FULL ARTICLE

Infant mortality in Turkey: Causes and effects in a regional context

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Abstract

This study attempts to identify the causal and/or direct effects of sociocultural determinants of infant mortality in Turkey within a regional context using causal graph analysis and global and local spatial models. The conceptual framework, combined with the data, shows that fertility and consanguinity have direct effects on infant mortality rates, and that female illiteracy, as a proxy for maternal education, is the main cause of rising infant mortality even in the presence of latent confounding. The surface of estimates further shows that the local effects of female illiteracy and consanguinity are non-stationary across space, calling for location-specific policies.

KEYWORDS

causal graph, female education, infant mortality, spatial dependence, spatial heterogeneity

JEL CLASSIFICATION

I11; I15; R11

1 │ INTRODUCTION

Infant mortality is a widely used measure of population health and quality of healthcare and a powerful indicator of socioeconomic development in general and mother and child healthcare in particular. The simplicity in its measurement enables inter-regional comparisons to track geographic disparities in infant mortality, identify regions of priority, assess needs in mother and child healthcare, and evaluate the impact of public health policies.

Studies on infant and child mortality in Turkey date back to the 1980s, thanks to the quinquennial national population surveys conducted by *Hacettepe University, Institute of Population Studies.* First, although exceptionally high

infant mortality rates and persistent regional variations in Turkey are known, the implications of spatial patterns remain largely unknown due to the unavailability of fine-scale georeferenced data.[1] Only a scant number of studies in Turkey visualized the spatial patterns in the assessment of local socioeconomic and environmental factors and the resulting disparities in infant mortality rates using geographic information system (GIS) analysis (Arslan, Cepni, & Etiler, 2013; Babaoğlu, Işsever, & Hapçıoğlu, 2016) and other spatial tools (Karahasan & Bilgel, 2019). While the GIS analysis of Arslan et al. (2013) suggests a link between poor environmental conditions and perinatal mortality in the heavily industrialized province of Kocaeli in the Marmara region, the province-level spatial analysis of Karahasan and Bilgel (2019) suggests an increasing geographic polarization in infant and child mortality rates and a non-robust link between healthcare access or utilization, and child mortality in the already-developed regions of Turkey.

Second, the *modus operandi* of socioeconomic and sociocultural determinants remains largely unknown due to the rare use of the conceptual frameworks of Caldwell (1979) and Mosley and Chen (1984), for example, for statistical modeling. This begs the question of how the links between socioeconomic or sociocultural factors and child mortality work (Masuy-Stroobant, 2002). Empirical studies are likely to encounter identification problems and hidden biases in the absence of a guidance on which covariates one should control for and which covariates one should not.

This study aims to identify the causal effects of several socioeconomic and sociocultural determinants of infant mortality in Turkey within a regional context. For this purpose, this paper invokes a causal graph analysis and uses the global and local cross-sectional spatial models using provincial and regional data for the 2009–2017 period. The conceptual framework, combined with the data, shows that female illiteracy, as a proxy for maternal education, is the major cause of rising infant mortality in Turkey. The global results indicate that female illiteracy exerts a direct effect on infant mortality along with fertility and consanguineous unions. Depending on the specification, 33–52% of the total effect of female illiteracy on infant mortality is the direct effect, whereas the remaining is mediated through birth and fertility, healthcare utilization, and cultural factors. Using an electoral measure as an exogenous source of variation, the causal effect of female illiteracy is identified under the possibility that it might be endogenous to mortality. The robustness checks show that the impact of female illiteracy on infant mortality is resistant to alternative functional form and to the choice of the spatial weight matrix. The local results show that all spatially varying effects of female illiteracy are non-stationary across space and monotonically positive in the range of 0.586 and 0.778 deaths per 1000 live birth of newborns, and that the largest impact is concentrated in the adjacent provinces of Marmara and the Western Black Sea regions. On the contrary, all spatially varying effects of consanguinity are also monotonically positive in the range of 0.117 and 0.792 deaths per 1000 live birth of newborns, and the largest impact is concentrated in the adjacent provinces of the Mediterranean and the Southeastern Anatolia regions.

Section 2 develops a conceptual framework using directed acyclic graphs (**DAG**) that serve as the first part of the building blocks of the identification strategy. Specifically, Section 2.1 constructs a set of causal graphs and searches for admissible sets to identify the total and direct effects; Section 2.2 introduces a signed **DAG** to assess the sign of monotonic effects and covariances, and Section 2.3 graphically assesses latent exposure-outcome confounding in an instrumental variables (**IV**) setup. Section 3 complements the conceptual framework by identifying patterns of spatial dependence through spatial error models (**SEM**) and patterns of spatial heterogeneity through a multiscale geographically weighted regression (**MGWR**) model and by invoking an **IV** strategy to account for the possibility of latent confounding; Section 4 reports the results and performs several robustness checks, and Section 5 assesses the limitations of the analysis and suggests avenues for future research.

