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Working Papers / Documents de travail

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Florent Dubois

WP 2017 - Nr 20

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Florent Dubois†
Aix-Marseille Univ., CNRS, EHESS, Centrale Marseille, AMSE
Université du Maine

May 18, 2017

Abstract

Few studies tried to quantify the relative importance of each determinants of residential segregation. This mainly comes from a reverse causality problem which hampers the identification of the quantity of interest. In this paper, we decompose the whole change in segregation between 2001 and 2011 in South Africa by using segregation curves. We show that, even without an experimental setting (which might be impossible to obtain), identification of the causal effects can still be achieved by using the dynamics of the phenomenon. The provision of basic public services appears to be one of the main explanation of the gap observed, while differences in sociodemographic characteristics play a minor role only for the least segregated neighborhoods. Housing market is responsible for an important part only among neighborhoods intermediately integrated, while past segregation and income influence moderately segregation throughout more than half of the South African neighborhoods.

Keywords: Post-Apartheid South Africa, Generalized Decompositions, Residential Segregation, Causal Inference

1 Introduction

Segregation and the formation of ghettos is often a major concern for local governments. However, empirical researchers do not have devoted much attention to the questions of the determinants of segregation and how much they are responsible

*I would like to thank Ahmed Tritah for helpful discussions.
†florent.dubois@univ-amu.fr

for the levels observed. To the best of our knowledge, only Bayer et al.[4] try to answer this fundamental question.[1] On the contrary, there is an abundant empirical literature on the consequences of segregation.[2] In this paper, we tackle this question and provide causal evidences on the relative importance of each determinant of residential segregation.

The difficulty of the analysis of the causes of segregation is probably an important limiting factor. In fact, analyses of segregation suffers from a reverse causality problem as each cause of segregation might as well be caused by segregation itself. Hence, it is much easier to find an exogenous variation of a single cause and study its multiple consequences rather than exogenous variations of multiple causes on a single consequence. The development of causal inference in the 1980's[3] and the beginning of randomized experiments of relocation policies such as the Gautreaux program or the Moving To Opportunity experiment were probably important factors in the orientation that empirical research took.[4] Indeed, these programs provided a natural experiment with an exogenous variation of segregation allowing researchers to compare the economic and social performance of similar individuals in different segregation contexts. Therefore, inferring on the consequences of segregation was possible.

This huge bias in the empirical research might as well be due to a more pragmatic reason than the development of the necessary tools. In a sense, focusing this much on the consequences of segregation is a way to answer indirectly the fundamental question that Cutler and Glaeser[17] ask explicitly: "Are ghettos good or bad ?".[5] Indeed, why would we have to care about segregation if it had no effect at all? Therefore, if segregation has no impact, it is pointless to learn about the causes of the phenomenon. However, previous researchers have shown that segregation is responsible for worse outcomes for Blacks in schooling, employment, and single parenthood, among other socioeconomic outcomes.

On the other hand, providing causal evidences of the sources of segregation is highly difficult because there does not exist a natural experiment that would be informative of all the determinants of segregation simultaneously. Thus, quantifying the relative importance of each determinant of segregation is almost impossible.

[1] Vigdor[54] answers the important question "Are the residential preferences of each racial group compatible?". He shows that it is impossible to match the ideal Black neighborhood. Although interesting, neither he quantifies the importance of preferences in determining the levels of segregation observed, nor he provides causal evidences, and he limits his analysis to preferences only.

[2] See Massey[34], Crane[16], Cutler and Glaeser[17], Katz et al.[31], Oreopoulos[42], and Krivo et al.[33] among other references.

[3] See Holland[28] for a review of the earliest development of causal inference.

[4] See Keels et al.[32] for the Gautreaux program and Katz et al.[31] for the MTO experiment.

[5] To the best of our knowledge, only two other papers try to answer explicitly this question, Borjas[11] and Cutler et al.[19]

In their attempt, Bayer et al.[4] lack a credible causal reasoning for their decomposition analysis of segregation in the San Francisco Bay Area. However, note that usually decomposition analyses do not treat the causality question and stay at a descriptive level. Thus, their analysis still provides interesting indications. Differences in basic socioeconomic characteristics explains around 30% of the segregation experienced by Blacks and Whites.

In this paper, we provide a general identification strategy of the causal effects of each determinant of segregation. By exploiting the dynamics of the process, we demonstrate that conditioning on all the determinants of segregation at the previous period is enough to identify causal effects, even without a natural experiment. Following Chernozhukov et al.[15], we further propose a detailed decomposition method for segregation curves applied to segregation in South Africa.[6] We use distribution regression methods to estimate counterfactual segregation curves. Then, by adding sequentially sets of covariates along all the possible sequences, we can recover the causal contributions of each of the determinants of residential segregation.

We do not limit our analysis to the decomposition of the mean levels of segregation. This allows us to identify precisely which neighborhoods are essentially affected by a change in a particular covariate. For instance, we find that the basic characteristics of a dwelling (surface, number of rooms...) have only an impact for neighborhoods already experiencing some intermediate levels of integration, while sociodemographic characteristics only matter for almost completely Black neighborhoods. This brings nuances to the work of Bayer et al.[4]. From our findings sociodemographic characteristics matter only for a small share of neighborhoods. Moreover, when it matters, it accounts for around 25% only. Finally, our results seems to point toward the provision of public services as the main source of segregation.

The paper proceeds as follow: section 2 describes our identification strategy of causal effects. Then we detail our method for the detailed decomposition of segregation curves in the next section. A presentation of the data is then provided in section 4. Finally, we discuss our results and their implications in the last section.

[6]See Duncan and Duncan[21], Massey and Denton[35], and Hutchens[29] for a complete overview of segregation curves.

2 *"Correlation is not causation"* versus *"No smoke without fire"*

2.1 General approach to causal inference

Empirical researchers are usually interested in causal questions such as "Does X cause Y?" and if so the following natural question "By how much does X change Y?". Decomposition analyses consider mainly the second question while leaving the first question unsettled. Thus, most decomposition studies are simply descriptive and do not draw much attention on inference and causality questions.

But answering the question of causality is not a simple problem, and most of the time the decomposition analysis is already interesting on his own. But if we want to further understand complex social phenomena such as residential segregation some causal inference has to be made.

The problem of causal inference is that we usually cannot be sure, when two variables X and Y correlate, if this is truly a causal relationship.[7] It might be because the causal relationship is interpreted in the wrong direction, X does not cause Y but Y does cause X. Or it might be that a third unobserved variable Z is causing both. Or it might simply be randomness.[8]

When interpreting the correlation between X and Y, we usually have a model in mind such as quantitative easing will generate inflation or the consumption of a household will increase if its income raises. If only one direction is possible to explain the correlation between the two variables, then the problem of reverse causality is avoided. In the previous examples, quantitative easing might be implemented in response to a rate of inflation too low, creating potentially a reversed causality, whereas the increase of consumption of a household will most likely not increase its income, thus it is safe to think about a relationship going from income to consumption.

The threat of a confounding variable Z which might cause both X and Y is much more serious. This potential confounding variable usually arises from theoretical reasonings and the fact that the key variables are unobserved from the researchers. In the consumption example, you might be studying agricultural households in a developing country where consumption and income are determined by their agricultural production which might not be observed or that you did not take this particularity into account in modelling your problem.

To overcome this endogeneity problem, we either rely on instruments for the particular mechanism at play or on experimental/quasi-experimental settings.[9]

[7] Thus, the famous "correlation is not causation" mantra.

[8] Although some basic reasoning usually rules out randomness.

[9] Treatments in this experimental setting can also be viewed as an instrument.

Both methods aim at ensuring that no confounding variables still threaten the estimation of a causal effect. The experimental setting is actually the dominant approach to talk about causality. It is dominant because of its simplicity and the credibility it gives to estimates making social sciences looking like hard sciences.

