index became statistically significant. This means that in an average municipality, more household expenditure was associated with a lower Framingham score. Moreover, if one compared two equivalent persons in household income and in the other controls, but one living in a municipality with higher multidimensional poverty by 10%, he or she would have a 0.11 higher Framingham score, a 2% increase; see graph W. However, this association with poverty became non-significant once inequality was accounted for in Model 5. Models not shown indicated that the assets poverty measure yielded very similar results; while it remained non-significant in all the models, the homicide rates for 2012 and the homicide rate *change* had the same coefficients.

Table 7

HLM Models Showing Between Municipality Predictors of the Framingham Score 2012 when Person-level Covariates are Controlled

Person-level predictors	**Framingham score**						
Municipality-level predictors	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6	Model 7
Intercept	7.647**	7.179**	7.181**	7.037**	7.045**	7.194**	7.10**
	(0.149)	(0.105)	(0.104)	(0.116)	(0.117)	(0.122)	(0.114)
Homicide rate 2012		0.010*	0.009*				
		(0.005)	(0.005)				
Homicide rate change 06-12				0.014*	0.013*		
				(0.005)	(0.005)		
Homicide rate2006				-0.006	-0.006		
				(0.009)	(0.009)		
Multidimensional poverty 2010		0.005	0.004	0.011*	0.009		
		(0.0052)	(0.005)	(0.005)	(0.005)		
Inequality 2010			-2.026		-1.579		
			(2.598)		(2.639)		
Low (CH) HR & low MP						-0.806**	0.069
						(0.204)	(0.892)
Low (CH) HR & high MP						-0.455*	-0.062
						(0.208)	(0.337)
High (CH) HR & low MP						0.535◊	0.569*
						(0.312)	(0.282)
High (CH) HR & high MP						0.361	0.527

						(0.316)	(0.318)
Household expenditure		-0.0001**	-0.0001**	-0.0001**	-0.0001**	-0.0001**	-0.0001**
		(0.00002)	(0.00002)	(0.00002)	(0.00002)	(0.00002)	(0.00002)
Fear of crime		-0.0179	-0.0178	-0.0183	-0.018	-0.017	-0.019
		(0.039)	(0.039)	(0.039)	(0.039)	(0.039)	(0.039)
Collective efficacy		0.045*	0.045*	0.045*	0.045*	0.045*	0.045
		(0.015)	(0.015)	(0.015)	(0.015)	(0.015)	(0.015)
Personal victimization		-1.396**	-1.398**	-1.378**	-1.380**	-1.418**	-1.407**
		(0.219)	(0.219)	(0.219)	(0.219)	(0.217)	(0.218)
Property victimization		-0.352	-0.351	-0.357	-0.351	-0.362	-0.342
		(0.292)	(0.291)	(0.292)	(0.291)	(0.292)	(0.292)
Variance							
Municipality level (U_0)	0.791**	0.447	0.440	0.439	0.434	0.384	0.484
Household expenditure		0.000	0.000	0.000	0.000	0.000	0.000
Fear of crime		0.028	0.028	0.028	0.028	0.020	0.022
Collective efficacy		0.005	0.005	0.005	0.005	0.005	0.079
Individual level (r)	16.821	15.647	15.647	15.643	15.643	15.669	15.696
R^2	4.4%	2.5%	2.4%	2.4%	2.4%	2.1%	2.7%

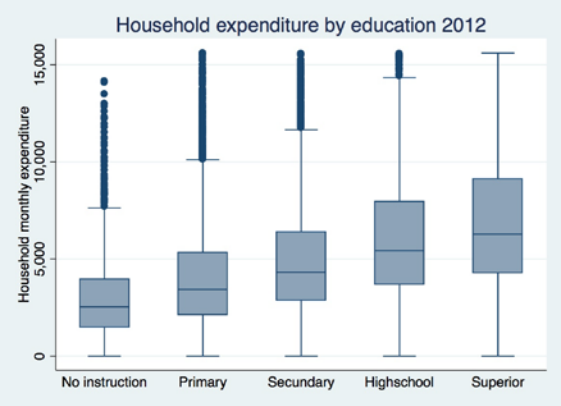
a= Grand-mean centered; b=uncentered; ◊=p<.10;*=p<.05; **=p<.01. All with robust std errors and 10 MI datasets. Controlling by: single, indigenous, disability**, household size**, and chronic diseases**; and population density at municipality level. 17.612

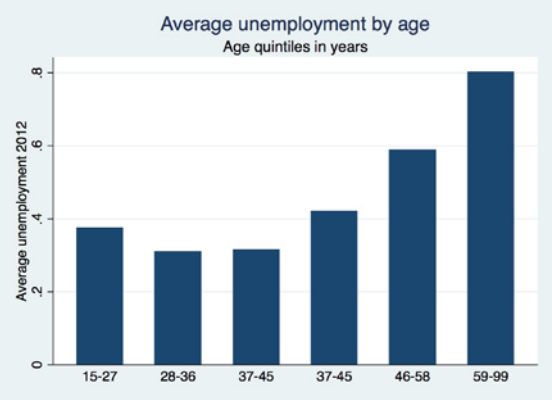
The last hypotheses concerned the interaction effects of poverty and violence. An attempt to model the multiplicative effect of multidimensional poverty with the homicide rate and then with the change in homicide rates did not yield significant results. Therefore, the next step was to try the dummy variables representing these interactions.

Model 6 showed a partial confirmation of the stress gradient. Municipalities with low levels of poverty and low homicide rates in 2012 had significantly lower scores in the Framingham index, by 0.806, than the average municipality: 11% lower. Interestingly, municipalities with low homicide rates but high percentages of poverty, while controlling for population density, also had significant lower scores, -0.455, than the average municipality: 6.3% lower. Unexpectedly, municipalities with high homicide rates and high poverty percentages were not statistically different than the average municipality, although their score was higher. As in previous results, it was violent and affluent municipalities that had the highest scores. They were not statistically different from the average municipalities with an alpha of 0.05 (p=0.089). However, as graph X in Figure 5 shows, a different comparison highlighted key differences.

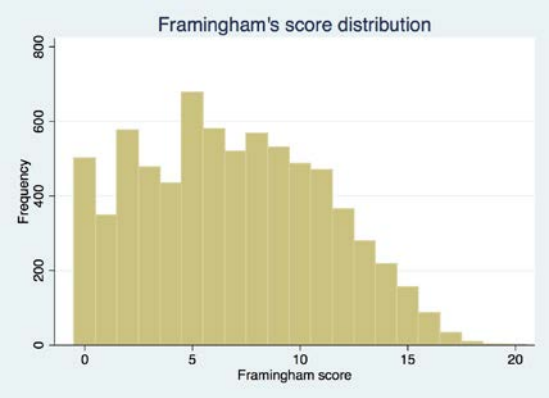
Municipalities with high homicide rates were not different among them, regardless the level of poverty. Notably, however, the difference between municipalities with low poverty differed by 1.34 in the Framingham score. This means that two poor municipalities, one with high violence and the other with low violence, have a 17% difference in their Framingham score when other covariates are controlled for. When comparing two municipalities with high levels of poverty, the difference was 10.8% higher in the violent municipalities; however, the confidence intervals indicated that this difference was not statistically meaningful. These results showed that high homicide rates in 2012 mattered more than high poverty percentages in explaining high scores in the Framingham index.

In Model 7, the 2012 homicide rate variable was substituted by the *change* variable and then combined with multidimensional poverty in the same way as in model 6. Results revealed that the only statistically significant difference with the average municipality was with municipalities with high *change* in homicide rates and low multidimensional poverty, which had higher scores by 8%. As in the previous model, municipalities with low *change* in the homicide rates had the lowest scores in the Framingham index. Unfortunately, the confidence intervals were too wide as to conclude that the difference was significant between high *change* and low poverty against low *change* and high poverty (Graph Y).

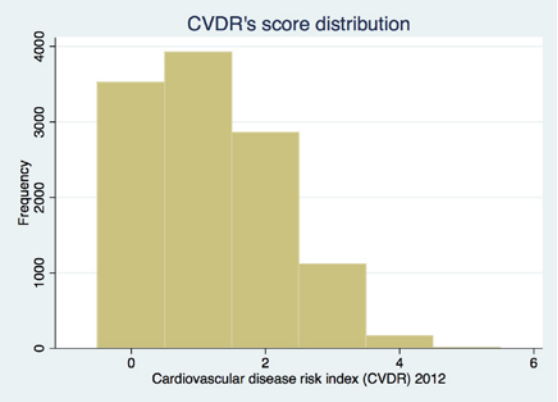




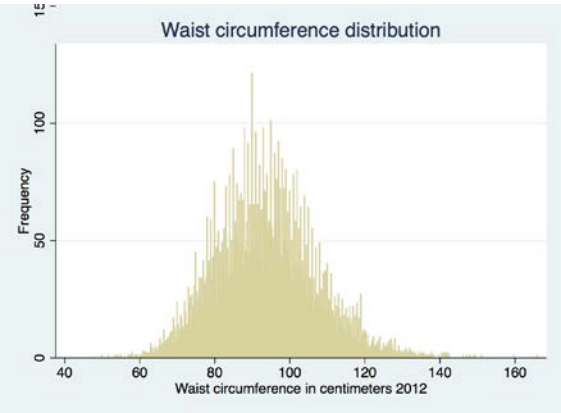
A


Framingham's score distribution
Frequency
0
200
400
600
800
0
5
10
15
20
Framingham score

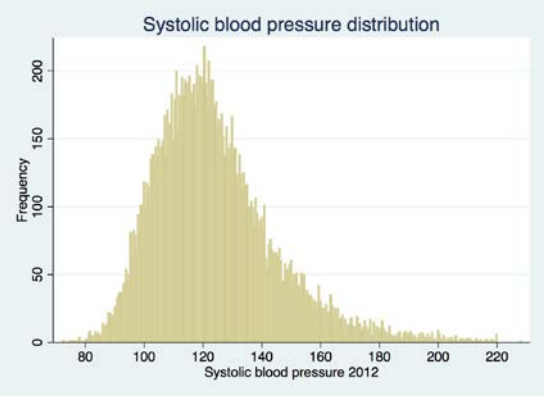
B


CVDR's score distribution
Frequency
0
1000
2000
3000
4000
0
2
4
6
Cardiovascular disease risk index (CVDR) 2012

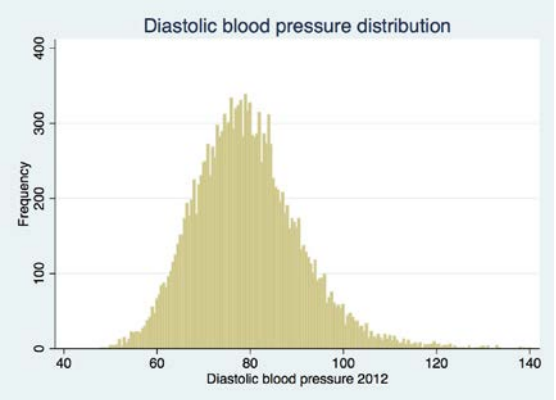
C


Waist circumference distribution
Frequency
0
50
100
40
60
80
100
120
140
160
Waist circumference in centimeters 2012

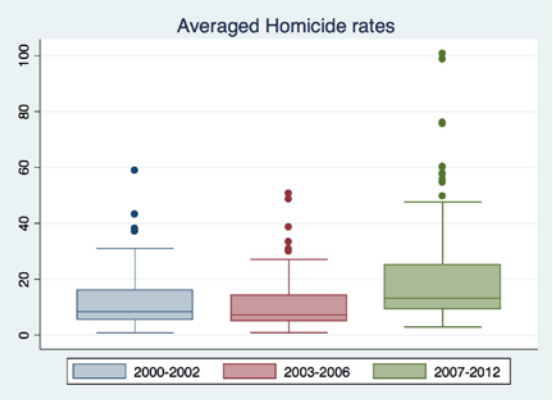
D


Systolic blood pressure distribution
Frequency
0
50
100
150
200
80
100
120
140
160
180
200
220
Systolic blood pressure 2012

E

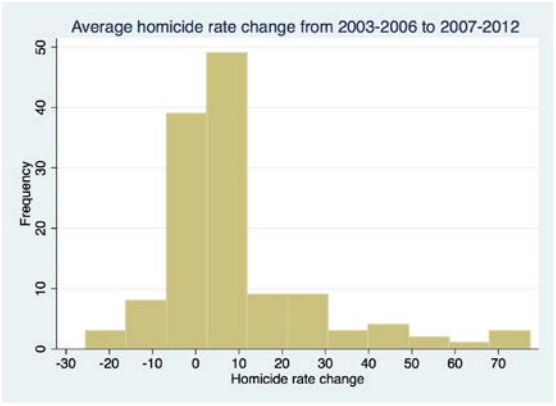

Diastolic blood pressure distribution
Frequency
0
100
200
300
400
40
60
80
100
120
140
Diastolic blood pressure 2012

F

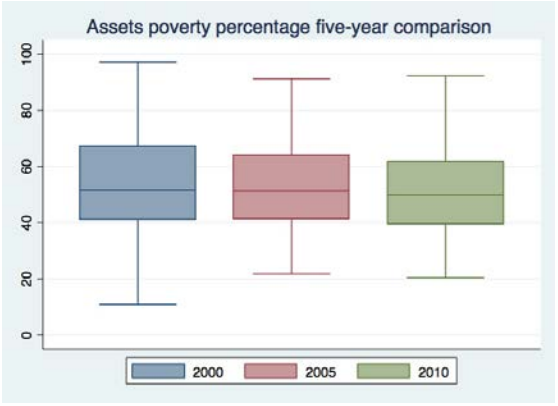

Averaged Homicide rates
0
20
40
60
80
100
2000-2002
2003-2006
2007-2012

G

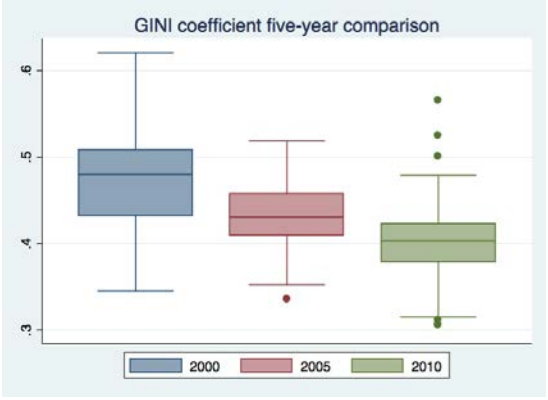
H


Average homicide rate change from 2003-2006 to 2007-2012
Frequency
0
10
20
30
40
50
-30
-20
-10
0
10
20
30
40
50
60
70
Homicide rate change

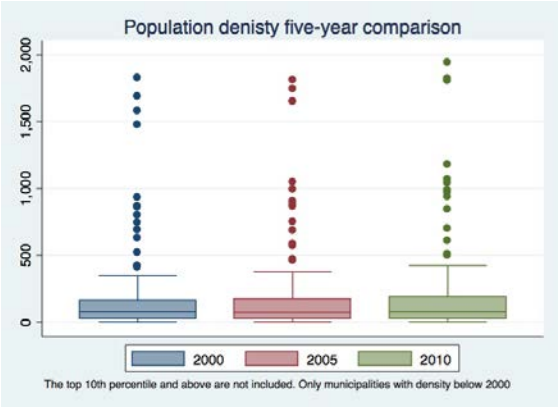
I


Assets poverty percentage five-year comparison
0
20
40
60
80
100
2000
2005
2010

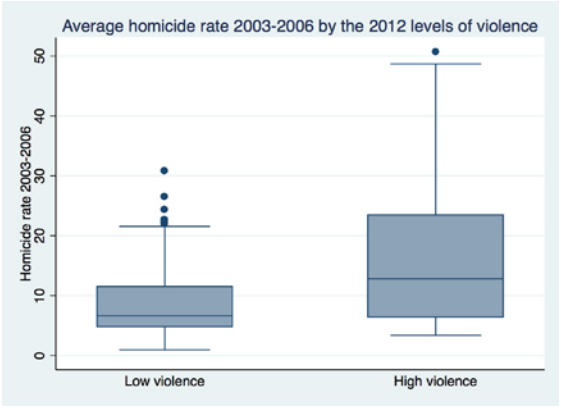
J


GINI coefficient five-year comparison
.3
.4
.5
.6
2000
2005
2010

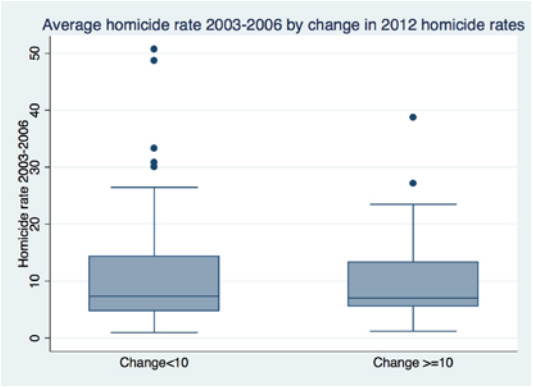
K


Population denisty five-year comparison
0
500
1,000
1,500
2,000
2000
2005
2010
The top 10th percentile and above are not included. Only municipalities with density below 2000

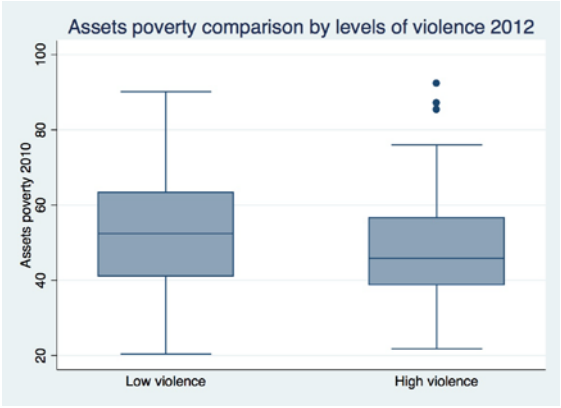
L


Average homicide rate 2003-2006 by the 2012 levels of violence
Homicide rate 2003-2006
0
10
20
30
40
50
Low violence
High violence

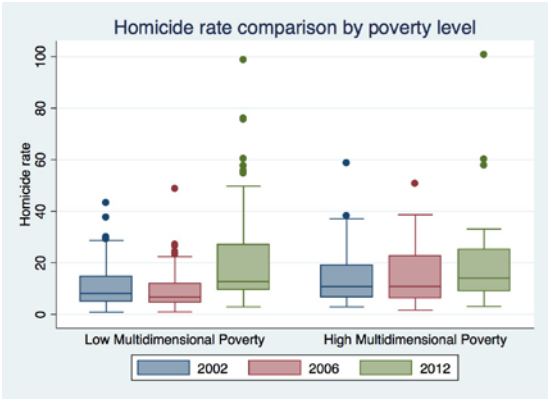
M


Average homicide rate 2003-2006 by change in 2012 homicide rates
Homicide rate 2003-2006
0
10
20
30
40
50
Change<10
Change >=10

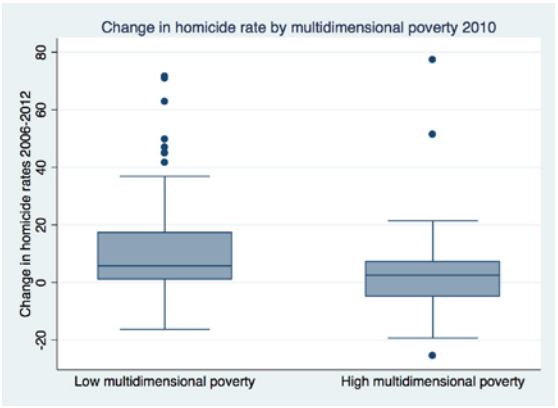
N


Assets poverty comparison by levels of violence 2012
Assets poverty 2010
20
40
60
80
100
Low violence
High violence