2 | CONCEPTUAL FRAMEWORK

Appropriate statistical modeling requires a conceptual framework that identifies the causal relations and the interactions among the sociocultural factors, known to affect infant mortality. For this purpose, we invoke a **DAG** analysis

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[1] See Akşit and Akşit (1989) for a review of the research on (aspatial) determinants of infant and child mortality.

(Pearl, 1995, 2000). It is a completely nonparametric framework that shows the importance of specific types of endogenous variables of a system of causal relations. A **DAG** consists of a set of nodes and directed edges where causation is unidirectional (i.e., no reverse causation). The objective is to represent explicitly all causes (including unobserved/unmeasured) of the outcome of interest and to help choose the covariates to be included in the statistical model to minimize bias.

2.1 | Identifying causal estimands using causal graphs

Consider the baseline causal graph in Figure 1a, in which the outcome variable is infant mortality (*Y*). The exposure variable is maternal education (*E*), and the gray filling denotes the latent variables of the model.[2] As the substantive knowledge of the causes of infant mortality is formalized, these links are operationalized on the causal graph by referencing to each directed edge.

The roots of the problem can be traced back to linguistic and cultural barriers. While the linguistic barriers (*B*) refer to the inability to integrate to the social environment that manifests itself by a restriction of diffusion of health knowledge, the cultural factors refer to practices implied by traditional-patriarchal family structures (*P*) (Smits & Gündüz-Hoşgör, 2003). Especially in the Kurdish-populated eastern and southeastern regions of Turkey, an over-whelming proportion of Kurdish women tend to speak their mother tongue (Smits & Gündüz-Hoşgör, 2003), have not completed primary education, and thus have not learned Turkish at all (Yüceşahin & Özgür, 2008) (this link is shown in Figure 1a by the directed edge $B \rightarrow E$). The reluctance to speak the official language either altogether restricts access to maternal care utilization (*M*) or results in non-compliance with the written and oral directions of healthcare professionals when care is sought ($B \rightarrow M$). A particular consequence of this non-compliance is the lack of contraception use that leads to high fertility rates (*F*) in these regions (Şahin & Şahin, 2003; Yüceşahin & Özgür, 2008) ($M \rightarrow F$). Higher fertility implies shorter birth intervals and a higher likelihood of postnatal complications (congenital anomalies, preterm delivery, hypertensive disorders, and diabetes), and thus higher infant mortality (Keskinoğlu et al., 2007) ($F \rightarrow Y$).

On the contrary, the traditional patriarchal family structure (*P*) enforces consanguineous marriages (*C*), especially among the Kurdish and Arap populations to retain family ties and familial property holdings ($P \rightarrow C$) and early marriages (*A*) to alleviate financial burden of poor families (Keskinoğlu et al., 2007) ($P \rightarrow A$), prevents women to engage in legal paid work in non-agricultural sectors (*L*) ($P \rightarrow L$), and regards them as lower status whose duty is to frequently give birth as soon as they begin to experience sexual feelings (Ertem, Saka, Ceylan, Değer, & Çiftçi, 2008) ($P \rightarrow F$). Although 78% of the births in Turkey occur at a health facility, this rate is much lower in the eastern regions and rural areas, possibly due to traditional home delivery by a non-professional midwife (Çelik & Hotchkiss, 2000; Erdem, 2003; Kültürsay, 2011) ($P \rightarrow M$). This practice is likely to increase mortality rates especially in remote rural areas due to the failure to tackle potential perinatal complications ($M \rightarrow Y$).

Ample evidence shows that education is an important determinant of consanguineous unions (Hussain & Bittles, 2000; Abdalla & Zaher, 2013) ($E \rightarrow C$), maternal care utilization (Çelik & Hotchkiss, 2000) ($E \rightarrow M$), adolescent marriages (Ertem et al., 2008) ($E \rightarrow A$), and fertility (Işık & Pınarcıoğlu, 2006; Yüceşahin & Özgür, 2008) ($E \rightarrow F$). Past demographic research on the determinants of child health has also shown that maternal education is the precursor to decrease child mortality rates (Caldwell, 1979; Caldwell & McDonald, 1982; Cochrane, Leslie, & O'Hara, 1982) ($E \rightarrow Y$). Low levels of educational attainment impede women's participation in non-agricultural jobs ($E \rightarrow L$). Absence of paid-work prospects forces women to have frequent pregnancies and leads to higher fertility rates ($L \rightarrow F$).