Imagine that we are interested in the variable Y which might depend on having received a treatment or not. This could be a medical trial for a new drug or the implementation of a conditional cash transfer. If we could compare the value of Y for the same individual with and without the treatment, the difference between the two would give us the causal effect of the treatment on Y. However, the fundamental problem of causal inference is that we cannot observe the same individual with *and* without the treatment simultaneously.[10] If we want to study the effect of an additional arithmetic lecture on the score of a fourth-grader to a maths test, then we could only observe the score of the pupil if he takes the lecture or do not, but not both. Thus, the value of the variable Y that the individual would have get if he was not treated will be a counterfactual value for the treated denoted Y^c, while the value obtained when treated will be Y^t.

By making some homogeneity assumptions concerning the temporality or the individuals, we can recover some meaningful causal effect. If a treatment does not persist when the individual is no longer exposed and if it does not matter when the individual is exposed to the treatment then we could recover the causal effect by exposing the same individual to the treatment. For instance, if we want to study the brightness of a light when functioning, we will simply compare the value of the same light when the switch is on and off. If two individuals can be considered identical, then we could give the treatment to only one of the two individuals and compare the values obtained. Usually, we cannot make these assumptions in social sciences.

However, if instead of studying the treatment effect, we rather look at the average treatment effect, we would be able to overcome the confounding variable problem under the assumption of ignorability. The average treatment effect is $T = \mathbb{E}[Y^t] - \mathbb{E}[Y^c]$ but we only observe $T = \mathbb{E}[Y^t|P = t] - \mathbb{E}[Y^c|P = c]$ where P denotes the value of the treatment attributed to individuals. Then under the assumption that $Y^t, Y^c \perp P$, *i.e.* the treatment is attributed at random,[11] we finally have that $\mathbb{E}[Y^t|P = t] = \mathbb{E}[Y^t]$ which solves the problem.

2.2 Segregation and causality

The ignorability assumption is crucial for causal inference. However, in the case of residential segregation, we cannot use this approach for several reasons. First,

[10] Holland[28] discusses at length this problem.

[11] If there is other covariates in the model explaining the value of interest, the ignorability assumption is recast as $Y^t, Y^c \perp P|X$

residential segregation suffers from reverse causality. For instance, does an increase in crime cause more segregation or is it the reverse? It could be that individuals move if the level of criminality increases because they are risk averse.[12] Criminality might rise in more diverse areas because of the tensions associated with the interactions of the different groups.[13] Or it might be that segregation, by limiting the opportunities of the minority group, shift downward the opportunity cost for engaging in criminal activities, thus raising the criminality in the area.[14]

The second reason is the difficulty to find simultaneously an exogenous variation for all the potential determinants of segregation. In our context, it would mean finding simultaneously an exogenous income shock and an exogenous criminality shock (in the hypothesis that segregation is only caused by criminality and income) at the subplace level in South Africa in the period 1996-2011, which seems (almost) impossible. At least to the best of my knowledge, there are not such data.

However, we can disentangle the problem of causality by looking at the different potential determinants. But first, we must acknowledge the fact that causality is limited by the state of our knowledge at the moment of the analysis. Thus a variable might not be considered as a cause before some (robust) confirmatory evidences, or a cause might be revoked by (robust) dissenting evidences. For instances, smoking cigarettes was associated with lung cancers only at the beginning of the 20th century,[15] while hysteria was believed to be the consequence of women menstruations until 1980.[16]

For residential segregation, we actually know that a lot of variables are candidate for causing segregation.[17] We can actually divide the different theories into three groups labelled *Place stratification*, *Spatial assimilation*, and *Income sorting*. The *Place stratification* hypothesis posits that if individuals are segregated, this is due to prejudice and discrimination. Discrimination in the housing and mortgage lending markets, preferences for the racial mix, or legal segregation all fall in this category.[18] The *Spatial assimilation* hypothesis claims that segregation is the result of differences in socioeconomic characteristics and acculturation. Thus, differences in performances at school, unemployment rate, income levels, or exposure

[12]See O'Flaherty and Sethi[39].

[13]See O'Flaherty and Sethi[40] and Easterly and Levine[22] for the impact of diversity on the probability of conflict.

[14]See Becker[5], and O'Flaherty and Sethi[41].

[15]See Proctor[46] for a detailed historical review of the establishment of the causal relation between lung cancer and cigarette.

[16]See Tasca et al.[51] for a detailed review.

[17]See Charles[12] for a detailed review.

[18]See Schelling[48] for the impact of preferences on residential segregation. See Zhao et al.[56], Blanchard et al.[8], Ouazad[43], and Ouazad and Rancière[44] for discriminations in the housing and mortgage markets. See Cutler et al.[18] for the impact of legal segregation.

to crime are parts of this theory.[19] Finally, the *Income sorting* hypothesis come after Tiebout[53]. It postulates that individuals will relocate according to their willingness to contribute to a public good. Thus, if individuals share some common preferences about the level of public good with the members of their ethnic group, voting with the feet leads to segregation.[20] It is different from the *Place stratification* hypothesis in the sense that the decision to relocate is not associated with race directly. In this case, racial residential segregation is a side effect, while it is a pure direct effect related to your neighbours in the *Place stratification* hypothesis.

On the side of the consequences, segregation is believed to be responsible for unemployment through a higher distance to job, crime through a lower opportunity cost of engaging into criminal activities. These two effects are parts of a theory called *Spatial Mismatch*.[21] Segregation also leads to differences in human capital through the lower quality of public goods, higher rates of high school drop-outs, ghetto habits and the oppositional culture.[22] But we might believe also that preferences for the racial mixes depends on the number of contacts you have with the other group. Thus, if individuals do not have any contact because they are segregated, they might develop aversion to the other group through stereotypes. This hypothesis is known as the *Contact hypothesis*.[23]

All the potential causes and effects discussed before are summarized in Figure 1. In our analysis, we put aside the *Place stratification* hypothesis because it is extremely difficult to identify preferences and discriminatory behaviors. This is a caveat of our analysis. Usually preferences are either identified by value surveys, such as the World Value Survey or the Afrobarometer, or by revealing the preferences through structural estimations. In the case of value surveys, they are designed to be nationally representative but provides only small samples[24] and they are usually not available at a sufficiently disaggregated geographical level. We could in principle infer the most probable location of each individual with some matching techniques but matching around 3000 individuals to more than 21000 subplaces would most likely perform poorly. On the other hand, structural esti-

[19] See Bénabou[6] or Bayer et al.[4].

[20] See Bayer and McMillan[3].

[21] The lack of positive role model is also emphasized as responsible for these two effects (Wilson[55]). See Kain[30] and Smith and Zenou[50] for the first effect. See Glaeser et al.[26], Glaeser and Sacerdote[25], and Gould et al.[27] for the second effect.

[22] See Borjas[9][10], and Alesina et al.[1] for the lower quality of public goods. See Crane[16] and Cutler and Glaeser[17] for the higher rates of high school drop-outs. See Fordham and Ogbu[23], and Sáez-Martí and Zenou[47] for the oppositional culture

[23] In its initial formulation (Allport[2]), this theory postulates that if individuals increase the number of contacts they have with the other group, they will adopt a more positive attitude toward the other group. Thus, the reverse proposition about the lack of intergroup contacts.

[24] In the World Value Survey, you have 2935 individuals in 1995 while in the Afrobarometer there are 2399 individuals in 2013/2015, for South Africa.

mation relies heavily on the good specification of the underlying structural model which we do not know *a priori*. The main approach follows McFadden[36]. Preferences are estimated from a discrete location choice model with strong parametric assumptions on the distribution of the errors. When these errors are distributed as a Gumbel type 2 double exponential distribution, the structural model reduces to a standard logit model. However, using structural estimations in generalized decomposition might ruin the interpretation of the decomposition.[25] Finally, we might still have a sense of the importance of discrimination as the unexplained component of the decomposition is usually attributed to discriminatory behaviours. In our case, these discriminatory behaviours could be either occurring in the housing or mortgage lending markets or through preferences without any possibility to distinguish between the two.