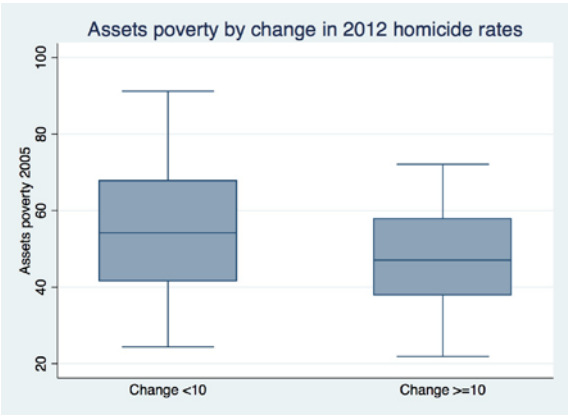
O


Homicide rate comparison by poverty level
Homicide rate
0
20
40
60
80
100
Low Multidimensional Poverty
High Multidimensional Poverty
2002
2006
2012

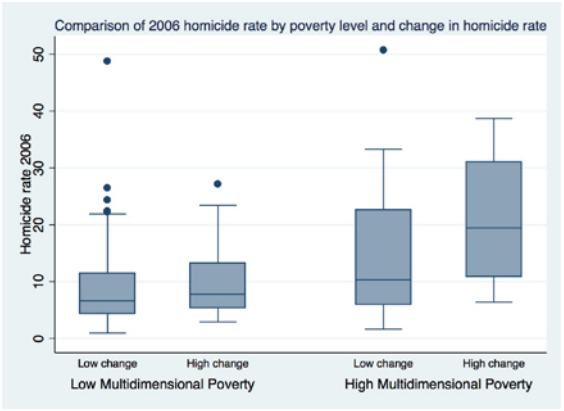
P


Change in homicide rate by multidimensional poverty 2010
Change in homicide rates 2006-2012
-20
0
20
40
60
80
Low multidimensional poverty
High multidimensional poverty

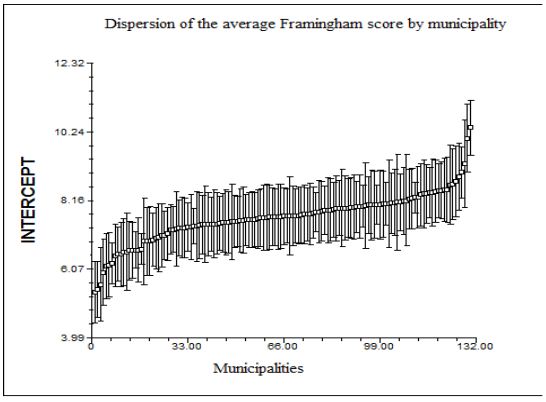
Q


Assets poverty by change in 2012 homicide rates
Assets poverty 2005
20
40
60
80
100
Change <10
Change >=10

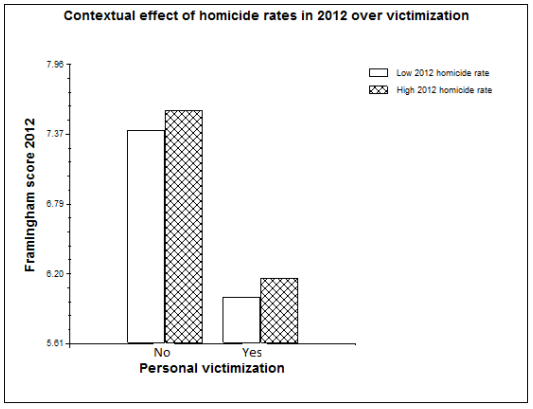
R


Comparison of 2006 homicide rate by poverty level and change in homicide rate
Homicide rate 2006
0
10
20
30
40
50
Low change
High change
Low change
High change
Low Multidimensional Poverty
High Multidimensional Poverty

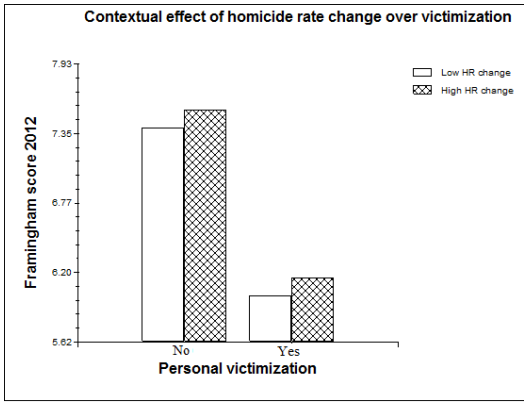
S


Dispersion of the average Framingham score by municipality
INTERCEPT
12.32
10.24
8.16
6.07
3.99
0
33.00
66.00
99.00
132.00
Municipalities

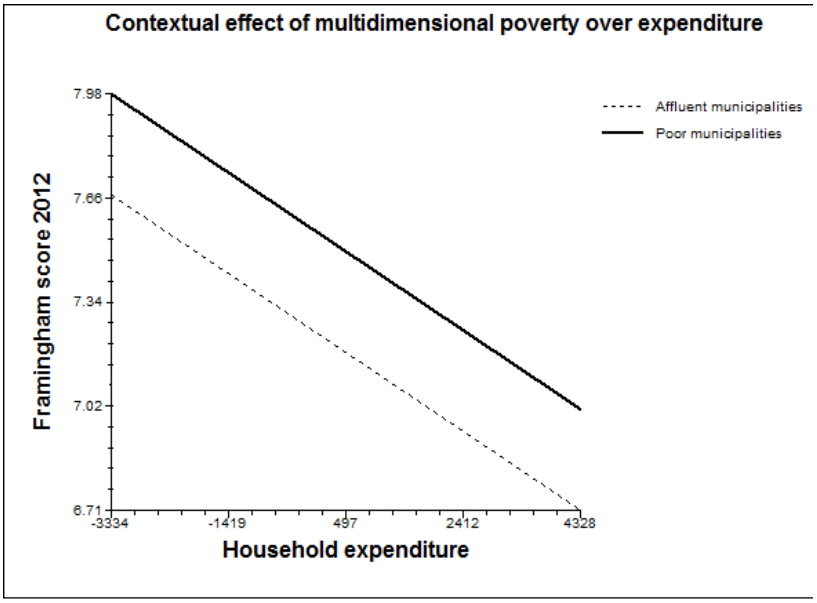
T


Contextual effect of homicide rates in 2012 over victimization
Framingham score 2012
7.96
7.37
6.79
6.20
5.61
Low 2012 homicide rate
High 2012 homicide rate
No
Yes
Personal victimization

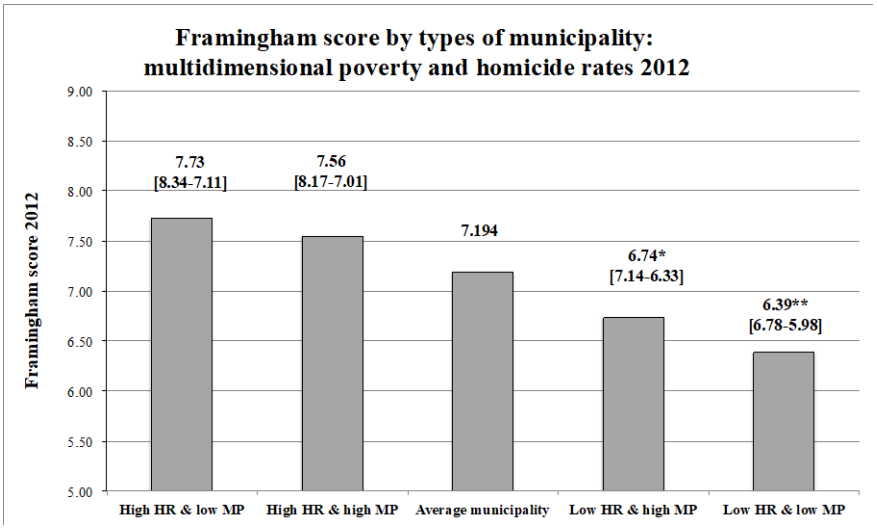
U


Contextual effect of homicide rate change over victimization
Framingham score 2012
7.93
7.35
6.77
6.20
5.62
Low HR change
High HR change
No
Yes
Personal victimization

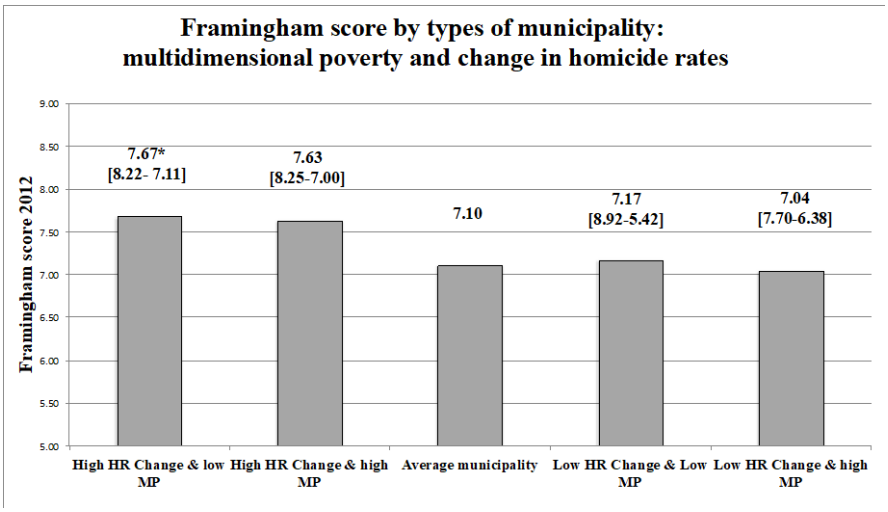
V


Contextual effect of multidimensional poverty over expenditure
Framingham score 2012
7.98
7.66
7.34
7.02
6.71
-3334
-1419
497
2412
4328
Household expenditure
Affluent municipalities
Poor municipalities

W


Framingham score by types of municipality:
multidimensional poverty and homicide rates 2012
Framingham score 2012
9.00
8.50
8.00
7.50
7.00
6.50
6.00
5.50
5.00
7.73
[8.34-7.11]
7.56
[8.17-7.01]
7.194
6.74*
[7.14-6.33]
6.39**
[6.78-5.98]
High HR & low MP
High HR & high MP
Average municipality
Low HR & high MP
Low HR & low MP

X



Y

Figure 5. Univariate and bivariate distributions and associations of independent and dependent variables that illustrate the exploratory analyses. Then the graphs depict the results from the HLM-sectional analyses. All person-level variables correspond to 2012.

5.2 Within- person analyses

The focus of the second section of the findings was to analyze the patterns of change in the dependent variables. Therefore, the first objective was to model within person differences. The Framingham score was the preferred dependent variable in the previous section because it included most of the biomarkers and was thus the strongest dependent variable available. However, two important disadvantages were its inherent adjustment by sex and age, and the fact that it was only available for 2012. Therefore, the CVDR was the preferred dependent variable when two waves were required and single biomarkers when three waves were needed. The first part of the second section describes simple mean differences in the CVDR to assess how much between and within variation the index captures. Key time-varying independent variables were also described.

Afterwards, the analyses examined the influence of predictors on change at the person-level. Selected municipality characteristics were used as "treatments" of a natural experiment that

divided the population in two groups with measures before and after the exposure. Difference-in-difference models were conducted in order to establish the sensitivity to three different treatments, each with three thresholds, and to its interactions.

Finally, the analyses examined change at the person and at the municipality levels, simultaneously, using growth curve models. Since these models required at least three data points, the three waves were used. However, only single biomarkers were available, therefore the dependent variables were: waist circumference and the two types of blood pressure. These models aimed to integrate the between and within variance with all the key independent variables.

5.2.1 Person-level univariate longitudinal change

The longitudinal comparison of time-varying person-level variables between 2006 and 2012 was made in several ways; see Table 8. First, the means with their confidence intervals for the two waves were estimated with 20 datasets from multiple imputations. The third column in table 8 displays the percent change of the means between both periods. While the t-tests or chi-square in the fourth column indicated if the change was meaningful, the large number of observations due to multiple imputations made the slightest difference statistically significant. Therefore, the difference in the confidence intervals was more reliable[14]. The next comparison was with the variance partition; the between-person standard deviation reflected the variations across individuals and the within person standard deviation corresponded to the variation in time on the

[14] The problem is that STATA allows the estimation of the means with multiple imputation but it does not allow the estimation for the t-test or the chi-square test. Therefore, for the difference tests STATA considers an artificially large dataset (Nx20). Thus, the means and CIs were appropriately estimated.

same individual. For the dichotomous variables, percentages indicate the transitions from one state to the other[15].

The vector of chronic diseases increased by 36% from 2006 to 2012. The variance between persons (0.22) was higher than the one within the same individual over time (0.17). Household expenditure had a very small increase (1.3%). Although the t-test indicates that the difference was significant, the confidence intervals overlapped, suggesting it was not meaningful. As expected, the differences between persons were larger than the differences in expenditure from 2006 to 2012. Household economic shocks increased by 33.4% and 30% of the respondents reported an economic shock at some point during both periods, either 2006 or 2012. Among them, 46.5% reported an economic shock in both periods and 32% did not experience one in 2006 but suffered one by 2012.

Table 8

Mean and variation comparison of person-level variables between the 2006 and 2012 waves

	Mean/ Proportion 2006 [CI]	Mean/ Proportion 2012 [CI]	% diff.	Diff. test.	Between person variation	Within person variation	% changed to other
Chronic diseases [a]	0.066	0.095	36%	-49.86	0.22	0.17	
	[0.06-0.07]	[0.09-.10]		P=0.00			
Household expenditure [a]	4,669.3	4,733.7	1.3%	-14.9	2,875	1,612	
	[4611-4726]	[4687-4780]		P=0.00			
Household economic shock [b]	24.81%	34.76%	33.4%	2.1	30.2%	46.5%	32%
	[24.08-25.52]	[34.03-35.48]		P=0.00			
	[35.20-36.65]	[31.66-33.08]		P=0.00			
Personal victimization[b]	7.32%	8.36%	13.2%	6.6	7.8%	12.4%	5.3%
	[6.86-7.78]	[7.87-8.86]		P=0.00			
Household victimization[b]	3.76%	4.77%	23.7%	1.6	4.3%	7.12%	4.4%
	[3.43-4.09]	[4.43-5.12]		P=0.00			
Fear of	1.144	1.322	14.4%	-7.42	1.71	1.45	

[15] In STATA, with the dataset in long form, the partition of variance was done with the command xtsum. The transitions of the dichotomous variables were conducted with xttab and xttrans.

crime[a]						
	[1.11-1.17]	[1.29-1.35]		P=0.00		
Collective efficacy [a]	15.99	15.72	1.7%	12.34	4.26	2.68
	[15.90-16.08]	[15.64-15.80]		P=0.00		
CVDR [a]	0.671	1.181	55.1%	-1.9	0.85	0.48
	[0.65-0.68]	[1.16-1.19]		P=0.00		
Waist [a]	90.92	93.68	3.0%	-1.6	12.74	4.53
	[90.67-91.17]	[93.48-93.88]		P=0.00		
Systolic [a]	117.60	123.97	5.3%	1.8	16.74	9.72
	[117.3-117.8]	[123.6-124.2]		P=0.00		
Diastolic [a]	75.63	79.34	4.8%	-1.5	9.58	6.35
	[75.44-75.83]	[79.17-79.51]		P=0.00		

Means were estimated with 20 datasets from multiple imputation. A=mean & ttest/ b= proportion & chi-square; results are in the fourth column, "diff-test" for difference test.

Personal victimization had a significant increase of 13.2% by 2012. Almost 8% of the sample was victimized at some point in both periods; 12.4% of them were victimized in both periods and only 5% of the total was not victimized in 2006 but suffered one by 2012. Household victimization had a significant 23.7% increase but only 4.4% of these respondents did not suffer a victimization event in 2006 but did in 2012. Fear of crime had a 14% increase during this period and the variation was similar within than between persons. Collective efficacy did not change on average. However, there was considerable variation within persons during this period (2.68).

Surprisingly, the dependent variables showed different patterns of change. The CVDR had a 55% increase; only one third of the variation was due to changes within persons (0.48) and two thirds was variation between persons (0.85); see Graph A in Figure 6. The single biomarkers, waist circumference and the two types of blood pressure, had small but significant increases between periods (3%-5%). Most of the variation was between persons.

5.2.2 Difference in differences for the CVDR index

The difference-in-difference model provided the estimation of within person change in comparison to a "treatment" i.e. some distinct circumstance that may explain different pathways

of variation. The model also partitioned the cross-sectional and the longitudinal variance. This modeling approach was supported by the previous findings in the descriptive longitudinal statistics, which showed that the CVDR had between and within variance.

A null model of the CVDR index in Table 9 indicated that the unadjusted grand mean for both waves was 0.906. More importantly, the null model confirmed that the partition of variance was necessary: the difference between the two sources of variance was statistically significant and the intra class correlation meant that 32.1% of the variance was between persons and 67.9% of the variance was within persons. Therefore, the mixed estimation method was warranted. All the following difference in difference models had a statistically significant variance partition.

The first group of models aimed to establish a meaningful threshold for each of the three treatments: homicide rates, homicide rate *change*, and multidimensional poverty. The results for the difference in difference models showed that the most meaningful threshold for the homicide rate was 35; for *change* it was 10, and for multidimensional poverty it was 65. The next paragraphs describe how the analyses reached these conclusions.

Once the null models established the ICC, the analyses started assessing the 2012 homicide rates, first with a threshold at 15, then at 25, and finally at 35. The second column in Table 9 starts with the CVDR intercept, which represented the CVDR mean at time 0, in 2006 (0.647), in the control group, for the municipalities with homicide rates below 15. The time effect represented the first difference or the mean change from time 0 to time 1, i.e. from 2006 to 2012, in the control group. In the three models the CVDR means of the control groups significantly increased by 0.487 or 0.490 from 2006 to 2012. The second difference was the group effect or the difference between the control group and the treatment group at time 0 i.e. the CVRD mean difference in 2006 between the municipalities with homicide rates above and below a homicide rate of 15. The objective in natural experiments is to find no significant differences between both

groups at baseline. The second difference was not significant for the 15 and 25 thresholds but it was for 35. This means that municipalities with homicide rates above 35 were not equal in 2006 and the treatment group started with a higher mean by 0.046. The key coefficient was the difference in difference. It represented the mean CVDR difference between groups at time 2. This third difference was not significant in the 15 and 25 thresholds. However, it was very close to statistical significance in the highest threshold (p=0.071). See graph E in Figure 6 for the mean estimates and comparison. The CVDR mean at Time 2 in the control group was 1.138 and in the treatment group was 1.233. The results meant that, on average, everyone increased their CVRD means between 2006, but the increase was 8.3% higher for people living in a municipality with homicide rates above 35.

The second part of Table 9 displays the results for the thresholds of the homicide rate *change*. The baseline means for the control groups (0.64) and the time increase (0.483-0.491) were very similar for the three thresholds. As expected, the group effect was not significant at baseline; except for the third model, with the threshold at 30. The third difference was statistically significant in the thresholds at 10 and 20. Interestingly, the difference was of equal size (0.052) regardless of the change in the intensity of the threshold. Therefore, the effect of the exposure was similar above a 10-point increase in the homicide rates. See graph F in Figure 6. This difference was not significant at the maximum threshold, perhaps because of lower statistical power; it only grouped 11.5% of the municipalities. However, at the lowest threshold, the narrative was even more straightforward than in the previous treatment. In 2006 people were equivalent in their CVDR means and everyone had a significant increase by 2012. Nevertheless, the increase was 7% higher in people living in municipalities that experienced a change in their homicide rates of more than 10 points.

The third part of Table 9 used thresholds of multidimensional poverty to compare the means of the CVDR index. The results were very similar regardless of the level of the threshold. The three models evidenced the important increase in the index between 2006 and 2012 (0.481-0.494). The group effect was always significant but in the opposite direction. The hypothesis indicated that the higher the poverty threshold, the more stressful environment and thus a higher mean CVDR index. However, poor municipalities did not seem to be more stressful but less so than more affluent ones. Nevertheless, there was no significant difference in either group with the interaction of group and time.