We are thus left with explanations coming from the characteristics of the individuals (education, unemployment, income) and the context they are living in (crime, public goods). We will denote graphically the statement "A cause B" by an arrow going from A to B with a full node to stress that A is observed and the origin of the causal statement.[26] With this notation, the relations expressed previously are an intricated network where basically reverse causality occurs between all the variables (Figure 2). However, the reverse causality problem is deeply rooted in the contemporaneousness of the relations studied. But, by definition, a cause has to occur before its effect. Thus, contemporaneous associations cannot be claimed to be causal. The timing is crucial for causal inference. Then, we recast our intricated network by introducing lagged variables and removing contemporaneous links in Figure 3.

2.3 Identification of causal relationships: When "No smoke without fire" beats "Correlation is not causation"

The difficulty to identify causal relations appears when a third variable is implicated. There exists three different cases. First, the relation $A \rightarrow B$ occurred through the third variable C as a chain of mediation $A \rightarrow C \rightarrow B$. Then, both A and B cause C, $A \rightarrow C \leftarrow B$. Finally, both A and B are caused by C, $A \leftarrow C \rightarrow B$. These three situations generate correlations between A and B the same way as they would have been a direct relation between A and B. Then, the impossibility to distinguish with the sole correlation between a pure direct relation and the three cases where a third variable is inserted between justifies the "*Correlation is not causation*" mantra.

[25] See Fortin et al.[24] for details on this point.

[26] Textually, we will denote the statement that "A causes B" by a directed arrow between A and B, $A \rightarrow B$.

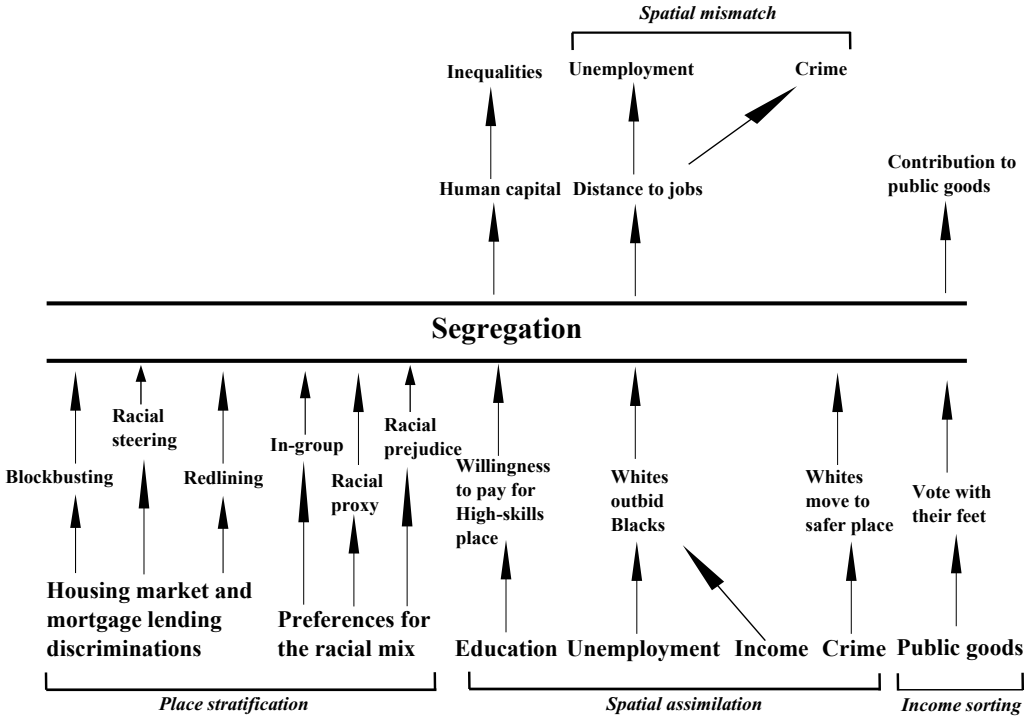


Figure 1: Synthesis of the causes and consequences of residential segregation

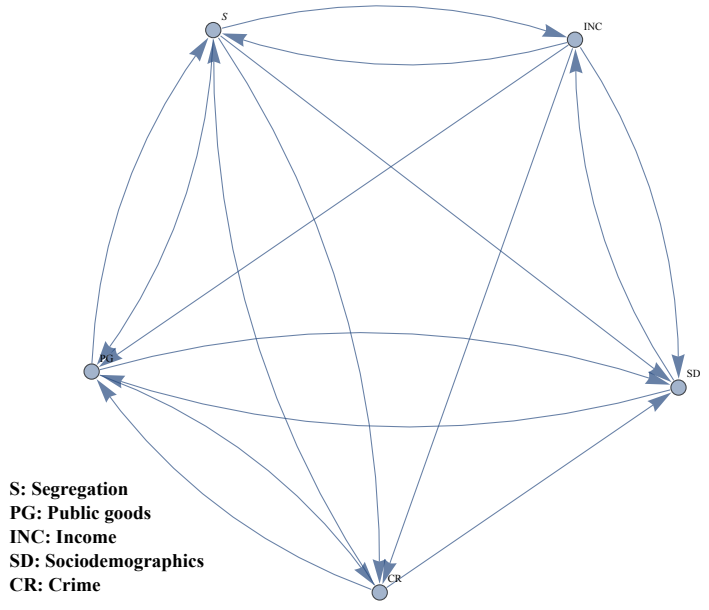


Figure 2: The reverse causality problem for residential segregation

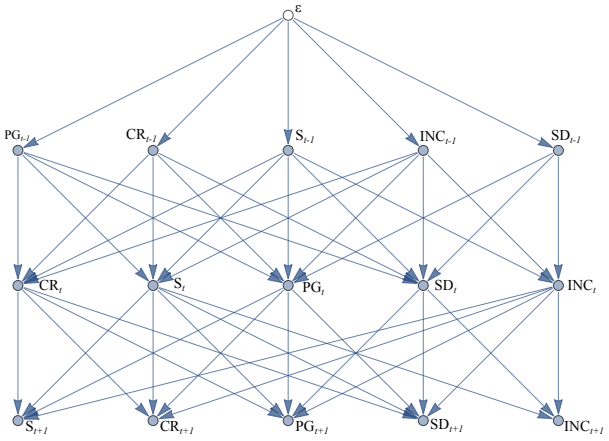


Figure 3: The directed acyclic graph for residential segregation

If $A \to C \to B$ (and $A \to B$), or $A \leftarrow C \to B$, the causal effect of A on B is directly identified by conditioning on C. However, in the last case $A \to C \leftarrow B$, conditioning on C would generate a spurious correlation between A and B. When we condition by a common consequence C, we introduce a selection bias relating the two independent causes, A and B, because knowing that C and one of the cause have occurred makes the other cause more or less likely to occur.[27] For instance, imagine you are interested in a risk factor analysis of a disease, say diabetes as the risk factor and cholecystitis the disease.[28] Then, knowing that the individual came to the hospital without diabetes increases the probability that he entered for other reasons related to cholecystitis, even though there is no relation at all in the general population between diabetes and cholecystitis.

Thus, in order to identify causal effects, we need to condition on variables that are effectively causes and not on consequences. This criterion is called the *Back-door criterion.*[29] It asserts that a causal effect is identified if all back-door paths are blocked. *A path* is any sequence of connections in any direction between two variables. For example, in Figure 3, the sequence $INC_t \leftarrow INC_{t-1} \to PG_t \leftarrow CR_{t-1} \to S_t \to S_{t+1}$ is the path connecting the levels of income at the t-th period and segregation at the next period. Moreover, *a back-door path* is a path where a directed edge points towards the initial variable. It is important to control for these back-door paths because they generate bias in the estimation of the true causal effect, in our example $INC_t \to S_{t+1}$. The previous example is a back-door

[27] Pearl[45] discusses this point at length.

[28] Cholecystitis is the inflammation of the gallbladder. It was believed to cause or aggravate diabetes (see Berkson[7]).

[29] See Pearl[45] and, Morgan and Winship[37] for more details on this point.

path for instance. We will further say that a path is *blocked* if we can find a set of control variables Z to condition on which are either the middle variable of a chain of mediation or the common cause of two consequences. If a control variable Z is a consequence of two independent causes, it will spoil the analysis due to the spurious correlation mentioned earlier. However, if we can additionally control for another variable Z' which is not a consequence of two causes, we will thus still block this back-door path. Then the *back-door criterion* identifies causal effects by controlling for all the other potential causes.

Our causal graph in Figure 3 has a lot of back-door paths potentially threatening the identification of causal effects. In fact, every path is a back-door path because a determinant is itself caused by another determinant at the previous period. Then, a directed link is pointing towards our starting variable, thus creating a back-door path by definition. But we can block all these back-door paths by conditioning by all the determinants at the previous period. Due to the definition of a cause, we have this nice autoregressive structure that allows only descendent links from the previous period to the actual one. Thus, we have only two types of back-door paths, those with common consequences and those without. For instance, between $CR_t \rightarrow S_{t+1}$ we can have the following back-door paths, without common consequences $CR_t \leftarrow CR_{t-1} \rightarrow S_t \rightarrow S_{t+1}$ and with $CR_t \leftarrow CR_{t-1} \rightarrow PG_t \leftarrow INC_{t-1} \rightarrow SD_t \rightarrow PG_{t+1} \leftarrow S_t \rightarrow S_{t+1}$. For the first back-door path, we can block it by conditioning either on S_t or CR_{t-1} as S_t is in the chain of mediation $CR_{t-1} \rightarrow S_t \rightarrow S_{t+1}$, and CR_{t-1} is the common cause of S_t and CR_t. For the second back-door path, there are two common consequences on which we should not condition, PG_t in $CR_{t-1} \rightarrow PG_t \leftarrow INC_{t-1}$ and PG_{t+1} in $SD_t \rightarrow PG_{t+1} \leftarrow S_t$. However, it can be blocked by conditioning on all the other variables, for instance S_t.

The dynamic structure is crucial to identify causal effects, and it is not specific to the study of segregation. Here, we have a general identification result as long as this kind of dynamic causal structure can be established.

Theorem 1. *If the relation between a cause and its determinants can be expressed as a dynamic autoregressive structure of order 1 with no contemporaneous correlations, then, by conditioning on all its determinants at the previous period, a causal effect is identified, and correlation is thus causation.*

The proof of this theorem comes from the observation that a path with a common consequence has always also either a chain of mediation or a common cause with a determinant at the previous period as a middle variable.[30] Otherwise, the

[30]Note the similarity between this theorem and the classical results on the omission of relevant regressors in the econometrics literature. The theorem is equivalent to saying that no relevant regressors are missing for the analysis of the model.

initial and final variables are not connected by a path because there is no contemporaneous correlations. So, to connect the two variables, there always is a chain of mediation or a common cause which will be between the common consequence and the final variable. Then, we always can condition on a determinant at the previous period to block back-door paths containing common consequences. Thus, by conditioning on all the determinants at the previous period, we block all back-door paths, thus proving the theorem. So, applied to our study of the determinants of segregation, if we can control for differences at the previous period in crime rates, segregation levels, access to public goods, sociodemographic characteristics, and income levels, then we will be able to estimate causal effects of each determinant.

3 Methodology

3.1 Segregation curves

Segregation curves are close parents of the Lorenz curve used for income inequalities. They both belong to the family of concentration curves.[31] They are constructed by sorting census tracts[32] by the proportion of Blacks.[33] Then, the cumulative distribution of Blacks and Whites are computed on this ranking.[34] Finally, each point of the segregation curve $S(i)$ until the i-th location, is constructed as:

$$S(\{F_B(i); F_W(i)\}) = \{(F_B(1); F_W(1)); (F_B(2); F_W(2))...(F_B(i); F_W(i))\} \quad (1)$$

Like Lorenz curves, they start at the origin (0;0) and end at the point (1;1). Segregation curves are important in that researchers can derive any segregation index measuring unevenness directly from them. For example, the Dissimilarity index is the maximum vertical distance between the segregation curve and the 45-degree line (representing perfect integration). The Gini index is the area between the segregation curve and the 45-degree line.[35]

Segregation curves are not exempt from drawbacks. They only account for an unequal repartitions of individuals across a particular set of demarcations. Thus

[31] See Hutchens[29].

[32] In our case, we will use subplaces. But segregation curves can be computed for any level of geography. So, we will use the more generic term "census tract" in the sense of an area demarcated for census for the rest of the discussion.

[33] It is also possible to sort census tracts by the proportion of Whites which will interchange axes.

[34] See Duncan and Duncan[21], and Hutchens[29].

[35] See Duncan and Duncan[21], and Massey and Denton[35] for more details on this point.

neglecting other dimensions of segregation such as interaction, concentration, centtralization, or clustering.[36] They only give a complete ranking of two configurations if one curve dominates at every point the other. If two curves cross, researchers have to make additional normative choices to define a complete ranking.[37] Finally, like indices of unevenness, segregation curves attribute a measure of segregation identical for each group.

However, we will use them as they are the closest statistical concept from the spatial distribution of the two groups. Moreover, comparing directly the spatial distribution of the two groups would be a much more difficult problem. Finally, by relying only on segregation indices, we would have missed a lot of the heterogeneity that might be involved in the formation of segregation. They are designed to provide general trends but we are also interested in which kind of individuals and locations that are integrating or segregating more.

3.2 General decomposition framework

As segregation curves are functionals of the distributions of individuals across census tracts, a counterfactual segregation curve can be defined by using a counterfactual distribution instead. Thus, it becomes possible to generate different types of counterfactual segregation curves (denoted by an exponent C), one with a counterfactual distribution of Whites, one with a counterfactual distribution of Blacks, and a last with both counterfactual distributions.

$$\begin{aligned} S^C(x) &= \{F_W^C(x), F_B(x)\} \\ S^C(x) &= \{F_W(x), F_B^C(x)\} \\ S^C(x) &= \{F_W^C(x), F_B^C(x)\} \end{aligned} \tag{2}$$

Thus, a counterfactual segregation effect will simply be the difference between the observed and counterfactual segregation curves. Thus, we can define different segregation effects depending on the type of counterfactual used.

$$\begin{aligned} S(x) - S^C(x) &= \{F_W(x), F_B(x)\} - \{F_W^C(x), F_B(x)\} \\ &= \{F_W(x), F_B(x)\} - \{F_W(x), F_B^C(x)\} \\ &= \{F_W(x), F_B(x)\} - \{F_W^C(x), F_B^C(x)\} \end{aligned} \tag{3}$$

We are particularly interested in the gap observed between segregation in 2001 and 2011 in South Africa. Thus, this particular gap can be decomposed in an Oaxaca-Blinder fashion as:

[36] See Massey and Denton[35].

[37] See Hutchens[29] for more details on this point.

$$S(i)_{2011} - S(i)_{2001} = S(i)_{2011} - S(\{F_W^C(i), F_B^C(i)\})_{2011} + S(\{F_W^C(i), F_B^C(i)\})_{2011} - S(i)_{2001} \quad (4)$$

where the term $S(i)_{2011} - S(\{F_W^C(i), F_B^C(i)\})_{2011}$ is the structure effect and $S(\{F_W^C(i), F_B^C(i)\})_{2011} - S(i)_{2001}$ is the composition effect.