Table 9

Difference in difference null model and models without covariates for the three treatments

	Null	**HR>15**	**HR>25**	**HR>35**
CVDR Intercept	0.906**	0.647**	0.652**	0.648**
Time effect		0.487**	0.490**	0.490**
Group effect		0.188	0.016	0.046*
Diff in Diff		0.026	0.033	0.049◊
Residual variance	0.623	0.512	0.511	0.511
Random Intercept variance	0.321**	0.363**	0.363**	0.363**
ICC	34%			
		HRch>10	**HRch>20**	**HRch>30**
CVDR Intercept		0.647**	0.648**	0.646**
Time effect		0.483**	0.487**	0.491**
Group effect		0.028	0.033	0.069*
Diff in Diff		0.052*	0.052*	0.046
Residual variance		0.511	0.511	0.512
Random Intercept variance		0.363**	0.363**	0.362**
		MPov>45	**MPov>55**	**MPov>65**
CVDR Intercept		0.713**	0.694**	0.685**
Time effect		0.481**	0.494**	0.489**
Group effect		-0.116**	-0.109**	-0.155**
Diff in Diff		0.032	0.004	0.042
Residual variance		0.512	0.512	0.512
Random Intercept variance		0.361**	0.361**	0.360**

Estimations were done without multiple imputation. ◊=p<.10; *=p<.05; **=p<.01.HR= Homicide rate; HRch= Homicide rate *change;* MPov= Multidimensional poverty. The numbers in the columns refer to the thresholds, rates for homicides, and percentages for poverty.

5.3 Differences with covariates

The models in Table 10 honed into these results to examine if these contexts had heterogeneous effects among subpopulations. In other words, it tested if the context effects varied when other sociodemographic controls were introduced. The same threshold models for the three contextual treatments were estimated for males and females separately. In addition, each gender group was adjusted by age. Therefore, these results reflect the mean differences on people under 40 years old.

Important gender dissimilarities were appreciated in the baseline mean for the control groups in the two treatments with homicide rates: females scored on average 0.433 in the CVDR index while males scored 0.511. Moreover, both groups increased their means between 2006 and 2012 but the raise was larger in women, 0.720, than in men, 0.632. Likewise, the effect of age was significant in the two groups but it was larger for females, 0.376, than for men, 0.251. The coefficient meant that females older than 40 years had a 0.376 higher score than younger females in the control group at baseline. As expected, in the first two treatments, the differences with the control groups were not significant at baseline.

The key results for the homicide rate treatment was in the 35 threshold: the third difference was significant for females (0.78) but not for males. Females living in municipalities with a homicide rate above 35 had a 10.6% higher CVDR mean than females living elsewhere; see graphs G & H in Figure 6. Thus, the 0.046 difference in graph E was largely driven by females. When personal victimization was added as a third covariate, the model became non-significant for females with less than 40 years of age who were not victimized between 2006 and 2012. The third difference coefficient dropped from 0.78 to 0.67 ($p=0.085$). The coefficient for men remained non-significant. After removing personal victimization, 5 new models were estimated to assess the mean differences in each economic quintile. Interestingly, they were very

similar along the socioeconomic gradient; nonetheless, the largest coefficient was in the poorest quintile (0.083) and the lowest in the third quintile (0.074). The gradient actually showed a U-curve going back up to a difference of 0.079 in the fifth quintile. All coefficients were statistically significant. Economic shocks did not make an important difference since the coefficients were non-significant.

A similar heterogeneity could be observed in the homicide rate *change* treatment. In the above-10 threshold the third difference was significant, 0.059, only for women and not for men. The size of the difference was larger than in the models without covariates, thus indicating that females were driving it. The size of the difference was the same in the next threshold, 0.059 (p=0.074), and in the maximum threshold it was even larger, 0.69 (p=0.078); however, they were not significant at a 95% confidence interval. When the personal victimization covariate was added, the coefficient dropped from 0.059 to 0.047 and stopped being significant. Men did not change. Apparently, the association may be confounded by the victimization stress. However, as it was previously discussed, the victimization coefficient was -0.321 and significant. Therefore, victims had lower means in the index, not higher. The assessment of the effect in each socioeconomic quintile revealed a more traditional socioeconomic gradient but it was very smooth i.e. the differences between quintiles are minimal. The largest coefficient was in the poorest quintile, .083, and the lowest in the wealthiest quintile, 0.058. The p-values from the second to the fifth quintiles ranged from .052 to .064. Economic shocks were not important covariates.

In the poverty models, the intercepts or baseline means were higher than in the previous models. Moreover, the group effect was significant and, unexpectedly, the mean was lower than in the control group. The CVDR mean was 38% lower for females and 22.5% lower for males in municipalities with poverty levels above 45. The third difference was not meaningful for women.

However, the difference was significant for men at the 65 threshold. As graphs I and J from figure 6 show, the lower mean at baseline meant that the third difference was also below the control group for men. These poverty contexts were independent for women's stress and seemed to be *less* stressful for men by 10%. The addition of victimization left women unchanged and reduced the men's coefficient from 0.087 to 0.059 and lost statistical significance. The victimization coefficient was negative and significant. The gender difference remained in the quintile-by-quintile assessment. For men, all the coefficients were significant and the difference increased with the quintiles; from 0.079 in the lowest quintile to 0.094 in the highest quintile. Economic shocks did not change these estimates.

Table 10
Difference in Difference Models with Covariates for the Three Treatments

	Females			**Males**		
	HR>15	**HR>25**	**HR>35**	**HR>15**	**HR>25**	**HR>35**
CVDR Intercept	0.430	0.431	0.434	0.513	0.510	0.503
Time effect	0.720**	0.721**	0.719**	0.627**	0.632**	0.641**
Group effect	0.026	0.11	0.045	-0.005	0.002	0.051
Diff in Diff	0.026	0.043	0.078*	0.040	0.044	0.017
Age >40	0.376**	0.376**	0.377**	0.252**	0.251**	0.251**
	HRch>10	**HRch>20**	**HRch>30**	**HRch>10**	**HRch>20**	**HRch>30**
CVDR Intercept	0.429	0.433	0.433	0.509	0.506	0.498
Time effect	0.715**	0.719**	0.723**	0.629**	0.632**	0.642**
Group effect	0.042	0.036	0.060	0.007	0.023	0.098
Diff in Diff	0.059*	0.059◊	0.069◊	0.051	0.051	0.005
Age >40	0.377**	0.376**	0.377**	0.251**	0.261**	0.251**
	MPov>45	**MPov>55**	**MPov>65**	**MPov>45**	**MPov>55**	**MPov>65**
CVDR Intercept	0.498	0.479	0.467	0.573	0.552	0.543
Time effect	0.714**	0.727**	0.725**	0.620**	0.637**	0.633**
Group effect	-0.115**	-0.108**	-0.144	-0.129**	-0.125**	-0.205**
Diff in Diff	0.033	0.007	0.036	0.052	0.025	0.087*
Age >40	0.377**	0.377**	0.379**	0.257**	0.257**	0.263**

Estimations were done without multiple imputation. ◊=p<.10;*=p<.05; **=p<.01.HR= Homicide rate; HRch= Homicide rate *change;* MPov= Multidimensional poverty. The numbers in the columns refer to the thresholds, rates for homicides, and percentages for poverty. Age is in years.

Once the thresholds were established and a few patterns of heterogeneity were identified, the next step was to establish the equivalence at baseline on other variables besides CVDR. The first column in Table 11 compared the means or proportions of several person-level independent variables in 2006 divided by the 35-threshold in 2012 homicide rates. The violent municipalities had very few indigenous populations and smaller household sizes compared with the peaceful ones, both indicators of greater poverty in the peaceful municipalities. Accordingly, the education levels starting in primary were slightly higher in the violent municipalities. Household expenditure was higher in violent municipalities by about 10 dollars. Surprisingly, both types of victimization were lower in the peaceful municipalities. Collective efficacy was a point higher in the violent municipalities.

The *change* variable had similar differences. The share of indigenous population was lower, household size was smaller, and education higher in municipalities with a homicide rate *change* above 10. Notably, the municipalities with more *change* had fewer chronic diseases than the stable municipalities. Even though these variables suggest that stable municipalities were poorer, household expenditure did not have significant differences and people in municipalities above the *change* threshold suffered less economic shocks and fewer personal or property assaults. Collective efficacy was also a point higher in the municipalities with greater change in homicide rates.

Table 11

Mean and proportion differences at baseline for the significant thresholds.

Baseline measures: 2006	**Homicide rate: 35**		**Homicide rate change: 10**		**Multidimensional Poverty: 65**	
	Below N=26,046	**Above** N=4,925	**Below** N=22,065	**Above** N=8,906	**Below** N=25,353	**Above** N=5,618
Male	43.7%	44.0%	43.7%	43.9%	43.6%	44.3%
Age	44.9	45.2	44.9	44.9	44.6	46.5**
Indigenous	8%	0.4%**	8.5%	2.5%**	3.9%	22.5%**

Disability	2.2%	2.4%	2.2%	2.5%	2.2%	2.8%
Household size	5.37	5.10**	5.42	5.11**	5.23	5.76*
Single	31.7%	29.4*	31.5%	30.8%	31.6%	30.1%
Chronic diseases	0.079	0.069	0.081	0.069*	0.081	0.062*
Education: No instruction	10.2%	7.3%	10.8%	7.2%	7.5%	19.9%
Primary	42.6%	43.4%	43.2%	41.5%	40.3%	53.9%
Secondary	25.2%	26.3%	24.5%	27.4%	27.5%	15.5%
High school	13.0%	14.0%	13%	13.6%	14.4%	7.7%
College or higher	8.9%	8.9%**	8.4%	10.3%**	10.3%	2.9%**
Household expenditure	4,627.8	4,796.9*	4,649	4,667	4,947	3,365**
Household Economic shocks	25.8%	19.5%**	26.8%	19.8%**	24.7%	25.4%
Personal victimization	7.5%	6.2%*	7.7%	6.4%*	8.3%	3.0%**
Property victimization	3.9%	2.8%*	4.1%	2.9%*	4.0%	2.6%**
Fear of crime	1.14	1.13	1.15	1.12	1.23	0.73**
Collective efficacy	15.83	16.82**	15.73	16.6**	15.84	16.66**
Municipality level baseline measures	**Below** N=111	**Above** N=19	**Below** N=93	**Above** N=37	**Below** N=100	**Above** N=30
Homicide rates	10.5	13.6	11.1	10.7	9.7	15.1*
Assets Poverty	53.22	48.7	54.6	47.4*	46.7	71.8**
Income inequality	0.431	0.433	0.432	0.427	0.432	0.426
Population Density	1,259	800	1,376	359.1*	1,389	78.5*

*=p<.05; **=p<.01. Mean differences were conducted with T-tests. Differences in proportion were conducted with chi-squares. Education was tested as a continuous variable but it is displayed as ordinal to ease the comparison; it was always statistically significant. Estimations were done without multiple imputation. The numbers in the columns refer to the thresholds, rates for homicides, and percentages for poverty.

The poverty threshold had a different pattern. People living in poorer municipalities were two years older on average. The indigenous population was notably larger, as the percentage of people without instruction. Household expenditure was also lower by nearly a100 dollars. They also had less chronic diseases. They also experienced lower victimization rates and lower fear of crime. Again, collective efficacy was also a point higher in the poorest municipalities.

To sum up this section's results, the municipalities were not equal at baseline for the three thresholds. However, the differences were small and they did not seem to be important confounders. The differences with the poverty threshold only confirmed that the contextual measures mirrored to a great extent the person-level variables. Moreover, the violent municipalities and the ones with greater *change* in the homicide rates were richer at baseline, not

poorer. The other surprising finding was that victimization rates and collective efficacy did not reflect contextual violence or poverty: they were always lower and higher, respectively, in municipalities above any threshold. Therefore, there was no substantial evidence indicating that non-random assignment of the natural experiment was confounding the results of the difference – in- difference models.

Finally, table 12 displays the results for the dummy variables reflecting the interactions of the three main treatments (without covariates). The first set of models was the combination of high and low levels of poverty and homicide rates. The baseline averages of CVDR index in the control groups were very similar for the four models. Likewise, the time effect was large and consistent. The group effect had some key divergences. At baseline, the treatment and controls did not have significant differences when comparing municipalities with high violence. However, when both groups had low levels of violence, there was a positive difference at baseline of 15%. This difference was larger and negative in the group with low violence and high poverty (-0.174): the means were lower in the treatment group by 26% (see graph K). The only significant third difference was in the municipalities with high violence and low poverty: their increase in 2012 was 10% higher than in the control municipalities. The importance of the second and third differences can be appreciated in the graphs K and L from figure 6. The contrast between these types of municipalities shows a different pattern of differences. When violence was low and poverty high, the CVDR means were below the control group by 26% and this difference was meaningful since 2006. Conversely, municipalities with high violence and low poverty were equivalent to the control group in their CVDR means by 2006 but the increase was larger than the control group at 2012 by 10%. These results are similar to the ones comparing between municipalities in a cross-sectional design.

The models with the homicide rate *change* variable show similar results, although they were less clear. Baseline means and the average time effect were consistent with the previous models. Again, the group effect was non-significant when violence was high and significant when violence was low; the second difference was also negative for low violence and high poverty by 26%. The third differences for the models with high violence were statistical significant only at 90%; the model with low poverty had a coefficient of 0.050 (p=0.054), a 5.3% increase, and the model with high poverty had a coefficient of 0.108 (p=0.090), a 10.7% increase. In spite of it, the pattern of differences mirrored the one with the homicide rates. The municipalities with high violence revealed a distinct trajectory: equivalent to the control group at baseline and a larger increase at the endpoint. Municipalities with low levels of violence were not equivalent to the control groups at baseline, but in different ways depending on the poverty levels: higher poverty meant lower means and less poverty resulted in higher means. These results suggest that in Mexico affluent municipalities might be more stressful than poor ones.

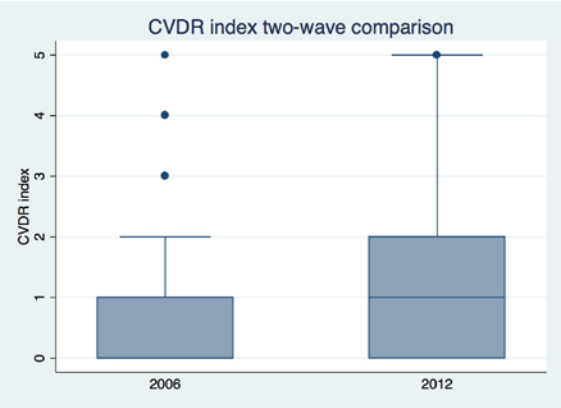
Table 12

Difference in Difference Null Models Without Covariates for Treatment Interactions

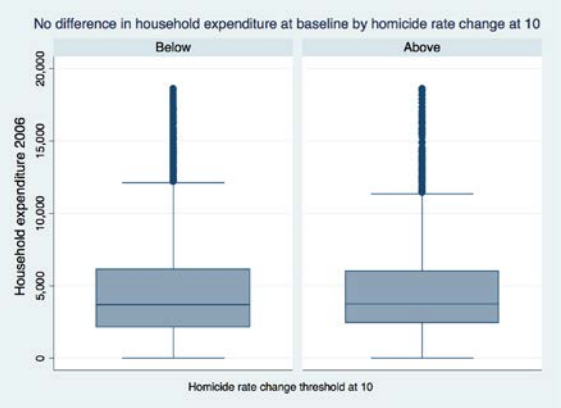
	Low violence & Low poverty	Low violence & High poverty	High violence & Low poverty	High violence & High poverty
CVDR Intercept	0.632**	0.670**	0.653**	0.656**
Time effect	0.505**	0.501**	0.489**	0.498**
Group effect	0.094**	-0.174**	0.026	-0.041
Diff in Diff	-0.032	-0.050	0.087*	0.127
Residual variance	0.512	0.511	0.511	0.511
Random Intercept variance	0.362**	0.361**	0.363**	0.363**
	Low violence *change* & Low poverty	Low violence *change* & High poverty	High violence *change* & Low poverty	High violence *change* & High poverty
CVDR Intercept	0.634**	0.677**	0.653**	0.655**
Time effect	0.503**	0.497**	0.489**	0.495**
Group effect	0.129**	-0.177**	0.011	0.015
Diff in Diff	-0.029	-0.011	0.050◊	0.108◊
Residual variance	0.512	0.511	0.512	0.512
Random Intercept	0.362**	0.360**	0.363**	0.363**

variance

Estimations were done without multiple imputation. ◊=p<.10;*=p<.05; **=p<.01. The reference category was always the municipalities with percentiles above 25 and below 75 in the municipality-level independent variables.



A



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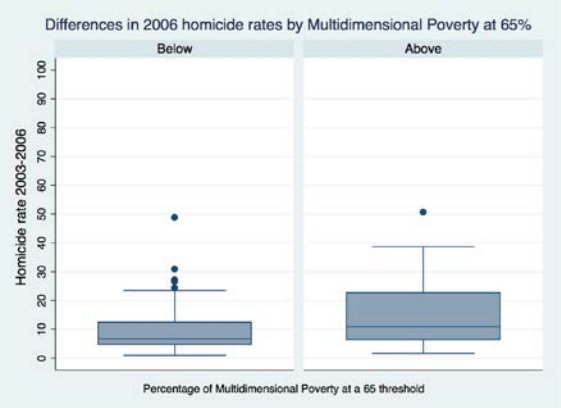

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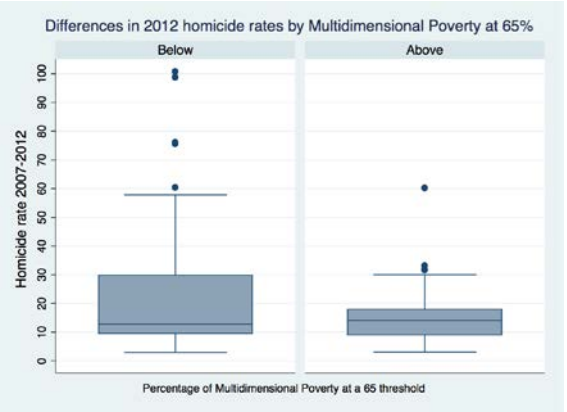

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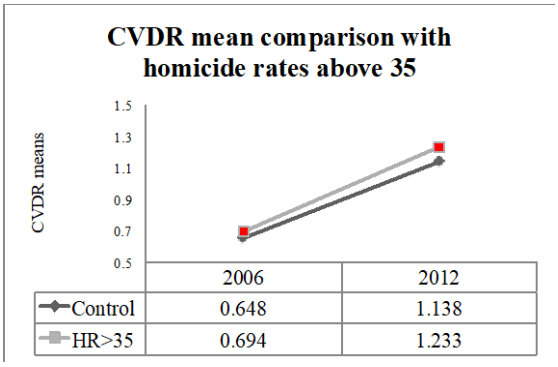

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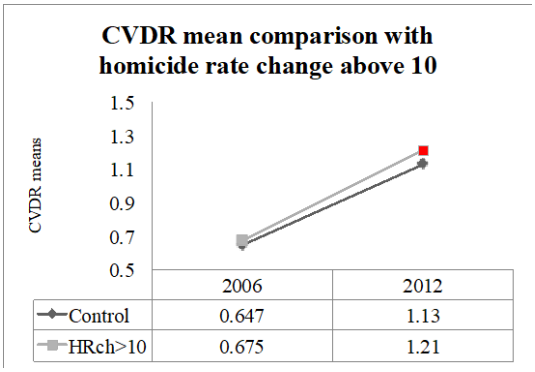