3.3 Distribution regressions

Now the problem is to get an estimate of the counterfactual distributions. Following Chernozhukov et al.[15], we will model the conditional distribution as

$$F_{Y|X}(y|x) = P(y_i < y|x) = \Lambda(P(x)'\beta(y)) \quad \forall \ y \in Y \quad (5)$$

with $\Lambda(.)$ a known link function, $P(x)$ is a vector of transformation of X (which might be polynomials or B-splines), and $\beta(y)$ is an unknown vector of function-valued parameters. Thus, if $P(x)$ is a polynomial of order 1, and $\Lambda(.)$ is a logit link function, this model reduces to a standard logit model where the dependent variable is $\mathbb{1}(y_i < y)$ when y is fixed. Then by spanning the entire values of Y (or at least some sufficiently fine grid of values), we are able to estimate completely the conditional distribution and the impact of the covariates on the whole conditional distribution.

On the pros, this approach implies estimating well-known discrete choice models depending on the link function chosen. It can be a probit, logit, linear, log-log, or Gosset/Student link function. But the choice of the link function is crucial only if your set of covariates $P(X)$ is not rich enough.[38] We also do not need to make specific assumptions about the smoothness of the conditional distribution since we are estimating it potentially at each values of Y. However, on the cons, it is computationally heavy since it implies running a lot of regressions. There might also be some problems with crossing of predictions therefore leading to distributions that might be decreasing. However, you might overcome this problem with rearrangements.[39]

Alternatively, we could have used quantile regressions to estimate the conditional distribution. However, this would have implied to inverse back the quantile function into the conditional distribution. But this is not necessary since distribution regressions provide directly such estimates. Ultimately, we are interested in segregation curves which are functionals of the conditional distributions. Then we do not need to go through the estimation of the quantile function. Finally, as mentioned earlier, quantile regressions require Y to be sufficiently smooth while

[38] See Chernozhukov et al.[15] for more details on this point.

[39] See Chernozhukov et al.[13][14] for more details about rearrangements.

distribution regressions do not. Then, in our set up, there will probably some mass points for Black and White ghettos which may violate this smoothness requirement.

Finally, we have to make a choice of the link function. We will use a logit link function since the distribution of individuals across space is a discrete location choice problem. The logit function has been the dominant link function in this literature following McFadden.[36] We will then evaluate the counterfactual distributions at all values of our dependent variable, namely the number of individuals of a group in a location.

3.4 Detailed decomposition

Our primary interest in this paper is to quantify each determinant of residential segregation. Thus, the aggregate decomposition is not enough to our purpose. We need to get a detailed decomposition of the gap between segregation curves. In the case of the standard Oaxaca-Blinder decomposition for the mean, because everything is linear, the aggregate composition and structure effects are respectively the sum of the contribution of each factor:

$$(\bar{X}_W - \bar{X}_B)\beta_W = \sum_{k=1}^{K} (\bar{X}_{kW} - \bar{X}_{kB})\beta_W^k \tag{6}$$

and

$$\bar{X}_B(\beta_W - \beta_B) = \sum_{k=1}^{K} \bar{X}_{kB}(\beta_W^k - \beta_B^k) \tag{7}$$

However, in nonlinear settings, this is not as simple. First, contributions of a particular covariate will be obtained by computing the marginal impact obtained when the counterfactual is computed by replacing the distribution of this covariate by the one of the other group. Then, there is two possibilities, either the researcher pursues with this strategy for the other covariates without changing back the distribution of the first covariate, or he can change back the first covariate to its observed distribution and changes the distribution of the second covariate instead. The first strategy might be qualified as "without replacement", while the second is considered "with replacement". But without replacement, the decomposition will be path dependent, while without it will not add up to the aggregate effect.

With distribution regressions methods, it is possible to compute a detailed decomposition without replacement. The marginal contribution C^k of a particular covariate k will thus be the difference between the aggregate decomposition with and without this covariate for respectively the composition (c) and the structure (s) effects:

$$C_c^k = \Delta^c(K) - \Delta^c(K \setminus \{k\}) \tag{8}$$

and

$$C_s^k = \Delta^s(K) - \Delta^s(K \setminus \{k\}) \tag{9}$$

with $\Delta^s(K)$ the aggregate structure effect for the set of covariates K, while $K \setminus \{k\}$ is the same set without the covariate k.

3.5 Shapley Value

Shorrocks[49] proposes a solution to the problem of path dependence in detailed decomposition. By averaging all the marginal contributions along all the possible elimination paths, i.e. the possible order in which a particular covariate can be added to other covariates, you obtain a contribution of this covariate which is unique, adds up to the aggregate effect, and is path independent. Contributions are called Shapley Value because they are computed exactly the same way. Initially, the Shapley Value was conceived as the solution to divide fairly the reward of a cooperative game among players according to their efforts.

For example, imagine we have three covariates, $X = \{X_1, X_2, X_3\}$ and we want to know the contribution of X_3. Then, we have exactly 3! ways to order our covariates. Of course, the order will not matter when the model is evaluated with all the covariates. But it will change the marginal contributions. Let us say that we include first X_1, then X_2, and finally X_3. We will get the particular elimination path $\{\{X_1\}; \{X_1, X_2\}; \{X_1, X_2, X_3\}\}$. Then, if we denote the model we will evaluate for a set of covariates by the function $V(.)$, we will get three different evaluations of this model, namely $\{V(\{X_1\}); V(\{X_1, X_2\}); V(\{X_1, X_2, X_3\})\}$. But this particular elimination path only interests us for the marginal contribution of X_3 to the evaluation of the model which is in this case $V(\{X_1, X_2, X_3\}) - V(\{X_1, X_2\})$. Finally, we have to repeat this operation for all elimination paths and computing the arithmetic mean of all these marginal contributions.

While it is easily tractable to compute the Shapley Value for a small number of covariates such as 3, it becomes computationally cumbersome to do it for a large number of covariates. Already for 4 covariates, you have 24 elimination paths to compute, with 5 it is 120, and 720 for 6. Simple combinatorics gives you that for n covariates there are $n!$ elimination paths for each covariate. Note however that, as we are interested only in marginal contributions, there will be redundancy in some marginal contributions. But still the method is computationally heavy. Then researchers have to precisely define which contribution they are interested in.

One way to reduce the computational burden might be to group covariates describing related phenomena together and then adding or removing them by group. For instance, we are interested in the contribution of the housing market to segregation but we only have some characteristics of the dwellings such as the number of rooms, the type of construction (hut, house brick, shacks...), some of their equipments (light, water, toilets...). Then, all these variables together are going to tell us something about the housing market. Thus, it does not make sense to evaluate their contribution individually. We will use this alternative in our analysis.

Thus, our final specification is:

$$\begin{aligned} Segregation_{ij}(t) = \alpha &+ Segregation_i(t-1) \times \beta_1 + Demographics_{ij}(t-1) \times \beta_2 \\ &+ Income_{ij}(t-1) \times \beta_3 + HousingCharacteristics_{ij}(t-1) \times \beta_4 \\ &+ PublicGoods_{ij}(t-1) \times \beta_5 + \epsilon_{ij} \end{aligned} \tag{10}$$

with $\beta_1, \beta_2, \beta_3, \beta_4, \beta_5$ column vectors of coefficients associated to a particular regressor in the corresponding subsets of regressors. $Segregation_i(t-1)$ is a row vector composed of two regressors, the cumulative distributions of Whites and Blacks of the subplace i at the previous period. $Demographics_{ij}(t-1)$ is a row vector composed of six regressors, the mean age, the marriage to divorce ratio,[40] the sex ratio, the average number of years of schooling, the share of individuals speaking "White" languages,[41], and the unemployment rate. All these regressors are computed by subplace i and population group j at the previous period. $Income_{ij}(t-1)$ is the mean income of group j individuals living in subplace i at the previous period. $HousingCharacteristics_{ij}(t-1)$ is a row vector composed of five regressors, the mean number of rooms in the dwelling, the share of houses in brick, the share of informal dwellings, the share of owners, and the rural to urban ratio. $PublicGoods_{ij}(t-1)$ is a row vector composed of two regressors, the share of households not having access to a refuse disposal, and the share of households not having access to public water. Finally, the dependent variable $Segregation_{ij}(t)$ is the cumulative distribution of group j individuals for subplace i a the current period.

3.6 Practical issues

We will face some practical issues with the estimation of the segregation curves and their inference. First, observed and counterfactual segregation curves might

[40] We aggregate all types of marriage (civil monogamous union, polygamous, and traditional).

[41] There are only two "White" languages in South Africa, English and Afrikaaner.

not be comparable directly since they might not be evaluated at the same values of the distribution of Blacks. Thus, we need to create a common grid and interpolate values on this grid for the observed and counterfactual segregation curves. Our common grid will be the all the percentile of the conditional distribution of Blacks.

For inference, we do not know precisely the sampling distribution of this functional. Then, we will bootstrap the statistic and make inference from the sample of the bootstrapped statistics. As it seems that it is far from normally distributed,[42] we will report the 2.5% and the 97.5% percentiles of the bootstrap distribution for the standard 95% confidence bands.

4 Data description

Our dataset comes from the Community Profiles of the South African Censuses of 1996, 2001, and 2011. They correspond to the 100% count of each census aggregated to different geographical levels to preserve the anonymity of the respondents. These geographical levels range from the enumeration area to the province, except in 2001 where it ranges from the subplace to the province.

When measuring segregation, we are very concerned about the geographic level and its stability over time. The geographic level has to be fine enough to represent something meaningful in terms of individuals' reality, but it should not be too fine too. If the geographic level is to aggregated everyone is integrated while everyone is segregated if the grid is too fine. Thus, we will concentrate on the subplace level because it is the finest level available throughout all the censuses. But primarily because it corresponds to some local housing market which individuals are using when they refer to where they live.

However, the subplace level does not remain stable over the different censuses. But we are going to overcome this problem by "freezing the history".[43] The idea is to identify all the boundaries changes with respect to the 2001 subplaces by overlaying the other boundaries. Thus, we can compute the share of each area that was/will be in each subplaces in 2001. Then, we reallocate individuals with respect to these weights and reconstitute some adjusted version of the subplace level in 1996 and 2011 to the 2001 subplace boundaries. Implicitly, this method assumes that individuals are uniformly distributed across space in their respective area which might not be the case in practice. However, this assumption becomes less and less heroic as the grid used to adjust is finer because individuals become more and more homogeneous. Thus, we use the enumeration area levels in 1996 and 2001 which represents in average 100 individuals to aggregate them back to the 2001 subplace level.

[42] The densities of the bootstrap sample can be obtained on request to the author.

[43] See Norman et al.[38] for more details about the creation of consistent boundaries.

Note also that even when a boundary is supposed to be the same, the overlap between the different boundaries system is not perfect and you might have some gap between two boundaries that are supposed to be the same. The magnitude of the gap is several meters. Thus, even if it causes some error it is first systematic since it looks like a translation of one boundary system several meters away. Second, and most importantly, it is completely uncorrelated with the data attributed to each subplace. So, in the end, this error-in-variable problem will not cause problems of endogeneity, and we will use our covariates as proxy of the covariates that should have been in the model.

Our sample is thus constituted of all subplaces in the 1996, 2001, and 2011. We eliminate particular subplaces such as national parks or some industrial sites, comprising very few individuals.

5 Discussion

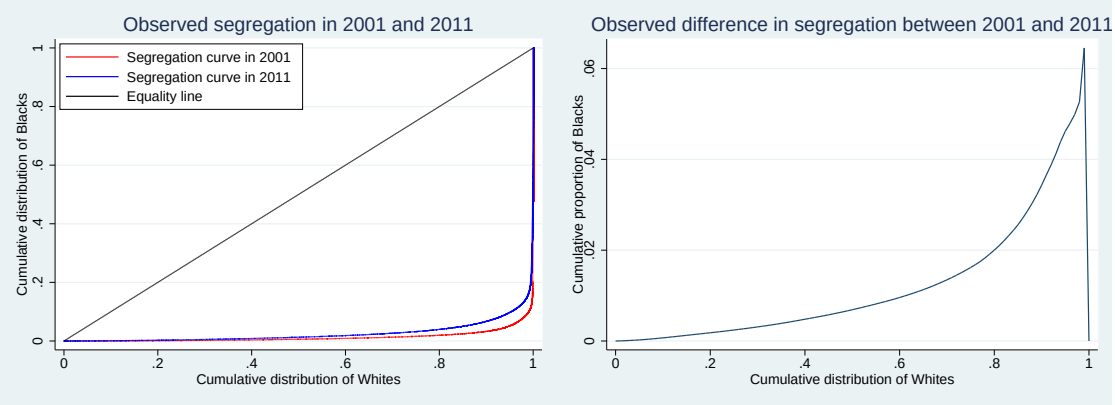
Before analyzing the results of the decomposition, let us look at what we are currently decomposing first. Segregation curves observed in 2001 and 2011 are plotted in Figure 4a while the observed gap between these two curves is depicted in Figure 4b. The gap seems pretty small at first because Whites lives with only a bit more than 6% Blacks more. In 10 years, we could have expected a more significant decline.[44] However, if we compare to the decline of segregation in the United States, Cutler et al.[18] find a decrease of between roughly 0.07 and 0.1 points of the dissimilarity index between 1970 and 1980, indicating that between 7 and 10% of individuals (Blacks or Whites) have moved to more integrated locations. Thus, the effect seems pretty close from what the United States have experienced in a similar context.[45] The White resistance to a more active implementation of integration policies might be an explanation of this close but lower effect in South Africa.[46]

However, despite an encouraging aggregate effort of integration, most of the integration occurs in few subplaces. More than two thirds of the increase occur in the 20% subplaces with the largest Black proportions. We can also note that the proportion of (almost) completely homogeneous subplaces has diminished in 2011. This can be seen from the decrease in the proportion of Blacks and Whites living with (almost) no members of the other group. In 2001, roughly 80% of Blacks

[44]Note that an increase of the gap means a reduction of segregation as the gap is positive because the segregation curve in 2011 is above (stochastically dominates) the 2001 curve and thus closer to the equality line.

[45]Remember that in 2011, we are 17 years after the end of the Apartheid with the election of Nelson Mandela in 1994. In 1980, the United States were 16 years after the Civil Rights Act of 1964 that put an end to the Jim Crow era.

[46]See Dixon et al.[20] for more details about the principle-implementation gap in South Africa.



(a) (b)

Figure 4: Observed segregation and difference between 2001 and 2011

lived in homogeneous subplaces, while this figure drops to roughly 50% in 2011. For Whites, the proportion goes from roughly 25% in 2001 to roughly 15% in 2011. These two facts seem to be in phase with the transformation of the South African society with the emergence of a rich Black elite and a Black middle class, and the improvement of living conditions of millions of (mainly Black) South Africans.

The results of the decomposition of the segregation effect between 2001 and 2011 are displayed in Figures 5 and 6. Remember that our counterfactual is the segregation levels that would have prevailed in 2001 if individuals had the characteristics of the South African population in 2011. Thus, the segregation effect can be interpreted as the variation in the segregation curve that would have prevailed due to the change in a particular characteristic from its level in 2001 to its level in 2011. Thus, when we look at the composition effect, of the five covariates only the housing market has a negative impact on the segregation gap. The four others, namely past segregation, income, sociodemographics, and public goods all have a positive impact. But all do not have an effect at the same place in the distribution. Thus, past segregation is significant from roughly 45%, the housing market matters only between roughly 45% and 75%, income counts roughly from 25%, while demographics affects segregation only in the last quartile, and public goods are relevant almost from the beginning at the 15th quantile. No matter the characteristic studied, there is always a fraction of the White population which resists integration. It represents at least 15% of the White population which is quite large.