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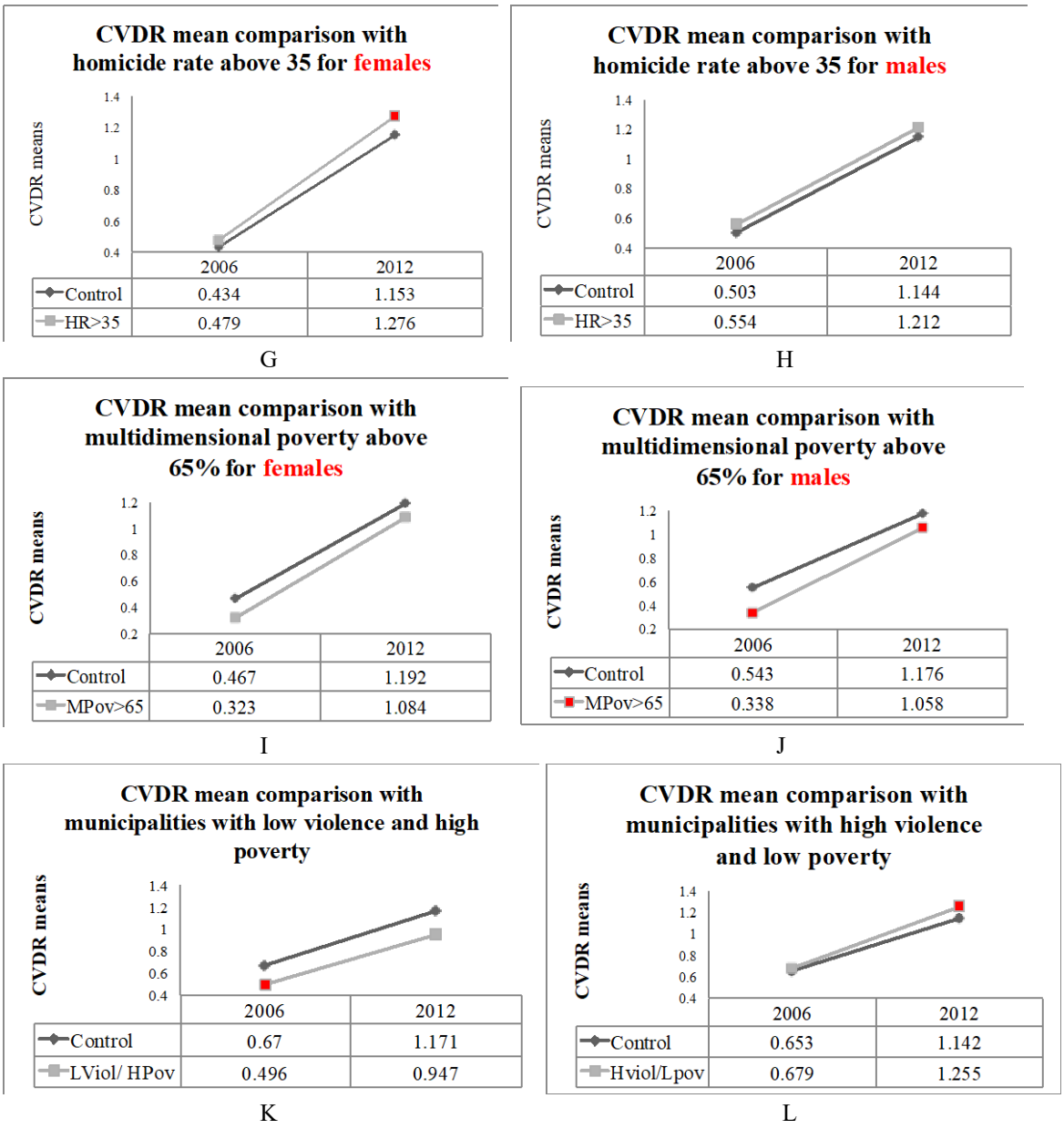


Figure 6. This set of graphs shows a few visual descriptions of within person change. In addition, it illustrates the key results of the within person analyses.

5.3 Within and between variation: Growth Curve Models

The final part of the analyses aimed to establish growth trajectories of the single biomarkers and to establish if they varied by type of municipality. Parallel three-level models were estimated for the single biomarkers: waist circumference (WC), diastolic blood pressure (DBP), and systolic blood pressure (SBP). The within-person variation was modeled in the first

level with the biomarker's trajectories by estimating an adjusted intercept at baseline (year 2002) and then the mean growth rate for the next two waves (years 2006 & 2012). The between-person variation was controlled for at the second level with individual sociodemographic, economic, and health controls as to focus the comparisons on equivalent, *average*, people. The between-municipality variation was modeled with contextual independent variables. The adjusted growth trajectories by types municipality were the main outcome of the analysis.

An exploratory analysis of individual growth trajectories exhibited considerable variability on the three biomarkers. Graph A in Figure 7 showed that the twelve first individuals in the sample, all from the first municipality, had multiple types of paths on their WC measures. In the decade under study, some gained a few waist centimeters, others lost some, and one or two remained mostly the same. Even though some had a missing value, those who had the three data-points evidenced that the shape of the path was not clearly linear; the third person kept the same waist from 2002 to 2006 but it then increased by 2012 but the ninth person decreased by 2012. Moreover, some had a fast growth rate, as person 8, but others a slower one, like person 10. Variability in direction, shape, and growth rate were also present on the individual trajectories of DBP and SBP; see Graphs B and C in Figure 7. These graphs illustrate within-person change. However, when then these trajectories were averaged, they depicted a different a pattern.

The null models showed the unadjusted linear trajectories of the biomarkers (Table 4.3). The intercept reflected the biomarker's mean at baseline, and year was the slope. The slope represented the average growth rate per wave i.e. how much it changes from one wave to the next. The mean of WC was 85.11 in 2002 and the statistically significant growth rate was 4.54 per wave, so the average WC in 2006 was 89.65, and the mean WC was 94.19 in 2012. The linear path for DBP was different. In the models, the 2002 mean was 77.79, but the growth rate of 0.15 was not significant. The path for SBP was unexpected as well. The mean was 124.31 in 2002 and

the growth rate was significant but negative (-1.42), implying that the values for 2006 were 122.89, and 121.47 for 2012. While WC matches with the descriptive statistics, the two trajectories for blood pressure were at odds with table 4.7, which clearly showed a significant increase between 2006 and 2012. The problem was the unusually high 2002 values. When using ten multiple imputations, DBP had a mean of 79.24 (CI: 75.51-75.88) in 2002, a mean of 75.70 (CI: 75.51-75.88) in 2006, and a mean of 79.27 (CI: 79.08-79.45) in 2012. Likewise, SBP had a mean of 128.08 (CI: 127.68-128.49) in 2002, a mean of 117.59 (CI: 117.33-117.85) in 2006, and a mean of 124.32 (CI: 123.98-124.65) in 2012. While descriptive statistics suggest a V-shape pattern, growth model forces a linear path, and thus offers a nonsignificant rate of change and a negative one. Most likely, measurement error during 2002 data collection due to single measurement of blood pressure was driving up the means and causing ill-defined trajectories.

The null models also provided the variance partition by level. Table 11 showed significant variability at the person level and at the municipality level for the three biomarkers. Variances at both levels were statistically significant in all models. However, the amount of variance at the municipality level was very small; 5.4% for WC, 2.9% for DBP, and 2.1% for SBP. This meant that most variability was within and between persons and only a very small part was explained by between-municipality variation.

The first model in Tables 14, 15 and 16 tested the effect of the 2012 homicide rate on the average trajectories of the biomarkers while person level covariates were controlled for. The hypothesis stated that the trajectories should be equal at baseline regardless of the level of violence in 2012 at the municipality for the three biomarkers. Since poverty rates were constant in the last decade, the baseline means are hypothesized to be significant in the poorest municipalities. Most importantly, the growth rate would have been positive for 2012 homicide

rates and the 2010 multidimensional poverty. This means that everyone had a positive growth rate but people living in poor or violent municipalities experience a faster growth.

Table 13

Null Models for the Three Biomarkers and Proportions of the Variance Partition by Level

	Waist Circumference	**Diastolic blood pressure**	**Systolic blood pressure**
Intercept	85.11	77.79	124.31
	(0.313)	(0.302)	(0.441)
Year	4.54**	0.15	-1.42**
	(0.105)	(0.177)	(0.263)
Variance			
Measure level (*r*)	48.25	94.28	229.89
Person level (*e*)	115.02**	26.08**	125.5**
Municipality level (*u*)	9.35**	3.64**	7.61**
Total variance	172.62	124	363
Person level ICC	66.6%	21%	34.5%
Municipality level ICC	5.4%	2.9%	2.1%

ICC= Intra-class correlation i.e. the percent of variance at that level.

Table 13 showed that municipalities with a 2012 homicide rate above average already had higher WC in 2002; for every 10 units in the homicide rate, the baseline mean of WC was almost half centimeter longer. Multidimensional poverty was also significant at baseline but in the opposite direction. For every ten percent points above the poverty mean, people had on average a 0.66 smaller WC. The growth rate was not significant for 2012 homicide rates but it was significant and negative for multidimensional poverty (-0.009). People living on an average municipality increased their WC by 4.8 cm every five years, but people who lived on poorer municipalities increased their WC at a slightly slower rate. Estimations for DBP in Model 1 in Table 13 showed a different pattern once the intercept was adjusted by individual controls. The mean DBP at baseline dropped down to 79.89 and the overall growth rate was now positive and significant (0.499). Municipality characteristics were non-significant at baseline but they were at

the growth rate pointing into opposite direction for violence and poverty. For every 10 points above the mean of the 2012 homicide rates, the average growth rate of DBP increased by an additional 0.1, a 20% increase. Moreover, for every 10 percent-points of multidimensional poverty above the mean, the growth rate of DBP decreased by 0.13, a 26% difference. Adjusted means of SBP did not change much from the null models; model 1 in Table 14. Although the mean SBP at baseline dropped as well to 120.3, the growth rate remained significant and negative (-1.493). Intercepts at baseline were similar except for multidimensional poverty. The growth rate was positive and significant for the 2012 homicide rate. This means that the overall linear trajectory declined by 1.493 each wave; however, the decline was slower in municipalities with a homicide rate 10 points above the mean, at a rate of 1.333, an 11% decrease.

Model 2 in each table examined if the previous associations were confounded by income inequality. The inclusion of the GINI coefficient did not change the coefficients for WC in the growth rate. Inequality had a significant and positive association with DBP at baseline and at the growth rate, indicating that more unequal municipalities had higher DBP but the growth increase was slower. The GINI coefficient reduced the strength of association with the growth rate of the 2012 homicide rate down to 0.007 (and it became significant only at $p<.10$) and increased the size of the negative association with poverty up to 0.018. Likewise, inequality had a significant and negative association with the SBP growth rate, indicating that the decrease is faster in more unequal municipalities. The growth rate for the 2012 homicide rate decreased in size as well, but remained significant.

The hypothesis for the third model was the same as the one for first model except that the homicide *change* variable replaced the 2012 homicide rate. Municipalities that experienced a spike in the homicide rate already had a wider WC in 2006 of about 0.57 cm for every 10 units of *change*. Conversely, poor municipalities had a 0.56 smaller WC for every 10 additional percent-

points. *Change* in the homicide rate did not have a significant increase in the growth rate of WC. However, poor municipalities did have a slower growth rate in WC than richer ones. DBP at baseline did not have significant differences based on type of municipality. However, for every 10 additional points in the *change* variable, their rate grew faster by 0.16 than those that did not change, a 46% increase. The growth rate of poor municipalities was slower; an additional 10 percent increase reduced the rate by 0.08, a 23% decrease. With SBP the change variable was also positively associated with the growth rate; the decline of SBP was 8.6% slower for every 10 points in the homicide *change* variable.

Model four in the three tables again tested if the associations were confounded by income inequality. The GINI coefficient was not associated with WC and the main associations remained mostly unchanged. As in model 2, inequality was positively associated with DBP at baseline and negatively in the growth rate. The inclusion of the GINI coefficient increased the size of the poverty coefficient and reduced the one of the violence variable but both remained significant. Inequality was negatively associated with the growth rate of SBP and the relationship between homicide change and SBP became nonsignificant.

Models 5 and 6 in tables 14, 15 and 16 tested the interaction hypothesis with dummy variables to ease the interpretation of the trajectories. The expectation was that violent and poor municipalities would have faster growth rates and that peaceful and affluent municipalities would have slower growth rates, both in comparison with the average municipalities. Unfortunately, these two combinations may have had low statistical power to find small associations because few municipalities had both characteristics at low or both at high. The mix between high and low poverty and violence will show if contextual violence or contextual poverty matter more for changes in the growth trajectories.

Table 14

Growth Curve Models for Waist Circumference (WC)

	Waist Circumference					
Municipality-level predictors	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	**84.65**	**84.65**	**84.11**	**84.13**	**84.51**	**84.60**
	(0.292)	(0.293)	(0.321)	(0.322)	(0.377)	(0.383)
Homicide rate 2012[a]	0.049**	0.047**				
	(0.013)	(0.014)				
Homicide rate change 06-12[a]			0.057**	0.054**		
			(0.014)	(0.015)		
Multidimensional poverty 2010[a]	-0.066**	-0.069**	-0.056**	-0.059**		
	(0.014)	(0.015)	(0.015)	(0.016)		
GINI 2010[a]		-3.694		-3.302		
		(6.247)		(6.203)		
Low (CH) HR & low MP[b]					0.459	1.006
					(0.700)	(0.862)
Low (CH) HR & high MP[b]					-0.704	-2.021*
					(0.918)	(0.774)
High (CH) HR & low MP[b]					2.11⋄	1.457⋄
					(1.068)	(0.739)
High (CH) HR & high MP[b]					1.606	0.112
					(3.125)	(1.514)
Year	**4.808****	**4.807****	**4.804****	**4.797****	**4.803****	**4.899****
	(0.125)	(0.093)	(0.095)	(0.096)	(0.099)	(0.101)
Homicide rate 2012[a]	-0.002	-0.002				
	(0.005)	(0.002)				
Homicide rate change 06-12[a]			-0.003	0.009		
			(0.003)	(0.020)		
Multidimensional poverty 2010[a]	-0.009**	-0.009**	-0.007*	-0.006*		
	(0.002)	(0.002)	(0.003)	(0.003)		
GINI 2010[a]		0.886		1.156		
		(1.291)		(1.303)		
Low (CH) HR & low MP[b]					0.168	-0.256⋄
					(0.114)	(0.135)
Low (CH) HR & high MP[b]					-0.757**	-0.624**
					(0.183)	(0.171)
High (CH) HR & low MP[b]					0.530**	0.178
					(0.164)	(0.131)
High (CH) HR & high MP[b]					-0.173	0.780*
					(0.660)	(0.306)
Variance						
Municipality level intercept (U_0)	5.92	5.91	5.77	5.75	7.75	8.55
Individual level (r)	95.1	93.1	93.1	93.2	93.2	93.5
Measure level (e)	45.6	45.6	45.6	45.6	45.6	44.7
R^2						

a= grand-mean centered; b=uncentered; CH=Homicide rate *change* 06-12; HR= Homicide rate; MP= 2010 Multidimensional Poverty; ⋄=p<.10;*=p<.05; **=p<.01. Ten datasets from multiple imputations were used. All models control at the person level by: gender, age in quintile, single, indigenous, disability, household size, household expenditure in quintiles, chronic diseases, comorbidities, smoking, drinks alcohol; and population density at municipality level. The expression "Low (CH) HR and low MP" means that model 5 refers to "Low 2012 homicide rates and low 2010 Multidimensional Poverty", and model 6 refers to "Low homicide rate *change* and low 2010 Multidimensional Poverty"; the same rationale applies for the next three rows.

Table 15

Growth Curve Models for Diastolic Blood Pressure (DBP)

	Diastolic Blood Pressure					
Municipality-level predictors	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	**75.89**	**75.87**	**76.04**	**75.97**	**75.94**	**76.14**
	(0.241)	(0.241)	(0.265)	(0.266)	(0.283)	(0.295)
Homicide rate 2012 [a]	-0.016	-0.012				
	(0.011)	(0.011)				
Homicide rate change 06-12 [a]			-0.016	-0.010		
			(0.012)	(0.012)		
Multidimensional poverty 2010 [a]	0.001	0.008	0.003	0.012		
	(0.011)	(0.012)	(0.012)	(0.013)		
GINI 2010 [a]		13.15*		13.143*		
		(5.13)		(5.119)		
Low (CH) HR & low MP [b]					0.213	0.175
					(0.506)	(0.639)
Low (CH) HR & high MP [b]					0.307	-0.486
					(0.680)	(0.601)
High (CH) HR & low MP [b]					-1.833*	-1.456*
					(0.766)	(0.555)
High (CH) HR & high MP [b]					2.273	0.807
					(2.373)	(1.151)
Year	**0.499***	**0.512****	**0.348***	**0.402***	**0.410***	**0.536****
	(0.119)	(0.119)	(0.126)	(0.127)	(0.130)	(0.132)
Homicide rate 2012 [a]	0.010*	0.007⋄				
	(0.003)	(0.003)				
Homicide rate change 06-12 [a]			0.016*	0.012*		
			(0.004)	(0.004)		
Multidimensional poverty 2010 [a]	-0.013**	-0.018**	-0.008*	-0.014**		
	(0.003)	(0.004)	(0.004)	(0.004)		
GINI 2010 [a]		-9.648**		-9.212*		
		(1.794)		(1.808)		
Low (CH) HR & low MP [b]					0.312⋄	-0.092
					(0.161)	(0.188)
Low (CH) HR & high MP [b]					-0.292	-0.554*
					(0.256)	(0.237)
High (CH) HR & low MP [b]					0.924**	0.603**
					(0.225)	(0.181)
High (CH) HR & high MP [b]					-1.556	-1.024*
					(0.961)	(0.426)
Variance						
Municipality level intercept (U_0)	3.65	3.66	3.59	3.59	3.59	3.53
Individual level (r)	20.59	20.64	20.60	20.64	20.60	20.61
Measure level (e)	93.57	93.44	93.54	93.43	93.55	93.54

a= grand-mean centered; b=uncentered; CH=Homicide rate *change* 06-12; HR= Homicide rate; MP= 2010 Multidimensional Poverty; ⋄=p<.10;*=p<.05; **=p<.01. Ten datasets from multiple imputations were used. All models control at the person level by: gender, age in quintile, single, indigenous, disability, household size, household expenditure in quintiles, chronic diseases, comorbidities, smoking, drinks alcohol; and population density at municipality level. The expression "Low (CH) HR and low MP" means that model 5 refers to "Low 2012 homicide rates and low 2010 Multidimensional Poverty", and model 6 refers to "Low homicide rate *change* and low 2010 Multidimensional Poverty"; the same rationale applies for the next three rows.