In terms of magnitude, if we compare the 60th quantile, the housing market is the strongest determinant of segregation as around 60% of the White population would have experienced a decrease by 20 percentage points of the Black population. Thus, we can deduce, that in this subplaces, the housing markets for Whites

and Blacks are really different and they are even more different in 2011 than in 2001. This can be due to the formation of Black ghettos of the middle class or simply because there are a lot of discriminatory practices such as redlining, or blockbusting. Income on the other hand is quite small relative to other sources. It is close to an increase of 5 percentage point at the 60th quantile. However, it changes its course near the 75th quantile and becomes one of the most important source of integration in the last quantile. This is really interesting because individuals around the 60th quantile were not really richer in 2011 from their counterpart in 2001. The similitude is even reinforced by the absence of sociodemographic difference in one decade for these individuals. Thus, they are not that different from their counterpart in 2001 but they do experience a more segregated housing market in 2011. Then, the explanation does not seem to be directly related to economic constraints or a deep social change which suggests that discrimination might be at play rather than the formation of rich Black ghettos. Access to public goods however has a larger impact on segregation. At the 60th quantile, it would have increased the proportion of Blacks by approximately 10 percentage points, twice as large as the effect of income. This might be explained by the huge investments in access to basic public services since Nelson Mandela's mandate.[47] Finally, past segregation also has a similar effect as public goods, around 10 percentage points of increase at the 60th quantile. This seems to indicate a weaker persistence of segregation through time. Counterfactuals are constructed by restricting the conditional distribution to the common support of interest. In this case, the conditional distributions of Blacks and Whites are computed by comparing subplaces in 2011 to those with similar levels of past segregation in 2001. Thus, with comparable levels of past segregation, Whites in 2011 still experience a higher share of Blacks. Hence, it seems more easy to break away from a legacy of segregation in 2011 than in 2001 which is particularly a good news for South Africans.

When we turn to the structure effect, we observe always the reverse of the composition effect. For each source of segregation, when the composition effect is positive, the structure effect is negative and vice versa. Though it looks like it is symmetric, it is not. There is always a gap between each structure and composition effect. Structure effects also are significant for segregation generally at the same quantiles as composition effects. Thus, past segregation, income, sociodemographics, and public goods have a negative impact on segregation, while the housing market has a positive impact on the gap. This indicates that in 2011 all sources except the housing market have a lower marginal impact on segregation than in 2001, therefore contributing to increasing segregation.

Finally, if we look at the relative contributions (Figure 7) of each source of seg-

[47]See Thomson[52] for more details about the RDP and GEAR programmes.