Table 16

Growth Curve Models for Systolic Blood Pressure (SBP)

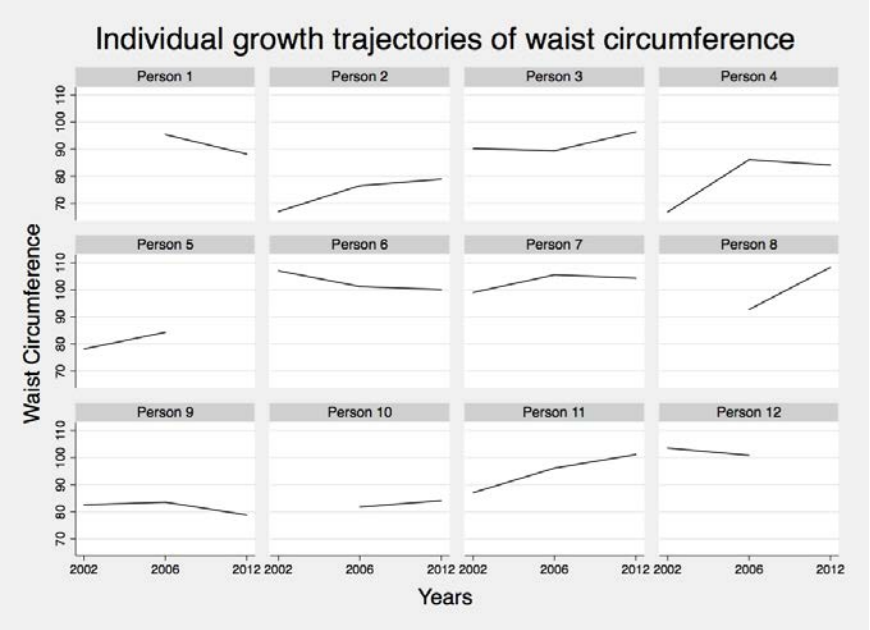
	Systolic Blood Pressure					
Municipality-level predictors	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
Intercept	**120.03**	**120.02**	**119.92**	**119.91**	**120.17**	**119.90**
	(0.381)	(0.379)	(0.410)	(0.409)	(0.444)	(0.456)
Homicide rate 2012[a]	-0.002	-0.003				
	(0.016)	(0.016)				
Homicide rate change 06-12[a]			0.011	0.011		
			(0.018)	(0.018)		
Multidimensional poverty 2010[a]	-0.033⋄	-0.037*	-0.018	-0.021		
	(0.026)	(0.018)	(0.019)	(0.019)		
GINI 2010[a]		-2.896		-1.769		
		(7.627)		(7.588)		
Low (CH) HR & low MP[b]					0.335	1.701⋄
					(0.764)	(0.960)
Low (CH) HR & high MP[b]					-0.358	-1.058
					(1.030)	(0.901)
High (CH) HR & low MP[b]					-1.585	-0.182
					(1.178)	(0.859)
High (CH) HR & high MP[b]					3.379	4.043*
					(3.503)	(1.736)
Year	**-1.493****	**-1.484****	**-1.62****	**-1.582****	**-1.636****	**-1.205**
	(0.191)	(0.191)	(0.197)	(0.19)	(0.212)	(0.211)
Homicide rate 2012[a]	0.016*	0.013*				
	(0.013)	(0.006)				
Homicide rate change 06-12[a]			0.014*	0.010		
			(0.006)	(0.007)		
Multidimensional poverty 2010[a]	0.006	0.002	-0.005	0.003		
	(0.006)	(0.006)	(0.006)	(0.006)		
GINI 2010[a]		-7.733*		-8.024*		
		(2.839)		(2.857)		
Low (CH) HR & low MP[b]					0.227	-1.234**
					(0.258)	(0.295)
Low (CH) HR & high MP[b]					-0.061	-0.366
					(0.408)	(0.362)
High (CH) HR & low MP[b]					0.524	0.146
					(0.364)	(0.301)
High (CH) HR & high MP[b]					-0.272	-2.287
					(1.426)	(0.668)
Variance						
Municipality level intercept (U_{00})	7.7	7.5	7.5	7.31	7.71	7.60
Individual level (*r*)	67.7	67.7	67.7	67.7	67.64	67.75
Measure level (e)	228.6	228.5	228.6	228.5	228.73	228.44

a= grand-mean centered; b=uncentered; CH=Homicide rate *change* 06-12; HR= Homicide rate; MP= 2010 Multidimensional Poverty; ⋄=p<.10;*=p<.05; **=p<.01. Ten datasets from multiple imputations were used. All models control at the person level by: gender, age in quintile, single, indigenous, disability, household size, household expenditure in quintiles, chronic diseases, comorbidities, smoking, drinks alcohol; and population density at municipality level. The expression "Low (CH) HR and low MP" means that model 5 refers to "Low 2012 homicide rates and low 2010 Multidimensional Poverty", and model 6 refers to "Low homicide rate *change* and low 2010 Multidimensional Poverty"; the same rationale applies for the next three rows.

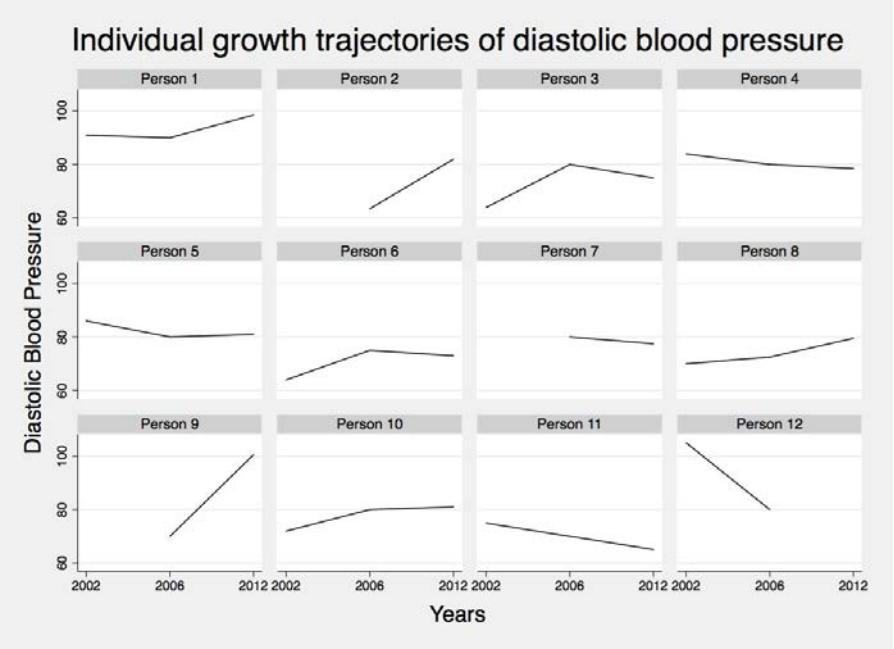
Model 5 estimated the interactions between 2012 homicide rates and 2010 multidimensional poverty according to the previously established thresholds. At baseline, municipalities were mostly similar in WC. The exception was violent and affluent municipalities, which started with 2.11 more cm in WC than the average municipality ($p<0.1$) and then had a faster growth rate by 0.53; an 11% increase per wave. Conversely, municipalities with low violence started slightly below the average municipalities but then had a slower growth rate in WC by 0.757; a 15% decrease. Both trajectories are shown more clearly in graph D from figure 7. While all the municipalities started in a very similar place, the trajectory of the average municipality ran in the middle, in dark blue; peaceful and poor municipalities moved below, at a slower pace; and violent and affluent municipalities advanced above, at a faster pace. Graph F, with the DBP trajectories, evidenced that the paths did not always run in parallel. On the one hand, peaceful and affluent municipalities started with the average and their growth rate was faster while poor and peaceful municipalities had a similar trajectory to the average municipalities all along. However, on the other, affluent and violent municipalities started with lower means at baseline (-1.83 cm) but they had the fastest growth rate (225% higher than the average growth rate) so they reached the average by 2012. Violent and poor municipalities did not have statistically significant differences (probably due to small sample size at the municipality), but they had a negative trajectory. The paths for SBP were equivalent at baseline and in their growth rates; see graph H.

Model 6 tested more interactions but substituted 2012 homicide rates for homicide rate *change*. Poor municipalities that did not experience sharp *changes* in their homicide rates started with lower means in WC and their growth rate was 12.7% slower than in the average municipalities. Affluent municipalities that did not *change* their homicide rates by 2012 had similar WC at baseline but the growth rate was also 5.2% lower ($p<0.1$). Interestingly, affluent

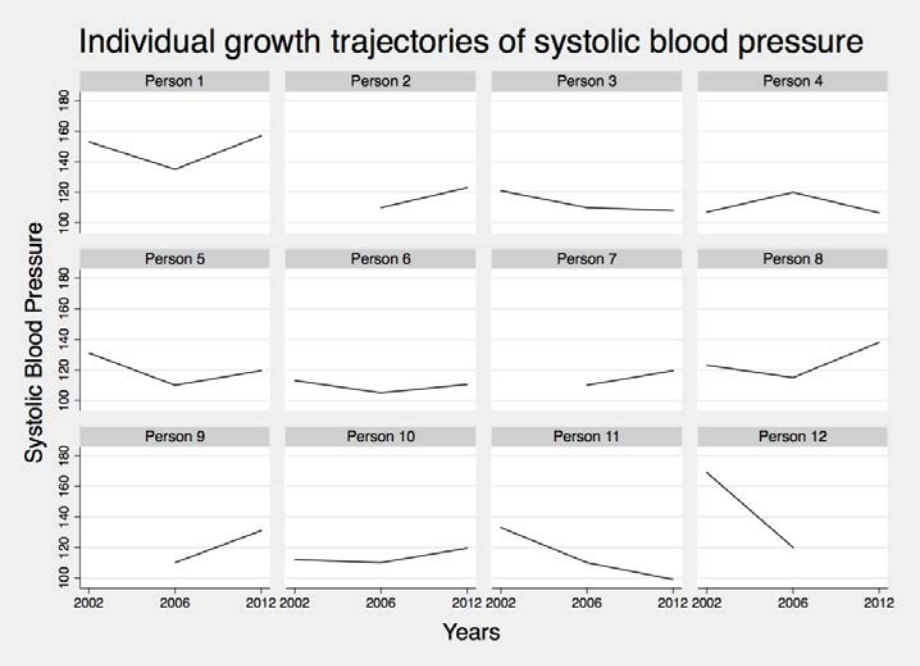
municipalities that experienced high *change* in their homicide rates had higher WC means at baseline (1.46 p< .01) but a similar growth rate as the average. It was poor and violent municipalities that were equivalent at baseline and then had a 16% higher growth rate per wave (0.780) when compared to the average municipalities; see graph E in figure 7. The paths of DBP were also different with the *change* variable; see graph G. Affluent municipalities with low *change* were very similar than the average municipality, both with a positive growth rate. Poor municipalities with low change were similar at baseline but their growth rate was negative (-0.018), not positive, so their paths clearly diverged by 2012. Affluent municipalities with high *change* had lower baseline values (-1.456) but their growth rate was 112% faster (0.603) than the average municipality so at the end they were almost with the same mean. Unexpectedly, poor municipalities with high *change* did not have differences at baseline but the growth rate was significant and negative. Finally, the SBP trajectories show that poor municipalities with high *change* had 4 extra cms at baseline but the growth rate was similar to the average municipality. Affluent municipalities with low change started with higher baseline means and their means declined at a faster rate than the average municipality by 102%.


Individual growth trajectories of waist circumference
Person 1
Person 2
Person 3
Person 4
Person 5
Person 6
Person 7
Person 8
Person 9
Person 10
Person 11
Person 12
Waist Circumference
70
80
90
100
110
2002
2006
2012
Years

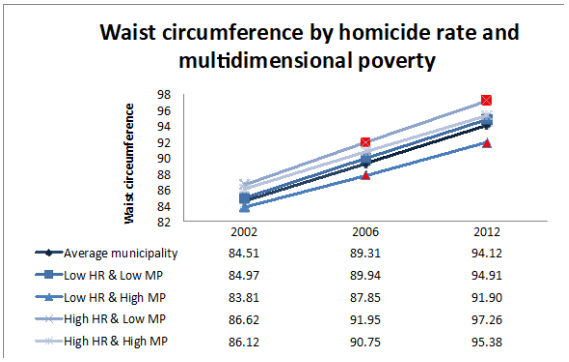
A


Individual growth trajectories of diastolic blood pressure
Person 1
Person 2
Person 3
Person 4
Person 5
Person 6
Person 7
Person 8
Person 9
Person 10
Person 11
Person 12
Diastolic Blood Pressure
60
80
100
2002
2006
2012
Years

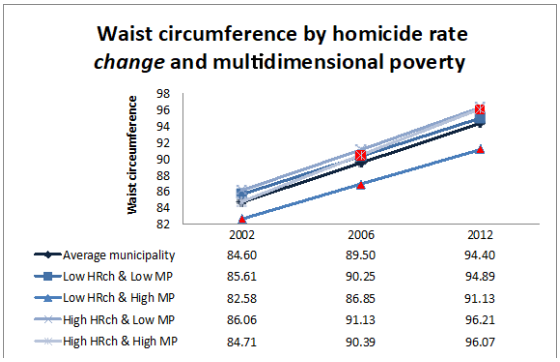
B


Individual growth trajectories of systolic blood pressure
Person 1
Person 2
Person 3
Person 4
Person 5
Person 6
Person 7
Person 8
Person 9
Person 10
Person 11
Person 12
Systolic Blood Pressure
100
120
140
160
180
2002
2006
2012
Years

C


Waist circumference by homicide rate and multidimensional poverty
Waist circumference
98
96
94
92
90
88
86
84
82
2002
2006
2012
Average municipality 84.51 89.31 94.12
Low HR & Low MP 84.97 89.94 94.91
Low HR & High MP 83.81 87.85 91.90
High HR & Low MP 86.62 91.95 97.26
High HR & High MP 86.12 90.75 95.38

D


Waist circumference by homicide rate change and multidimensional poverty
Waist circumference
98
96
94
92
90
88
86
84
82
2002
2006
2012
Average municipality 84.60 89.50 94.40
Low HRch & Low MP 85.61 90.25 94.89
Low HRch & High MP 82.58 86.85 91.13
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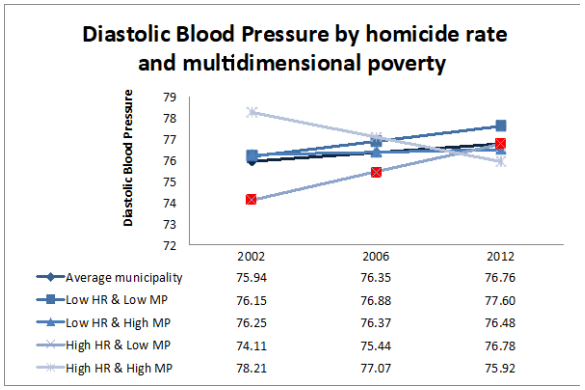
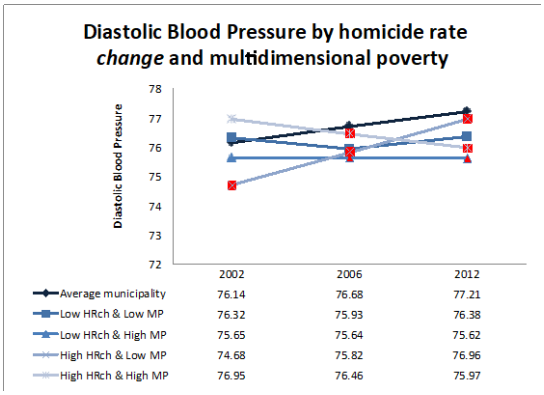
High HRch & High MP 84.71 90.39 96.07

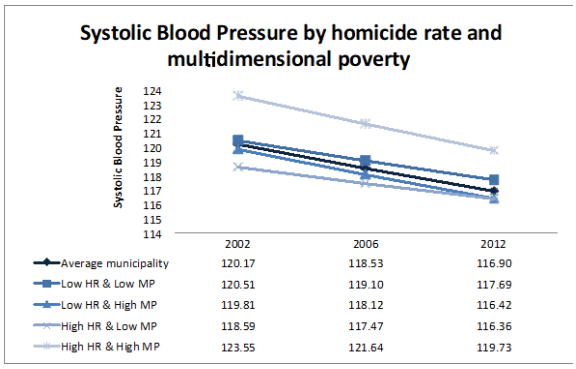
E


Diastolic Blood Pressure by homicide rate and multidimensional poverty
Diastolic Blood Pressure
79
78
77
76
75
74
73
72
2002
2006
2012
Average municipality 75.94 76.35 76.76
Low HR & Low MP 76.15 76.88 77.60
Low HR & High MP 76.25 76.37 76.48
High HR & Low MP 74.11 75.44 76.78
High HR & High MP 78.21 77.07 75.92

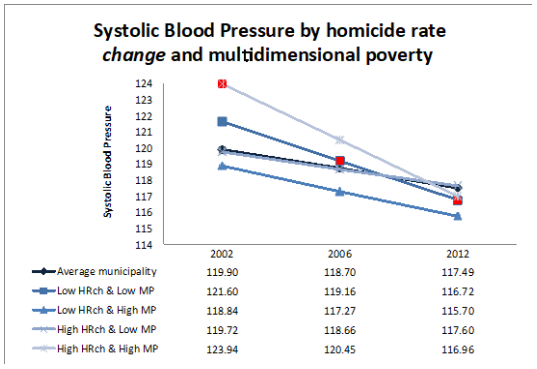
F


Diastolic Blood Pressure by homicide rate change and multidimensional poverty
Diastolic Blood Pressure
78
77
76
75
74
73
72
2002
2006
2012
Average municipality 76.14 76.68 77.21
Low HRch & Low MP 76.32 75.93 76.38
Low HRch & High MP 75.65 75.64 75.62
High HRch & Low MP 74.68 75.82 76.96
High HRch & High MP 76.95 76.46 75.97

G



H



I

Figure 7. These graphs graph depict the results from the within and the between person and municipality analyses. They are presented as trajectories based on the growth curve models.

Chapter VI. Discussion

The dissertation sought to examine the health consequences of the spike in the homicide rates in Mexico between 2006 and 2012. The main hypothesis was that, in urban environments, exposure to higher levels of contextual violence increased the levels of cardiovascular risk when individual characteristics are controlled for. The objective was to test this effect in isolation, independently from contextual poverty, and as an interaction, where violent and poor municipalities were theorized as the most stressful environments. Moreover, these effects were expected to have cross-level interactions with individual expenditure, fear of crime and collective efficacy and to have heterogeneous effects with subpopulations identified by gender, age, victimization, and socioeconomic status. The analytic strategy aimed to triangulate three statistical approaches according to the type of comparison –between persons, within persons, and both– with three different measures of cardiovascular risk as means to strengthen the findings. The results yielded nuanced responses to the main hypothesis.

6.1 Significance of Findings and Implications for Practice and Policy

A novelty of the investigation is that violence was not correlated with poverty in Mexico during the period under study. Descriptive analyses revealed key differences between Mexico and high-income countries –from where most of the empirical evidence comes from (R J Sampson, 2012). First, 2006 homicide rates were weakly associated with the 2012 rates – see figures 1.1 and 2.1– and *changes* in the homicide rates between periods were not correlated with the 2006 rates. This means that the social dynamics that were driving the homicide rate down in previous years, dramatically changed after 2006 (Escalante, 2012). In contrast, the poverty measures were notably stable during the study; there were no mean differences between the years 2000, 2005, and 2010 and their correlation was very strong (0.902); see graph J in figures 3.1. Thus, rich

municipalities kept rich and poor municipalities remained poor, regardless of the homicide rate increase. Second, contrary to substantial research (Land et al., 1990; McCall et al., 2010; Nivette, 2011), the 2012 homicide rate was not correlated with the 2010 poverty measures. This fact opened the possibility to isolate both effects and consider violence as an external shock, independent from poverty, just as other researchers have done (Brown, Montalva, Thomas, & Velasquez, 2017). Third, violence and poverty were only weakly associated in 2006 but it was affluent municipalities that mostly suffered the violence increase after 2006. Homicide rates were lower in richer municipalities in 2002 and 2006, but by 2012 the spike was greater in these richer municipalities and the difference reversed; see graph P in figure 4.1. These findings suggest that poverty and violence are not inextricably linked and they allowed treating the violence increase as an external shock and longitudinal analyses as a natural experiment.

Besides poverty, another secular trend threatened to confound the results. During the same period under study, Mexicans presented a notably high prevalence in obesity (Barquera et al., 2012), which is closely associated with cardiometabolic risk (Grundy et al., 2004). This prevalence was higher in urban municipalities but lower in the first socioeconomic tertile (Barquera et al., 2012), thus contradicting the social gradient of health (Marmot, 2015). The implication for the study was that sizable changes in the dependent variable between 2006 and 2012 were expected in every municipality, but they should be higher in the most violent municipalities. Rapid urbanization was also believed to be a worrisome secular trend, but the empirical effects were not salient.

An important assumption to test the hypothesis was that cardiovascular measures are sensitive to stress, as much theory suggests (Steptoe & Kivimaki, 2013). The validation process identified that the dependent variables are associated with health indicators: as health worsened, the dependent variables increased. In addition, the dependent variables were negatively

associated with household expenditure, reflecting a health gradient in which lower socioeconomic quintiles had higher cardiovascular risk than the upper quintiles; see graph A3.1. Waist circumference was the exception and the gradient went in the opposite direction. Violence variables at the individual level, however, were negatively associated with the dependent variables. The unexpected finding reflects that people who report and perceive more violent events belong to the upper socioeconomic quintiles and they are also the ones with better health. The technique used in the validation process was unable to separate the confounding effect between violence and SES at the individual level.

The validation process extended the scope of the analysis to test the isolated effects of context, as has been suggested for environmental covariates (Leal & Chaix, 2011), but results were mixed. Unadjusted context characteristics were not associated with the Framingham score but the CVDR index and blood pressure were associated to *change* in the homicide rates and waist circumference covaried with both violent measures. Contrary to the theoretical claims (Evans & Kim, 2010), multidimensional poverty was negatively associated with CVDR and the single biomarkers. These results imply that poor contexts are less stressful than richer contexts when individual SES was not controlled.

In spite of the mild response to context and the unanticipated findings, the dependent variables performed as planned with individual covariates and were thus considered to adequately reflect stress. The validation process was valuable because it shows a promising way to objectively measure gradients of stress. Moreover, the results confirm that risk indices do not run in parallel with single biomarkers, and indices that tap into multiple systems capture stress more accurately (Juster et al., 2010; Seeman et al., 2010). Previous research has demonstrated that subjective assessments of stressful experiences, like racial discrimination, may (Sawyer, Major, Casad, Townsend, & Mendes, 2012) or may not run in parallel with objective cardiovascular

measures (Gee & Payne-Sturges, 2004). The use of biomarkers to examine the effects of psychosocial influences opens innovative possibilities to identify how subjective appraisals in specific contexts may get under the skin to produce worse health outcomes (Kubzansky et al., 2014). Therefore, it is important to expand the type of data that combines social and health indicators with biomarkers from multiple systems.

Once the measures were validated and descriptive statistics gave an appropriate context, the models aimed to prove the main hypothesis i.e. exposure to higher levels of contextual violence works as a stressor by increasing the levels of cardiovascular risk. As theoretically expected (Ahern et al., 2008), the three statistical approaches provided evidence indicating that people living in more violent municipalities had higher cardiovascular risk. Violence was tested as a cumulative stressor with the homicide rates and as an intermittent stressor when the homicide rate *change* variable was included. Cross-sectional models comparing between-persons indicate that two females, none of them victimized and with average expenditures, but one living in a municipality with 10 additional points from the mean in homicide rates, has a 1.5% higher Framingham score. A third equivalent female living in a municipality departing 20 points from the average homicide rates has a 3% higher Framingham score. Likewise, for every 10 additional points in *change* in the homicide rates, the Framingham scores grows by 7.3%. Two-wave longitudinal models testing within-person changes identify that the significant threshold in which violence exerts statistically significant stress was 35 for the homicide rates and 10 for the homicide rate *change* variable. These models coincide with the previous results by showing how most of the sample alarmingly increased by 74% their CVDR means between 2006 and 2012, but the increase was 8.3% higher for people living in a municipality with homicide rates above the 35-threshold. Similarly, people had equivalent CVDR means in 2006 and for 2012 the average municipality notably increased the index, but the mean was 7% higher above the 10-point

threshold in the *change* variable. The models that tested within and between change found that high homicide rates and *change* in violence did not yield statistically different growth trajectories for waist circumference. Both violence variables were significant, however, for diastolic blood pressure and had mixed results for systolic blood pressure –although blood pressure had important measurement problems in 2002. Nonetheless, the first two methods confirm that homicide rates and homicide rate *change* had significant effects on raising the Framingham score and the CVDR index. Moreover, violence as an intermittent stressor had a stronger effect between persons and the threshold was lower in the within person models. Therefore, longer exposure to violent contexts wears down the body but the effect is stronger when the stress is a more recent disturbance for homeostasis.

A concern against these results could be the size of the effect. A common problem with context effects research is the small proportion of variance at the neighborhood level that apparently makes these effects look trivial (Galster, 2012; Jencks & Mayer, 1990). The present study finds that the amount of variance at the municipality level is 4.4% for the Framingham score and between 3% and 5% for the single biomarkers. Also problematic is that none of the municipality characteristics explained all of the available variance at this level. However, these results should not be interpreted as sharply separated from individual characteristics because neighborhoods make people and people neighborhoods (Oakes et al., 2015). It is better to follow Rose's perspective in separating individual and population health (Rose, 2001). A 7% or 8% difference in the CVDR index may be imperceptible between two individuals, but it is a sizable difference on a population's mean distribution, as the ones between municipalities. It is worth remembering that these differences are averages and, consequently, they are actually larger in specific municipalities at the end of the distribution, which concentrate much of the violence, as in Ciudad Juarez, a northern Mexican state (Osorio, 2012).

The significance of the findings also needs to be gauged by the type of violence they assess. While the direct effect of violence is mostly measured with victimization (Krug, 2002; UNODOC, 2014), its indirect effects on health are harder to apprehend (Lorenc et al., 2012). The official discourse in Mexico is that gangs killing themselves may explain homicide surges and so regular citizens are actually safe. This discourse is complemented by the claim of a normalization of violence (Schedler, 2015) in which there is a perception among Mexicans that violence "only touches those involved in the world of crime" (Ahmed, 2017). Results suggest that both opinions constitute a mismatch between subjective and objective measures of stress. Contrary to these trends, the findings show with an objective measure that contextual violence has an indirect effect on cardiometabolic risk factors and, therefore, the homicide levels do indeed *touch* everyone and not only criminals. Violence is thus not a focalized event in a few hotspots, but it tailors a specific context akin to community violence (Guterman et al., 2000) with considerable psychological effects (Fowler et al., 2009) that cannot be disparaged as mere subjective perceptions of fear of crime (Vilalta, 2013). The violence epidemic in Mexico should not be understood as *them*, the criminals, who bear the burden of the conflict, and *us*, the citizens, for whom the violence is an inevitable nuisance. Assessing the indirect effects with objective and subjective measures demonstrates that the epidemic is harmful for everyone and in multiple ways. It is from this perspective that the results contribute to the literature claiming that safety is a crucial element for well-being (Stiglitz, Sen, Fitoussi, & Progress, 2009).

The relevance of these results also resides in the type of effects they have. The most evident implication is to recognize violence as an important public health issue that goes beyond security institutions requiring an interdisciplinary perspective to assuage its effects (Gamlin, 2015). Direct health impacts are notorious in the reduction of life expectancy in Mexico (Canudas-Romo et al., 2016), especially among young men (Leenen & Cervantes-Trejo, 2014).

But adding cardiovascular risk to the indirect impact of vicarious victimization and fear of crime ends up as a sizeable burden (Boelen et al., 2015). These additional strains to the population require a strengthening of public health and social services in these types of contexts and practitioners need to be aware that the consequences of living in violent places go beyond trauma and the effects of a single cause may be diverse (Hertzman & Boyce, 2010), as increased cardiometabolic risk.

Although poverty was not correlated with violence, it had an important role defining the structure of stressful municipalities. Cross-sectional analyses show how poverty is not associated with the Framingham score when the homicide rates were included but the Framingham score is higher in poorer municipalities when the *change* variable was in the model. Nevertheless, the longitudinal within-person change analyses reveal a different relationship. The significant threshold for multidimensional poverty is 65 but the direction goes in the opposite direction. Although the hypothesis was that higher poverty percentages were associated with higher stress, people above the 65-threshold actually have 22.6% lower CVDR at baseline. Likewise, in 2006, for every ten percent points above the mean multidimensional poverty, waist circumference is 0.66 centimeters smaller. People in an average municipality increase their waist circumference by 4.8 centimeters every five years but those who live on poorer municipalities grow at a slower rate. These results suggest that poor municipalities are not as stressful as violent municipalities on cardiometabolic measures.

The overwhelming evidence in the literature supporting the association between poverty and health (Brunner, 2016) calls for a more in-depth analysis, especially because at the individual level household expenditure follows the expected health gradient, except for waist circumference. The takeaway from this finding is that poverty and violence are not unavoidably linked. It is a characteristic of high-income countries and lessons or solutions from these countries need to be

taken with caution. As Amartya Sen (2008) warned, there are many peaceful and poor places, such as Kolkata, and therefore the underlying causes of violence and poverty are not always interrelated.

Interactions at the municipality level served as a confirmation of the singular characteristics of the Mexican social dynamics behind the violence increase. Tellingly, multiplicative interactions between poverty and violence were not significant, indicating that violent and poor municipalities did not combine to generate a more deleterious context than those with the isolated effect. Rather, the most stressful environments were in the affluent and violent municipalities. Cross-sectional models display, on the one hand, that, as expected, peaceful municipalities have lower means in the Framingham scores than the average: 11% lower in affluent municipalities and 6% in deprived municipalities. On the other, however, whilst violent and poor municipalities show non-significant differences, the violent and affluent municipalities measured with the homicide rates have scores 7.5% above the average ($p<0.1$). This means that when comparing between municipalities with high and low violence, the difference in the Framingham score is 17% in the former than in the latter. The only significant combination with intermittent violence was between the average municipality and richer and violent ones, having the second 8% higher Framingham scores. The within-person change models found similar results. The only significant CVDR difference at end line indicates that rich and violent municipalities have a 10% higher mean when compared with the average. These same municipalities have a 5.3% difference ($p<0.1$) when the *change* variable was introduced. While the third difference was not significant in municipalities with low *change*, poor municipalities with high *change* have scores 10.7% higher ($p<0.1$). Growth models for waist circumference are alike at baseline municipalities. The exception, again, are the affluent and violent municipalities, which start with 2.11 additional centimeters at baseline ($p<0.1$), but then have a faster growth by

11% per wave. Conversely, poor and peaceful municipalities start with the average at baseline but the growth rate is 15% slower. The *change* variable shows the same pattern for poor and peaceful municipalities with a slower growth rate by 12.7% when compared with the average. The difference is that poor and violent municipalities have the fastest growth rate by 16% in waist circumference; the growth rate of affluent municipalities that experienced a spike in the homicide rates was not significant.

These results demand for future research a more nuanced understanding on how affluent municipalities are more stressful and how poor municipalities buffer the stress caused by the absence of institutional resources; especially when urbanization and economic inequality are controlled for. One of the most important policy implications is the realization that disadvantages tend to cluster but these clusters have different characteristics between them. Concentration of stressors and lack of social and institutional resources to deal with them (Massey & Brodmann, 2014) are distinctive contextual features that provide key clues to target municipalities in greater need. Nonetheless, it is is still necessary to identify specific within municipality mechanisms that mediate stressful environments with individual outcomes (Sampson, 2012). Such nuanced dynamics within neighborhoods may be best captured with qualitative research (Small, 2015).

An important obstacle for the study was the small variance in the association of person-level predictors and cardiovascular risk measures across municipalities. Small variance reflects that these associations were very similar in every municipality –did not randomly vary – and thus the mechanisms linking municipal characteristics with the dependent variables could not be identified. For example, victimization has a negative association with the Framingham score and fear of crime is non-significant with the score regardless of the level of violence in the municipality. Likewise, household expenditure has a negative association with the Framingham score in spite of the level of multidimensional poverty in the municipality.

Nonetheless, the within-person change models are able to detect valuable differences among subpopulations. It was expected to find that older people, over 40 years old, have higher CVDR means (Eichler et al., 2007). However, the important finding is that context has a clear effect on the younger population as well. Therefore, the cumulative effect of contexts starts early in life and young age was not a protection against CVDR (McEwen & McEwen, 2017). This type of findings highlights the need to incorporate a life-course approach to policy design. Even though age cohorts cope with equal contexts differently, these exposures are cumulative and have differential impacts on subpopulations.

For instance, in line with other research (Kavanagh et al., 2010; Sundquist et al., 2006; Zimmerman & Messner, 2010), females have a greater susceptibility to context characteristics (Berkman & Kawachi, 2014). Females living in a municipality with a homicide rate above 35 have a 10.6% higher CVDR mean than females living elsewhere and 8.8% higher when the *change* variable is tested; but the effect is not significant for males. The heterogeneity of effects is also identified by individual socioeconomic status. The two lowest quintiles have the highest CVDR means and the *change* variable yields a traditional gradient. In municipalities with poverty levels above 65%, the CVDR mean is 38% lower in females and 22.5% lower in males. However, the third difference is only significant for men, indicating that poverty contexts are 10% less stressful for men when compared with men living in more affluent municipalities. Surprisingly, intermittent socioeconomic shocks do not show significant differences, probably because of the temporal distance between the shock and the biomarkers' data collection.

Heterogeneity of effects provide valuable insights for research that aims to hone in specific subpopulations to assess sensitive periods in which the effects of deleterious contexts may be greater (Gee et al., 2012). In addition, they guide the type of combinations that may lead

to syndemics and thus offer evidence on the reasons why some treatments may be more or less effective (Singer et al., 2017).

6.2 Implications for Future Research

The literature review offers a broad panorama of the context effects literature in Sociology and Public Health. Most of the theoretical and methodological problems are exposed but, rather than solved, the objective of the dissertation is to show the promising value and possible pitfalls of an ecological approach. In particular, the study is an example on how the Chinese boxes paradigm (Earls & Carlson, 2001) –as opposed to the web of causation (Krieger, 1994)– could work to better understand the cascade of events going from social contexts, to individual sensitivities, and then to biological outcomes that lead to disease. Thus, an initial way to move research forward is by continue investigating on the type of causality that entails a focus on the multilevel influences on the clustering of social problems and disease distributions (Hertzman & Boyce, 2010).

A key avenue for future research should be a continuing effort to disentangle concentrated disadvantages as means to gain more granularity in the understanding of deleterious contexts. Wilson's index was very useful to understand a specific type of community in the USA (Wilson, 2012), but it hardly translates to different institutional architectures as the ones in other countries (Sampson, Winship, & Knight, 2013) and may portray a monolithic view of stressful contexts (Small, 2015). Natural experiments in which some disadvantage departs from the usual clustering can help understand its specific mechanisms and would favor better characterizations of stressful contexts. Future research should attempt to identify meaningful neighborhoods for the residents and refrain from taking the easy path of using of political demarcations. Moreover, greater refinement on the disadvantages will probably arise from measurement improvements of

contextual traits (Raudenbush & Sampson, 1999b). It is desirable to have a research program on neighborhoods' multiple inequalities in which the adequate ecological level is identified by theory and then the proper measurement of a contextual trait is designed and estimated in a way that avoids the ecological and the individualistic fallacies (Sampson, 2017). Hopefully high-quality data may help to overcome the statistical challenge of spatial polygamy and increase the use of cross-classified models.

These specific contexts will constitute just one piece of the puzzle because the life course perspective indicates that there is heterogeneity of effects and critical periods in which these contexts impact. Therefore, contexts need to be paired with a detailed description of the population under study. Broad studies like the present one can hardly examine age cohorts and other sociodemographic interactions and end up with encompassing averages. Further research should follow intersectional theories (Watkins-Hayes, 2014) and syndemics (Singer et al., 2017) to identify the most pernicious clustering of social and health problems.

Another fundamental avenue of research is to advance on the comprehension of the mechanisms linking specific contexts with certain individual outcomes. The present study failed to provide a more in-depth explanation on how violent contexts get under the skin. It is not enough to demonstrate that exposure to violence wears down the body because it is equivalent to proof that neighborhoods matter, when the critical question is how do they operate (Sharkey & Faber, 2014). Exposure to violence by itself is not an explanatory mechanism (Galster, 2012) because it does not offer alternatives on how to reduce its effects. It is not always feasible to remove an individual from the exposure to these damaging contexts (Oakes et al., 2015) so it is necessary to improve the understanding of its buffers and catalysts in order design appropriate policies at the context level. For this topic, the most promising mechanisms are the ones based on the access to institutional resources (Elliott et al., 1996; Massey & Brodmann, 2014) and social

interactions associated with collective efficacy (Sampson, 2012) because they may explain within neighborhood variability and offer more possibilities for policy interventions (Beck, Ohmer, & Warner, 2012; Browning, Dietz, & Feinberg, 2004; Cohen et al., 2006). At the same time, judging the maturity of the contexts effects literature for its inability to calibrate the dose, timing, and frequency of neighborhood to heal certain issue (Galster, 2012) is probably missing the point. A pharmacological metaphor still follows the paradigm of infectious agents and toxins when a more complex view of social causation –nonlinear, nonspecific, dynamic, recursive, mundane and symbolic– is required (Hertzman & Boyce, 2010). This is certainly an academic challenge, but one worthwhile.

The claim to a better definition of types of contexts and mechanisms corresponds to a claim to a nuanced understanding of stress and its consequences. Innovative research on allostatic load needs to be paired with more social environments and expanded to more countries (McEwen & McEwen, 2017). Expensive and technically challenging data collection of biomarkers hampers these efforts in a large scale (Loucks, Juster, & Pruessner, 2008). Nevertheless, it is important to start conducting local research on acute problems and vulnerable populations in an effort to improve the assessment of the indirect and cumulative effects of contexts.

6.3 Limitations

6.3.1 Limitations of Overall Design

An important strength of the study is the combination of cross-sectional and longitudinal research designs. Longitudinal designs in the form of natural experiments are particularly valuable because it is possible to compare within person change from baseline data. The study also estimates growth curve models using three waves. These models are better because they facilitate the identification of patterns of change within a multilevel framework. However, a

limitation for the accurate estimation of patterns of change was the number of data points that forced the trajectories of change to be linear, rather than quadratic, as descriptive analyses suggested. The few data points limited the number of random parameters as well, and thus cross-level interactions could not be tested.

A common limitation to the three approaches was the spacing between waves. It is possible that the time distance between exposure and outcome measurement was too long. The models could not adequately capture the fluctuation in the exposures, especially the homicide rates, and the distance among respondents varied by a year or more. Future research should try to shorten the distance between outcome and exposure and estimate with greater precision the cadence of intermittent stressors.

Attrition was relatively small and it is believed to be random. But people changing municipalities posed the threat of selection bias. Excluding those who moved entailed an important trade-off. While the study was able to differentiate between exposed and unexposed individuals to certain municipalities, the decision meant to work with a biased sample. However, as it is argued in appendix 1, the small bias may cause an underestimation of the effects, which was considered less severe.

Another unresolved issue in the dissertation was the uncertain geographic problem. Data availability forced the selection of municipalities as the key level. The assumption is that a municipality is the relevant level in which contextual exposures of interest coincide with political demarcations. However, Mexico City is comprised by 16 “delegaciones” or municipalities; one of them, the densely populated Cuauthemoc, houses 45 colonias with 79 zip codes. Conversely, Santiago Papasquiaro, in Durango, is sparsely populated and contains only14 zip codes. So, which is the adequate neighborhood and how homogenous should they be? It is not clear but the municipality was the smallest unit with the required data. An associated limitation was the

assumption that the most relevant exposures happen in the municipality of residence. This means that spatial polygamy reflected in two ways. First, people may move in two or three zip codes and eschew others within a municipality trying to evade deleterious exposures. The second is that people may move between municipalities and could spend most time elsewhere, for example, when the workplace is far from home. In both instances, the risk of misestimating the exposure was present, but municipalities still remain a common unit of analysis in Mexico.

6.3.2 Limitations with Sample

A worrisome limitation with the sample was the elevated number of missing values. Appendix 2 shows how this problem was dealt with. Nevertheless, the high percentages of missing values in the biomarkers –like cholesterol, especially in 2006– posed a serious threat of type II error where a null hypothesis is incorrectly retained.

Another concern with data collection was the different ways the biomarkers were gathered. The problem was evident with the 2002 blood pressure, which was taken only once and averages were higher than expected. But the issue loomed over the prevalences that were below the national estimates, as with hypertension. Nevertheless, these differences could be in the characteristics of the urban subsample and do not necessarily reflect a data quality problem. A greater problem with data collection would have yielded a higher proportion of implausible values but they were mostly within an acceptable range.

6.3.3 Limitations with Measures

A key limitation with the measures was that different biomarkers were collected each wave. Therefore, it was not possible to build the same robust indices for every wave and comparisons between methodologies are not exact. The next waves of the MxFLS should keep

the same biomarkers form 2012 and strive to include more biomarkers from different systems, as suggested by Juster et al. (2010).