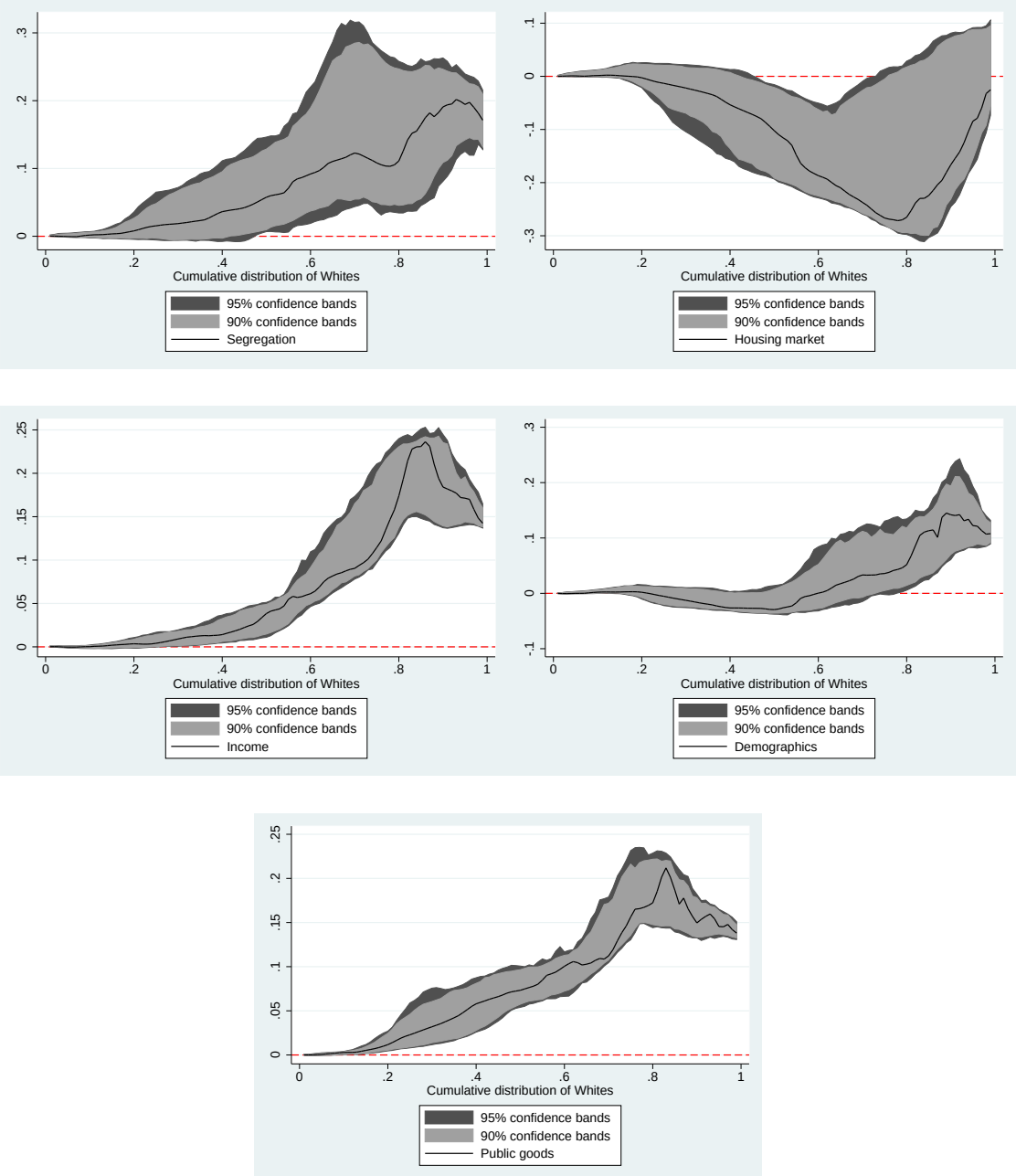


Figure 5: Composition effects of the detailed decomposition

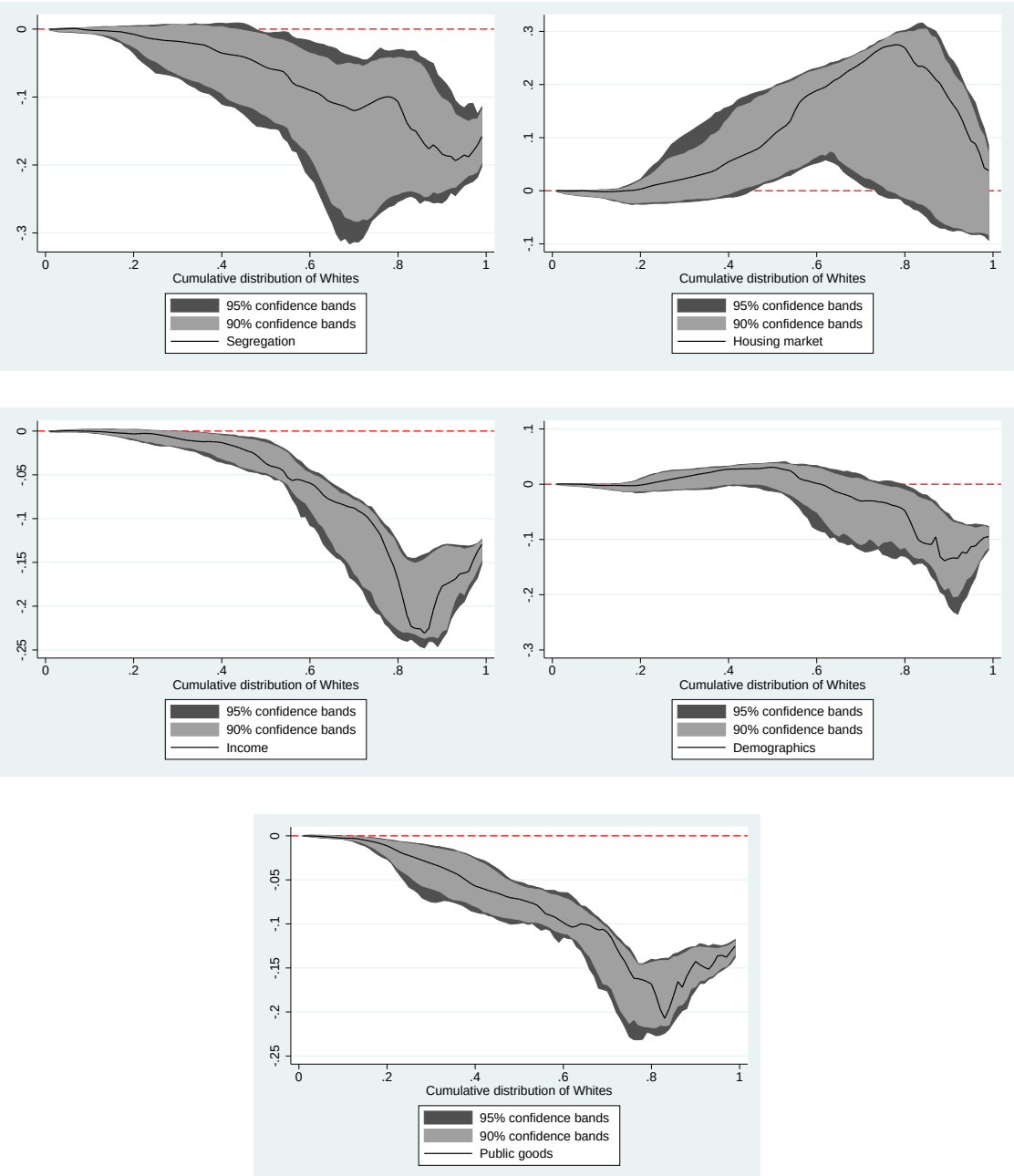


Figure 6: Structure effects of the detailed decomposition

regation,[48] it appears that sociodemographic characteristics and income explain very little of the segregation gap. They respectively are responsible for 11.3% and 13.8% in average of the segregation gap. Compared to the results obtained by Bayer et al.[4] for the United States,[49] sociodemographics have much less explanatory power while income is a more important factor, in South Africa. Collectively, sociodemographics accounts for 20.1% of the Black exposure rate and 28.5% for the Whites once the sole effect of income has been removed.[50]. For income, they find that it explains only 10.2% (14.2% with wealth and social benefits) for Blacks and 3.9% (6.6% with wealth and social benefits) for Whites.

Other sources of segregation cause much more variations in segregation. In average, they are all almost twice as large as income or sociodemographics as past segregation accounts for 20.5%, public goods represent 26.9%, and housing explains 27.4%. But there is a lot of disparities between each source across the cumulative distribution of Whites. Thus, past segregation is almost constant, but sociodemographics and income are very moderate until the 80th quantile. Housing starts to matter at the 20th quantile, peaks at the 60th quantile, then declines to be a small contributor at the end of the distribution. Public goods impacts segregation the stronger at the beginning of the distribution and declines continuously to still accounts for more than 20% at the end of the distribution.

In terms of public policies, our message is radically different from the one from Bayer et al.[4]. While they emphasize the importance in reducing differences in sociodemographic characteristics, we stress primarily the crucial role of the provision of basic public services. It has an impact relatively strong for almost all the distribution. We acknowledge that reducing differences in sociodemographics becomes an important focus for the subplaces mainly inhabited by Blacks in the last 20 quantiles. Fighting the persistence of ghetto through relocation policies like the Moving To Opportunity experiment is also important as it influences significantly segregation in more than half the South African subplaces. Finally, in subplaces experiencing already some integration (between the 60th and 80th quantiles), implementing anti-discriminatory measures would probably help a lot. However, a policy mix acting on all these sources would probably interact positively to promote an even faster integration.

[48] We focus on the relative contributions of each source of segregation to the composition effect as it will be very similar for the structure effect.

[49] Remember that Bayer et al.[4] decompose exposure rates (representing the interaction dimension) which are specific for each racial group while we decompose segregation curves (representing the evenness dimension) which are common for each group (bilaterally). This might not be exactly comparable but we still do the exercise since they are the only paper to decompose determinants of segregation to the best of our knowledge.

[50] The share of segregation explained by sociodemographics drops to 16.1% and 25.8% when they control for wealth and social benefits effects. This is closer from what we have but still higher, especially for Whites.

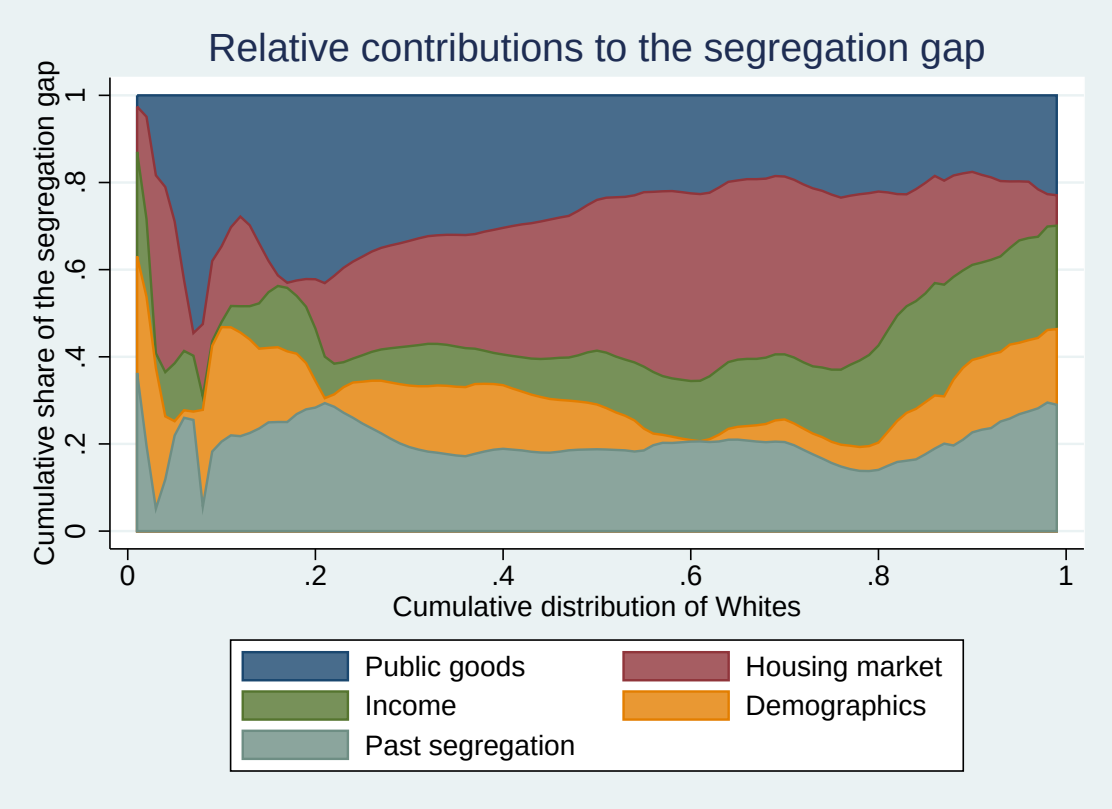


Figure 7: Relative contributions

Finally, our paper is far from to be perfect. Proposing a structural approach to identify directly the effect of preferences and getting reliable data on crime at a sufficiently disaggregated level would probably help completing the general picture, for example. While we leave these tasks for future research, we have proposed a generalized detailed decomposition for the sources of segregation within a strong causal framework. We believe this lays some ground for public policies and a deeper understanding of what cause segregation.

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