The extent to which these limitations pose a serious threat to the findings is quite speculative. Perhaps the most worrisome threats are data quality –data collection and missing values–, wave spacing, and, to a less extent, selection bias. The first two are important concerns because there is little a researcher could do to assess the size and direction of bias –in contrast with the efforts to understand selection bias. Nevertheless, it is reassuring that the triangulation of three research designs, different statistical techniques, and descriptive analyses coincide in the main findings.

6.4 Conclusions

The findings in the dissertation attest for the importance of Social Ecology for Social Work's theoretical base and practice. Acknowledging the role of harmful neighborhood stressors and the value of institutional resources –such as Social Work practice– is fundamental for an integrated understanding of human suffering. Concentrated disadvantage and violence is a common context for Social Work practitioners in the USA, however, cultural competency requires the ability to assess how contexts under other social dynamics may not follow these common trends. Moreover, these findings broaden the understanding of how to listen and comprehend the client's experiences because subjective accounts of a pernicious context may draw an insufficient perspective. A lesson from this study is that the naturalization of poverty and violence, when taken as indifference, is rarely true for the body. Quite the contrary, cumulative exposures to stressful contribute to the weathering of the body.

Appendix 1. Selection bias into municipality

A common critique to claims about context effects is selection bias. It is usually very hard to discern if a "good" neighborhood causes beneficial effects in whatever domain of life because of the resources it offers or because its inhabitants were already predisposed for it based on an (unknown) individual characteristic that also led them to choose to live there. The reasons for which people choose the neighborhoods they live in may also guide them to particular individual outcomes (Sharkey & Faber, 2014). For example, parents with low education are more likely to live in poor neighborhoods and children in poor neighborhoods are also more likely to drop out of school (Small & Newman, 2001). The counterargument frequently comes from analyses on the restricted possibilities people have to change neighborhood in the first place, especially from the poorest ones. Actually, people rarely move because even "bad" neighborhoods offer benefits, like social capital; and when they do move, they go to similar places, with similar problems; so new neighborhoods are not necessarily better places (Robert J Sampson, 2008). It is mostly prosperous people who get to choose the neighborhood where they wish to live in. See a more in-depth discussion in the Literature Review.

Common neighborhood selection may be driven by several circumstances (Sampson, 2008), but when violence is a driver the reasons to move may change. A study on international migration in Latin American countries found that only in Nicaragua economic conditions and violence increases were associated with a decision to leave the country. In contrast, in Mexico rising violence actually reduced the likelihood of migrating (Massey & Aysa-Lastra, 2011). Demographic characteristics of migrants are also different from the average population. Migration in Colombia was more likely in people with higher education, with network connections to migrants, and during periods of greater violence and increased police presence

(Silva & Massey, 2015). International migration, however, is only one type of migration. Forced internal migration is another type, more frequently associated with violence. Mexico does not have reliable accounts of the size of the phenomenon, but some estimates place it, for 2014, at 287, 358 in 2016 (Pérez Vázquez, 2016); around 0.002% of the Mexican population. Therefore, selection bias in violent contexts is an important threat to the validity of the results.

A1.1 Analytic rationale

This bias showed up in the dissertation in how the respondents self-select into a municipality. Did they choose to stay in the same municipality because it was peaceful? Did they choose to migrate because it was violent? The issue matters because the dissertation tested cumulative exposures to the same context. When someone changed from one municipality to another between waves, then the exposure was confounded in two places and the harms and benefits of one context are impossible to disentangle with these statistical models; the problem of spatial polygamy (Graif et al., 2014). Thus, people who changed municipality were excluded thereby opening the possibility of self-selection. Perhaps the ones who stayed either liked the place they live in or lacked the means to move and had to endure multiple hardships. However, the problem is that those who left might be the most sensitive to a deleterious context. If this were the case, then the analyses would underestimate the context effects. Moreover, if only rich individuals living in neighborhoods that became "bad" were able to leave, then the analyses could overestimate the interaction with individual SES.

A difficulty when analyzing those who left the municipality was attrition. On the one hand, a person who decided to move because of the violence increase in 2006 could be harder to find in 2012 to answer the last wave of the survey. However, the reasons of attrition are speculative and cannot be fully adjudicated to migration. Thus, part of the 2012 attrition could be

due to violence and another part is for a variety of unknown reasons. On the other hand, the MXFLS survey followed migrants and thus offered some information on the reasons of their decision. However, the group of migrants that continued to be panel respondents was probably also incomplete. In sum, neither the 2012 attrition group nor the migrant groups were complete or provided enough information to rule out selection bias. Nonetheless, both profiles suggested how the main results of the dissertation could be biased and offered additional information on the complex processes of attrition and changing place of residence.

A1.2 Methods

The objective of the appendix was to analyze with descriptive statistics the differences between these two groups. It first compared 2005 values between people lost to attrition in 2012 and the ones in the sample. It then contrasted 2012 values between people who stayed in the same municipality between 2006 and 2012 with those who left during the same period. The statistical analyses were independent sample t-tests for continuous variables and chi-squares for dichotomous independent variables.

The sample for the analyses followed a similar procedure as the one explained for Sample 1. The sample excluded respondents with the following characteristics, in this order: 1) People without data; 2) Younger than 15 years old and older than 100; 3) Residing in rural municipalities; and 4) With implausible values in the dependent variables. Respondents with missing values in all the dependent variables were left in the sample in order to avoid deleting observations from the attrition subgroup. These cases were excluded with list-wise deletion in the analyses. The analytic sample was 22,050 individuals.

"Attrition" was a dummy variable coded 1 for respondents who entered the survey in 2002 or 2006 and were not interviewed in 2012; it excluded with missing values people who

refreshed the sample in 2012. The sample had 16,993 individual responses in 2012 (77.1%) and the attrition from 2006 to 2012 was 14.09% or 3,107 individuals; 1,950 were missing values (8.84%). "Same municipality" was a dummy variable coded 1 for panel respondents that registered different municipalities in the 2006 and the 2012 surveys when asked about their place of residence. 90% of the sample stayed in the same municipality between 2006 and 2012[16].

A1.3 Results

Table A1 shows results for both comparisons. The first column contrasted 2012 attrition based on measures from 2006. The profile of the people who did not continue with the survey was different from the ones who did. They were younger by almost four years, with an average age of 40, and more were single. This group had 66% less people that identified as indigenous. The ones who did not continue with the survey were considerably healthier in chronic diseases and comorbidities. Economically, they had higher levels of education (they double the percentage with a college degree) and household expenditure, but they did not experience more economic shocks than the ones who continued. A higher personal socioeconomic status was likely associated with the fact that they experienced more personal victimization events and expressed having slightly more fear than the ones who continued, but the differences were not significant for property victimization. People who did not continue with the survey also had mildly higher perceptions that the violence was going to increase in the following three years. Notably, however, in 2005 just 0.63% of the total sample considered migrating due to insecurity and the percentage was very similar in 2012: 0.61%. There were no statistically significant differences in this variable by attrition.

[16] The 10% of the sample who changed municipality, the 3,757 respondents, dropped to 2,046 in most statistical analyses by list wise deletion. This means that 1,711 cases had no values for the independent variables. Thus, a more precise estimate of the people who left their municipality is 8.4%; this figure is closer to estimations for the analytic sample in the Methods section.

Comparisons with the context variables indicated no differences by attrition in the 2006 or 2012 homicides rates and not even in the *change* variable. The ones who responded in 2012 came from poorer, more equal, and less densely populated municipalities than those who did not continue with the survey.

Differences in the dependent variables revealed that the ones who did not continue had better values than the ones who continued. Their mean CVDR was lower, just like their waist circumference, which was smaller by 2 centimeters. Blood pressure was slightly lower as well.

The second set of comparisons was between people who decided to stay living in the same municipality and the respondents who changed to a different one, which were excluded from the analyses of the dissertation. These comparisons were made with the same individual variables but collected in 2012. The profile between these two groups differed as well. A higher proportion of men (3%) stayed in their municipality. The ones who left were strikingly younger than the ones who stayed in the municipality (32 years vs 43 years) and they were also single in a higher proportion. Those who stayed had many more comorbidities but not more chronic disease. Interestingly, the group that left had a slightly higher educational education level, especially when comparing people without instruction and with secondary or high school. The opposite happened in primary and in college, suggesting that the ones who left mostly belonged to the middle-class. However, the ones who left came from smaller households and had higher household expenditures. Groups did not differ in economic shocks. Another striking difference was the victimization rate. Almost half of those who left experienced an event of personal victimization when only 7% of those who stayed did. There were no differences in property victimization. Surprisingly, the ones who left did not perceive more fear, and had similar perceptions about the possibility of a raise in violence. The ones who left had no significant differences on whether they had ever thought about migrating because of the insecurity.

Table A1

Mean and Proportion Comparisons of Person-Level Variables by 2012 Attrition and If Respondents Stayed in the Same Municipality.

Individual variables	**Attrition 2006-2012**		**Stayed in the same municipality 2006-2012**	
N=22,050	**In** N=16,993	**Out** N=3,107	**Left** N=3,757	**Stayed** N=18,293
Male	45.7%	47.1%	43.3%	45.9%*
Age	43.84	40.01**	31.8	42.8**
Indigenous	6.4%	1.8%**	5.3%	5.9%
Disability	2.1%	2.0%	1.75%	2.34%
Household size	5.39	5.25*	5.6	5.45*
Single	33.6%	46.3%**	22.1%	31.9%**
Chronic diseases	7.1%	5.1%**	7.9%	7.3%
Comorbidities	11.1%	7.2%**	9.7%	14.3%**
Education: No instruction	9.2%	5.4%	4.7%	9.3%
Primary	41.2%	26.9%	30.3%	39.7%
Secondary	26.0%	31.0%	34.6%	26.7%
High school	14.1%	18.1%	21.0%	14.2%
College or higher	9.6%	18.6%**	9.3%	11.1%**
Household expenditure	4,687.8	5,219.2**	4,637.7	4,414.8**
Household Economic shocks	22.8%	23.1%	32.2%	31.5%
Personal victimization	7.6%	12.6%**	45.4%	7.1%**
Property victimization	3.9%	4.1%	5.4%	4.7%
Fear of crime	1.15	1.39**	1.4	1.33
Violence increase	.72	.77*	.719	.729
Migrate for insecurity	0.71%	0.61%	0.52%	0.62%
Context variables				
Homicide rates 2006	10.1	9.9	9.75	10.11
Homicide rates 2012	28.9	28.07	29.44	28.77
Homicide rate *change*	10.0	9.8	10.7	9.9
Assets Poverty	49.4	43.2**	50.7	49.2**
Income inequality	0.435	0.436*	.405	.404
Population Density	920	2083**	927	1101*
Dependent variables				
CVDR	0.647	0.586*	.790	.773
Waist circumference	90.5	88.2**	89.8	93.9**
Diastolic BP	75.6	75.4	76.3	79.1**
Systolic BP	117.3	116.3*	117.5	123.8**

*=p<.05; **=p<.01. The measures corresponded to 2006 when comparing attrition and they were from 2012 when comparing same municipality. The sample comparing attrition is smaller because it does not include 1,950 people who refreshed the sample in 2012. Mean differences were conducted with T-tests. Differences in proportion were conducted with chi-squares. Education was tested as a continuous variable but it is displayed as ordinal to ease the comparison; it was always statistically significant.

The context variables further suggest that the ones who left did not live in more violent places than the ones who stayed. There were no significant differences on the 2006 and 2012 homicide rates or in the *change* variable. The municipalities of those who stayed were slightly poorer and more densely populated but inequality was the same.

The dependent variables showed that the ones who left also had better results. Although the CVDR showed no statistical difference, waist circumference was lower by four centimeters and blood pressure was also lower.

A1.4 Conclusions

The profiles from the people lost to attrition in 2012 and the ones who changed to a different municipality by 2012 had some common features. These characteristics suggested where the biases in the dissertation´s analyses were. Both groups were younger and had a higher proportion of single individuals. They were also more educated, came from smaller households, had a higher household expenditure, and lived in more affluent municipalities. They also suffered more personal victimization events but did not differ in their perceptions of a violence increase in the next three years. Crucially, they would not migrate due to insecurity and they did not live in violent municipalities. The two groups were also healthier and this was evident in comorbidities and the dependent variables.

There were four important findings to judge selection bias. First, the proportion of people lost to attrition (15%) and due to changing municipality (10%) was somewhat low, so the validity threat is low as well. Second, there were no statistical differences in any of the groups when comparing the key independent variables at the municipality: the homicide rates. Most of the evidence suggested that the motivations to interrupt the survey or change municipality were

unrelated with insecurity. The only exception was personal victimization but, as explained in appendix 2, this rate was most likely confounded by socioeconomic status. Third, the differences in the dependent variables indicated that the analytic sample of the dissertation could be slightly overestimating the prevalence of CVDR, waist circumference, and blood pressure. Finally, differences in the independent variables showed that younger, healthier, and wealthier individuals were more likely to move to a different municipality and thus the effects on affluent municipalities may be underestimated.

A recent study using the same survey and context measures but with a different outcome variable faced the same issue i.e. "whether our sample of interest is selected due to attrition in a way that is correlated with the change in the conflict environment" (Brown et al., 2017). They did not find evidence that it was biasing their results. Nevertheless, as in the dissertation, their solution was to establish the 2006 municipality as baseline for further analyses:

> "While there is not a significant relationship between potential violence exposure and migration in general we do find evidence of violent crime induced selective migration that is related to the ruralness of the respondent's location of residence and the respondent's marital status. […] In order to shield our estimates from the bias of endogenous migration, we follow an intent-to-treat approach in our empirical specification. To do this an individual is assigned a conflict exposure level based on their municipality of residence in MxFLS2, before the rise in crime, rather than based on his/her current municipality of residence. Thus, the intensity of violence exposure assigned to a respondent is independent of any migration decisions made as a response to crime" (Brown et al., 2017, p. 13)

To sum up, although the validity threat is a possibility that only experimental designs can fully rule out, there is enough theoretical and empirical evidence to argue that this threat is low in the present study. Moreover, the results of this Appendix indicated the directions of this minor bias so readers can interpret the results with due caution.

Appendix 2. Dealing with missing values

As is common in most surveys both samples have plenty of missing values (Heeringa, West, & Berglund, 2010). However, an important limitation of the survey is the large number of missing values, particularly in the biomarkers. A sound strategy to deal with them is using multiple imputations (Raghunathan, 2016). A detailed account for each sample follows.

A2.1 Sample 1

The first sample comprises the 2006 and the 2012 waves. Table A2 shows a comparison of the amount of missing values in the dependent variables. Sociodemographic variables have few missing values, all below 5%. The independent variables have slightly higher percentages (5-10%); the highest are collective efficacy with 13%, household victimization with 12%, and personal victimization with 26.8% of missing values. However, the most worrisome variables are the ones from the biomarkers. Blood pressure and waist-circumference, as well as reports on comorbidities and smoking habit, are among desirable ranges (0-5%). The problem concentrates in the two cholesterol measures and glycosylated hemoglobin because they exceed 50%. Even though this is in part corrected by the self-reported diagnoses, they affect the number of missing values in the indices. The problem is worse for 2006. While the few records in total cholesterol are similar (63%), the percentage in missing values on blood pressure and waist circumference rise up to 30%.

The exclusion of several observations helped to establish that the missing data pattern is not monotone (i.e. due to attrition) or caused by filter questions. The problem with the two cholesterol measures mostly follows a univariate pattern because some people refused to provide blood samples (Raghunathan, 2016). The missing data mechanism for the rest of the cases, and for the other variables, seems to be random (MAR). A sound solution to avoid bias due to MAR

missing data is multiple imputations (Raghunathan, 2016). For datasets with variables at different levels of measurement the recommended algorithm is chained equations with at least 20 imputed datasets if the number of missing values exceeds 50% (Raghunathan, 2016).

Therefore, STATA software (version 13.1) was used to impute 20 datasets for the 2006 and 2012 sample using the mi impute chained command with "54321" as seed. The augment command was included to avoid runs with "perfect predictions" by adding a very small weight to a few observations. The imputation was mostly for the biomarkers but it used many auxiliary variables. All the biomarkers were included for 2012 and 2006. In addition, the weight variable, the sociodemographic variables, and the independent variables for the validation model were considered as predictors. The indices were built after the imputations (Raghunathan, 2016).

Table A2 shows a comparison between the means and standard deviations of the dependent variables. The first means were calculated for the original sample using the cross-sectional sampling weights of the health module of the survey (Heeringa et al., 2010). The "MI Means" consider the 20 imputed datasets. As expected, the imputed means and standard deviations are fairly similar to the original means, even for both types of cholesterol and the both indices. However, for the 2006 variables, the imputations tend to overestimate blood pressure, cholesterol, and waist circumference. In spite of the imputations the missing data is certainly a limitation if the expected effect of the models is small.

The municipality-level variables were complete, except for the homicide rates. Because most municipalities had at least one value, averaging the yearly homicide rate solved most of the missing values problem. However, one municipality did not have any of the rates for 2012 and 4 municipalities didn't for 2006. In an analysis not shown, a similar multiple imputation with 10 datasets was conducted. Since HLM software does not allow MI datasets at the second level, the MI datasets were averaged to obtain a point estimate, which substituted the missing values.

Table A2

Mean comparison between original dataset for sample 1 and imputed dataset (m=20)

Waves	Indicators	Original N	% Miss	Min-Max	Mean (sd) or proportion	MI Mean (sd)
2012	Female	16,679	0	0-1	36%	36%
	Age	16,679	0	15-99	55.09 (0.74)	55.71 (0.76)
	BMI	6,458	61.3	12.79-81.96	28.17 (0.42)	28.12 (0.36)
	Systolic	16,679	0	72-228	128.67 (2.29)	129.17 (2.07)
	Diastolic	16,679	0	48-140	80.97 (1.10)	81.20 (1.02)
	Total Cholesterol	6,015	63.9	150-300	196.33 (2.22)	195.60 (2.23)
	HDL-Cholesterol	7,645	54.2	25-99	39.22 (0.92)	38.59 (0.83)
	Diabetes	15,843	5	0-1	16%	16%
	Smokes	16,679	0	0-95	1.7 (0.93)	1.8 (0.81)
	Waist circumference	15,865	4.9	50-166	95.61 (0.88)	95.85 (0.78)
	Framingham	7,325	56	0-20	8.28 (0.31)	8.41 (0.29)
	CVDR	11,589	30.5	0-5	1.263 (1.12)	1.181 (1.02)
2005	Age	16,679	0	15-100	55.40(0.73)	56.01 (0.75)
	BMI	6,317	62.1	13.84-113.02	28.75 (0.52)	28.65 (0.45)
	Systolic	11,701	29.8	60-230	120.32 (1.38)	120.88 (1.28)
	Diastolic	11,701	29.8	30-140	77.55 (0.91)	77.78 (0.86)
	Total Cholesterol	6,015	63.9	150-300	191.88 (2.47)	191.80 (2.37)
	Diabetes	12,488	25.3	0-1	10	10
	Smokes	16,679	0	0-90	0.33 (0.21)	0.56 (0.30)
	Waist circumference	11,380	31.7	49-170	94.28 (0.87)	94.5 (0.81)
	CVDR	11,762	29.4	0-5	0.871 (0.91)	0.671(0.85)

A2.2 Sample 2

The sample for the growth curve models required a different treatment because the dataset had a different structure and the HLM software has different constrains. In these models three measures of the biomarkers (corresponding to the years 2002, 2006, and 2012) are nested in individuals who are then nested in municipalities. Therefore, the growth model was a three-level model but HLM software only allows multiple imputation at the first level i.e. only for the biomarkers. The key limitation was that individual variables were now at the second level and missing values were then handled with list-wise deletion. The issue became which controls to

include limiting the sample loss; controls with many missing values could dramatically reduce sample size. In the dissertation the most worrisome problem was personal victimization. Having victimization as a control meant a 27% sample reduction. Since other analyses showed personal victimization was highly correlated with household expenditure, only expenditure was used as a control.

Table A3

Mean comparison between original dataset for sample 2 and imputed dataset (m=10)

			No Sampling Weights	Sampling Weights	Multiple Imputation
	# obs	% missing	Mean	Mean	Mean
SBP long	35,630	20.1	123.2	123.8	123.3
DBP long	35,582	20.2	78.1	78.4	78.2
WC long	35,882	19.5	89.9	91.6	90.6
SBP 02	11,266	24.2	128.09	130.0	128.0
SBP 05	11,961	19.6	117.6	117.8	117.6
SBP 09	12,403	16.5	124.3	123.4	124.3
DBP 02	11,218	24.5	79.2	80.2	79.4
DBP 05	11,961	19.5	75.7	76.2	75.7
DBP 09	12,403	16.5	79.3	79.5	79.3
WC02	12,300	17.2	85.1	87.8	86.5
WC05	11,656	21.5	90.8	92.2	90.7
WC09	11,926	19.7	94.2	94.4	94.2

SBP=systolic blood pressure; DBP= Diastolic blood Pressure; WC= Waist circumference. The abbreviated years are 2002, 2006, and 2012.

The structure of the sample 2 also differed in the shape from sample 1. Whereas cross-sectional analyses had a wide form, longitudinal analyses required a long form, in which a row reflects a measurement occasion and not an individual. Multiple imputations were done in wide format and then restructured to a long form to load them in HLM software.

Table A3 showed that the missing value problem was less severe in sample 2 than in sample 1. The largest proportion of missing values was in the two measures of blood pressure obtained in 2002; both at 24.2%. Therefore, ten datasets sufficed for accurate estimation (Raghunathan, 2016); besides, these were the maximum number of imputations allowed in HLM

software. The same imputation procedure as in sample 1 was followed, using chained equations. Table A3 revealed that estimates were similar regardless of the form of the dataset, the use of longitudinal weights, or the estimation with multiple imputations. The largest differences were with waist circumference in the long form and for the years 2002 and 2006.

Appendix 3. Validation of dependent variables

A3.1 Rationale and statistical analyses for the validation process

The validation process aims to examine if a measure actually represents what it is intended to measure (DeVellis, 2016). It refers to the agreement between a measure and the quality it is believed to be measured (Kaplan & Saccuzzo, 2012). In the dissertation, the validation analysis assesses if the indices and single biomarkers are sensitive to stress, first at the individual level and then at the municipality level. While content validity is argued in the theoretical framework, construct validity is used in the absence of a well-established criterion, as is the case with stress. A researcher must then argue that a measure has meaning using convergent or divergent evidence. Convergent validity is how well a measure correlates with other tests, how they both narrow in into the same construct. Since there is no one criterion that serves as final proof, the convergent correlations should be demonstrated with several variables because each pair of measures is only partially associated among them (Kaplan & Saccuzzo, 2012).

In addition to the partial theoretical association, in validation studies correlations tend to be "modest" because tests frequently have low reliabilities (Kaplan & Saccuzzo, 2012). The reliability of a measure strongly depends on measurement, response, and processing error; even when sampling weights account for coverage, sampling and non-response error (Groves et al., 2009). For instance, Kaplan & Sacuzzo (2012) show how a college performance test with good predicting qualities only reaches a correlation of 0.40.

The traditional validation process is performed only with correlations, so the first attempt to show bivariate associations is with pair-wise correlations in STATA 13. A disadvantage of correlations, however, is that they confound the variance at both levels. In addition, validation

tests of person-level dependent variables are usually done with predictors at the same level (Kaplan & Saccuzzo, 2012). Nonetheless, a core tenet of the dissertation is that biosocial processes have cross-level influences. Therefore, the validation process was extended to the municipality, albeit measurement error could be even greater than at the person-level and thus the associations are probably weaker.

Accordingly, the validation process was complemented with multilevel regressions without covariates using HLM software. The estimation method was maximum likelihood with random intercepts and the models partition the variance at the person and at the municipality levels. The estimation was done with robust standard errors using 10 datasets from multiple imputations (the maximum allowed by HLM software). For simplicity in the interpretation of the results, even though the variance terms at the municipality level were always statistically significant -thus confirming the importance of variance partitioning- they were omitted in these analyses.

Theoretically, the risk Framingham score and the CVDR index, as well as the single biomarkers (waist circumference, systolic, and diastolic blood pressure), should be positively associated with several stressful situations. Subjectivity makes it difficult to predict which situations are more stressful than others so several variables for each set were tested. The hypotheses for the person level validation are as follows:

- The indices and single biomarkers are significantly and positively associated with these health variables: Chronic diseases, worse subjective health, depression, BMI, and negatively associated with hours of sleep.
- The indices and single biomarkers are significantly and positively associated with these economic variables: unemployment, household economic shocks, and negatively associated with household expenditure in quintiles.

- The indices and single biomarkers are significantly and positively associated with these violence variables: personal victimization, property victimization, fear of crime, perception of violence increase, and negatively associated with collective efficacy.
- At the municipality level, the indices and single biomarkers are significantly and positively associated with these context variables: 2012 homicide rates, *change* in the homicide rates (2006-2012), the 2012 multidimensional poverty index, the 2012 assets poverty line, the 2012 GINI coefficient, and population density.

A3.2 Person-level bivariate associations with sociodemographic variables

Sociodemographic variables are considerably associated with the dependent variables; see table A4[17]. Males have significantly higher scores than females in the single biomarkers. Surprisingly, however, there were no significant differences in the CVDR index. The Framingham score was not tested for gender and age because the index construction already adjusts for these variables. Regarding marital status, singles showed mixed findings in the direction of the association. They had higher means in the Framingham score and systolic blood pressure while they were lower in waist circumference. No significant differences were found with the CVDR index and diastolic blood pressure. Self-identified people as indigenous had lower means in waist circumference. Disabled people had consistently higher means in all the dependent variables except for waist circumference. Correlations indicate that education was negatively associated with all of the dependent variables and the strongest association was with the Framingham score. Likewise, older age was positively associated with the dependent

[17] The analyses on this table do not consider multiple imputation or sampling weights because of software restrictions.

variables and the strongest association was with systolic blood pressure; see graph A in Figure A3.1.

Table A4
Mean Differences and Correlations Between the Dependent Variables and the Sociodemographic Variables

	Framingham	**CVDR**	**Waist**	**Systolic BP**	**Diastolic BP**
Male [a]	--	0.03	-2.52**	-9.14**	-3.01**
Single [a]	-0.99**	0.05	3.32**	-1.31*	0.00
Indigenous [a]	-0.26	0.06	3.91**	-0.80	0.61
Disability [a]	-2.66**	-0.23**	1.41*	-11.81**	-1.57*
Education [b]	-0.390*	-0.192*	-0.091*	-0.242*	-0.099*
Age [b]	---	0.369*	0.235*	0.471*	0.211*

a= mean differences; b=correlations; *=p<.05; **=p<.01

A3.3 Person-level validation of dependent variables

The validation models tested the associations with the five dependent variables: the Framingham score, the CVDR index, the waist circumference, and systolic and diastolic blood pressure. For ease in the interpretation, the independent variables were grouped in three sets: health variables, economic variables, and violence variables. Each variable was tested by itself, without controls, one at the time. The results are in Table A5.

Health variables

The Framingham score was positively and significantly associated with all the health variables (i.e. chronic diseases, subjective health, symptoms of depression, BMI, and hours of sleep), except for hours of sleep. For instance, an additional chronic disease is associated with a 1.29 increase in the Framingham score. CVDR was very similarly associated with the health variables but the association was weaker with chronic diseases, perhaps due to age, and it was negatively associated with hours of sleep. Waist circumference was also positively associated with the independent variables and negatively with hours of sleep. For example, an additional level in reporting worse subjective health was associated with two extra centimeters of waist

circumference. Both types of blood pressure were similarly associated with the health variables but the systolic blood pressure showed a stronger relationship. Neither was associated with chronic diseases. However, an additional level in worse subjective health raised systolic blood pressure by 4 and diastolic blood pressure only by 1.3. Both had a negative association with hours of sleep.

These results suggest that both types of dependent variables, the indices and the single biomarkers, are sensitive to multiple indicators of worse health.

Economic variables

One of the important and expected findings is that the Framingham score was negatively associated with household expenditure; an extra quintile in expenditure reduces the score by 0.472. This clearly shows an economic gradient in health; see graph B in Figure A3.1. Likewise, the Framingham score is positively associated with unemployment but it is probably confounded with age. The score was positively related with household economic shocks, but at the 90% confidence interval ($p=0.08$). The CVDR index had similar associations as the Framingham score but they were weaker. The economic gradient is smaller and the association with economic shocks is not significant. Conversely, waist circumference was positively associated with household expenditure. Nevertheless, it was associated in the expected direction with economic shocks. Blood pressure was negatively associated with household expenditure and unrelated with economic shocks.

These results confirm the theoretical expectation of a socioeconomic gradient with health (M. Marmot, 2015), especially measured as household expenditure. Waist circumference showed a mild gradient in the opposite direction. Unemployment does not seem to be a precise variable for validation purposes for the full sample. Household economic shocks show very low

correlations, except for waist circumference. Perhaps the temporal distance between the events diminishes the associations.

Violence variables

All the dependent variables had an association with the violence variables (i.e. personal and property victimization, fear of crime, perception of a recent violence increase, and collective efficacy) but, unexpectedly, the relationships go in the opposite direction than theoretically expected. For instance, a person who was personally victimized had a 1.6 *lower* Framingham score and one who suffered property victimization had a 0.6 *lower* score than one who was not victimized. The same occurs with the CVDR index, waist circumference and blood pressure. Notably, the negative association appears as well with subjective measures such as fear of crime and the perception of a recent violence increase: the most fearful have better scores in the Framingham score.

Table A5

HLM regression coefficients on the dependent variables by health, economic, and violence variables

	Fram. score	**CVDR**	**Waist**	**Systolic BP**	**Diastolic BP**
Health Variables					
Chronic diseases [b]	1.291**	0.202**	0.913**	-0.076	-0.432
Subjective health [a]	0.148**	0.311**	2.181**	3.993**	1.310**
Depression [a]	0.078**	0.012*	0.042**	0.001	-0.014
BMI [a]	0.112**	---	1.896*	0.705**	0.543**
Hours of sleep [a]	0.005	-0.022*	-0.479**	-0.389*	-0.021**
Economic Variables	**Fram. score**	**CVDR**	**Waist**	**Systolic BP**	**Diastolic BP**
HH Income quintiles [a]	-0.472**	-0.041*	0.302**	-1.446**	-0.197*
Unemployment [b]	2.87**	0.259**	-0.240	0.989*	-0.554*
HH Economic shocks [b]	0.209 ◊	0.014	0.741*	0.259	0.282
Violence Variables	**Fram. score**	**CVDR**	**Waist**	**Systolic BP**	**Diastolic BP**
Personal Victimization [b]	-1.659**	-0.217**	-1.363**	-4.115**	-1.320**
Property Victimization [b]	-0.599**	0.053	0.322	-2.458**	-0.517
Fear of Crime [a]	-0.101**	-0.007	-0.061	-0.074*	-0.123*
Violence increase [b]	-0.0965	-0.019	0.015	-0.799**	-0.151
Collective efficacy [a]	0.045**	0.006*	0.009	0.167**	0.060*

a= grand-mean centered; b=uncentered; ◊=p<.10;*=p<.05; **=p<.01; Models were estimated with restricted-maximum likelihood and with robust standard errors. The multilevel models partition the variance of the dependent variable between the person-level and the municipality level; the variance at the municipality level was always small

but significant. The variances of the independent variables are fixed. All regressions were conducted with robust standard errors and multiple imputations: 10 datasets.

The paradoxical correlations between victimization and the biomarkers may be confounded by the economic gradient of health. On the one hand, as graph B shows, populations living in poverty (i.e. first and second economic quintiles by household expenditure) have worse levels on these biomarkers. On the other hand, populations with higher expenditure by household (i.e. fourth and fifth economic quintiles) suffer on average more personal assaults and report higher levels of fear than those in poorer strata; see graph C & D. People who report more violent events are also the ones in the population stratum with lower cardiovascular risk. Therefore, both stressors, poverty and violence, are confounded in the correlations and appropriate modeling to control for other factors would reveal the strength of these associations.

A3.4 Municipality-level validation of dependent variables

Table A6 contains the multilevel associations of the municipality variables with the six dependent variables. The results show that the indices were not as sensitive to the context predictors as the single biomarkers. The Framingham score was not significantly associated with any predictor; see graphs K-N in Figure A3.1. The CVDR was the most sensitive index to the context variables. While it was unrelated to the 2012 homicide rate it was positively associated with homicide rate *change*. However, it was negatively related to multidimensional poverty, suggesting that poorer municipalities are not as stressful as richer ones. The CVDR index was not associated with assets poverty, the GINI coefficient, or population density.

Waist circumference had the expected positive associations with the violence variables. People in municipalities with high homicide rates and a higher *change* in the homicide rates had larger waist circumferences. However, as with the CVDR index, both measures of contextual poverty were negatively associated with waist circumference. The two measures of blood

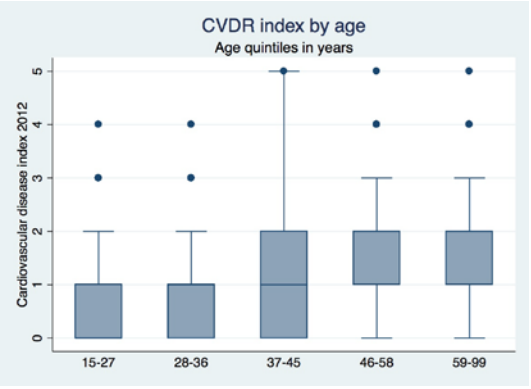
pressure were sensitive to some contextual predictors. They were sensitive to changes in the homicide rates but only diastolic blood pressure captured the negative association with the poverty measures. The GINI coefficient and population density were barely associated with systolic blood pressure.

Table A6
HLM regression coefficients on the dependent variables by municipality level predictors

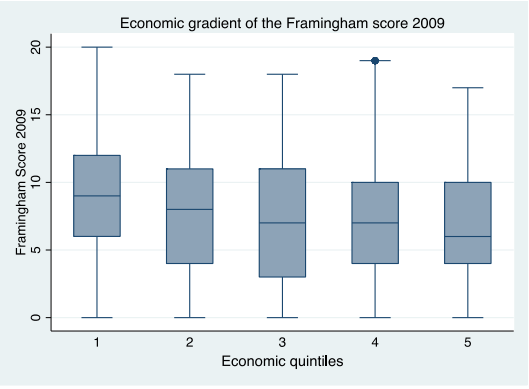
	Fram score	**CVDR**	**Waist**	**Systolic BP**	**Diastolic BP**
Homicide rate 2012	0.007	0.001	0.045**	0.027	0.007
Homicide rate *change* 2006-2012	0.005	0.002**	0.071**	0.044*	0.026*
Multidimensional poverty 2010	0.004	-0.003**	-0.081**	-0.016	-0.024*
Assets poverty 2010	0.004	-0.004	-0.106**	-0.030	-0.035*
GINI 2010	-2.516	-0.123	6.230	-15.56◊	-1.422
Population Density 2010	0.000007	-0.00005	0.00003	-0.001◊	0.000001

a= grand-mean centered; b=uncentered; ◊=p<.10;*=p<.05; **=p<.01. The multilevel models partition the variance of the dependent variable between the person-level and the municipality level; the variance at the municipality level was always small but significant. The variances of the independent variables are fixed. All regressions were conducted with robust standard errors and multiple imputations: 10 datasets.

The municipality level validation tests yielded mixed results. The indices did not seem to be the most adequate measures to capture contextual effects. CVDR was the most promising perhaps because it is created exclusively with biomarkers and does not control for age and sex. The single biomarkers proved to be sensitive to changes in context but they were not capturing exactly the same processes. Among the predictors, *change* in the homicide rates was the most consistent. The poverty measures were also important but in the opposite direction so they require further examination. The least useful predictors were inequality and population density, which were not associated with any predictor.



A



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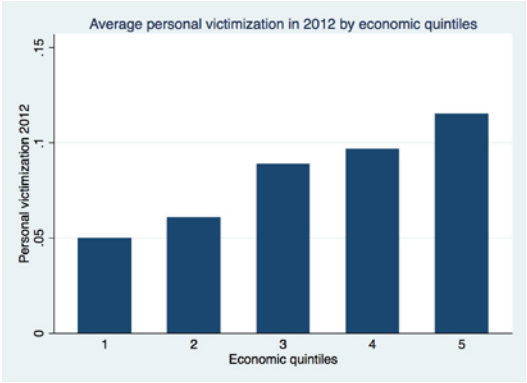

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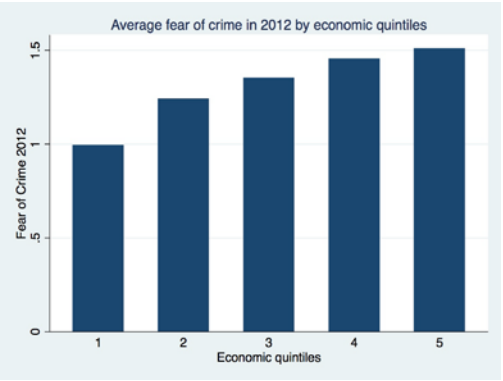
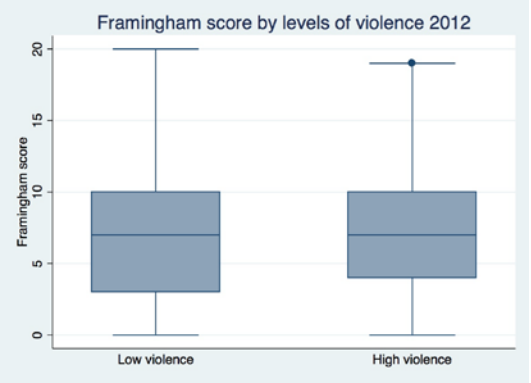
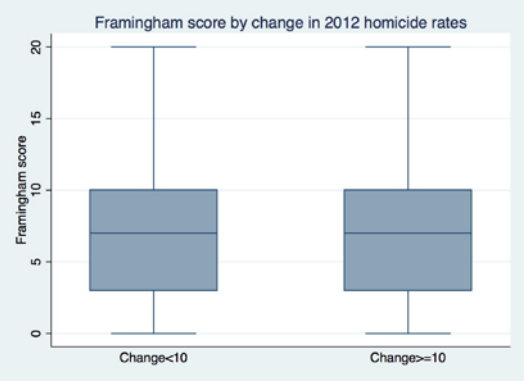


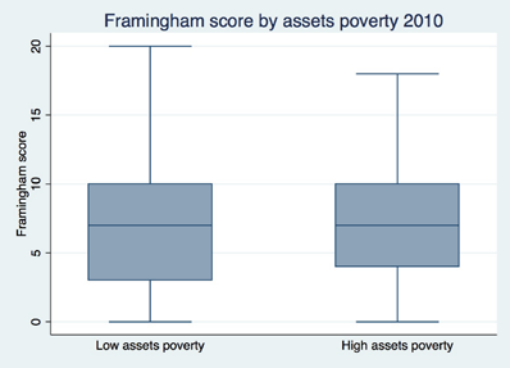
D



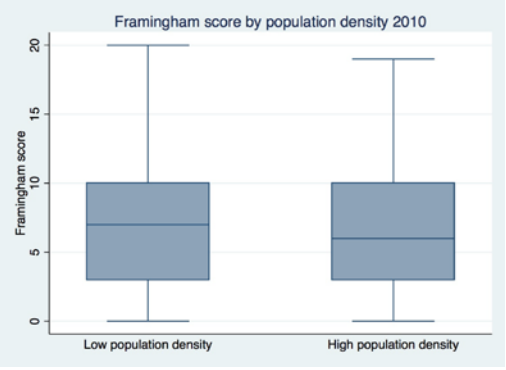
E



F



G



H

Figure A3.1. Graphs illustrating several issues from the validation process

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