High prevalence of infant mortality is associated with high rates of consanguineous marriages in the eastern and southeastern regions of Turkey. Consanguinity is known to increase the incidence of congenital diseases resulting
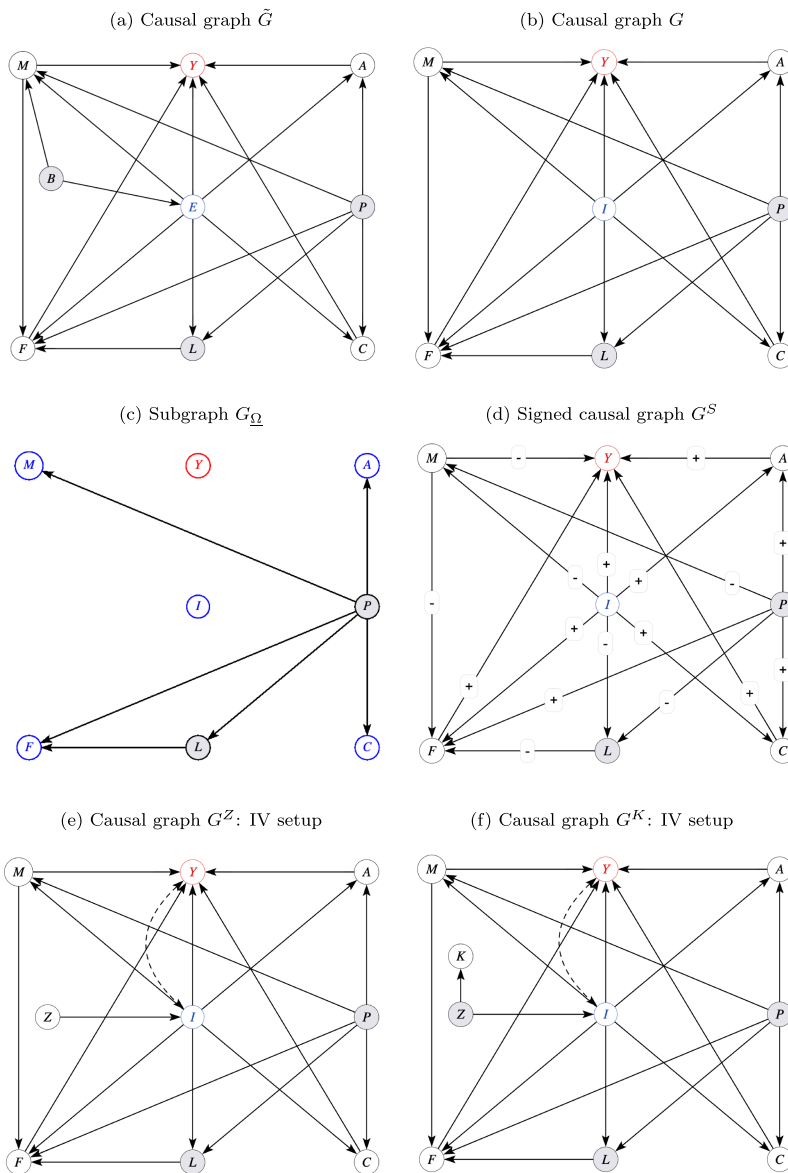
FIGURE 1 Conceptual framework

Note: *M*: maternal care utilization, *B*: linguistic barriers (latent), *F*: age-specific fertility , *L*: female labor force participation (latent), *C*: consanguinity, *P*: patriarchal structure (latent), *A*: adolescent pregnancy, *E*: maternal education (exposure, unmeasured), *I*: female illiteracy (exposure, measured), *Y*: infant or neonatal mortality (outcome), *K*: HDP vote share (measured proxy instrument), *Z*: ethnicity, income inequality (unmeasured causal instruments).

from autosomal recessive pattern of inheritance and manifests itself as genetic disorders (e.g., down syndrome, diabetes mellitus, and heart defects) and infant deaths (Bittles & Black, 2010; Hamamy, 2012; Yüksel, Kutlubay, Karaoğlu, & Yoloğlu, 2009) ($C \rightarrow Y$). Albeit still largely debated, a significant positive association between consanguinity and infant mortality has been reported (Bittles & Black, 2010; Tunçbilek & Koç, 1994). Consanguineous marriages, typically a union of first or second cousins, occur in about one-fifth of all marriages in Turkey (Kaplan et al., 2016; Koç, 2008). In the eastern regions, this figure amounts to a whopping 34.4% in Van (Akbayram et al., 2008), 30.6% in Kahramanmaraş (Dönbak, 2004), and 28.4% in Malatya (Yüksel et al., 2009).[3]

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[3]Although there is a strong association between consanguinity (*C*), adolescent pregnancy (*A*), and fertility (*F*), higher fertility rates and adolescent pregnancies among consanguineous unions are the result of the traditional patriarchal family structure that acts as a common cause. Therefore, causal paths between consanguinity and adolescent pregnancy (age-specific fertility) and adolescent pregnancy and age-specific fertility are not drawn in Figure 1.

Mother's age at birth is another important cause of high infant mortality rates for it represents the onset of a woman's exposure to the risk of pregnancy (Çelik & Hotchkiss, 2000; Ertem et al., 2008). Pregnancies that occur before the age of 20 are associated with increasing perinatal complications (Bükülmez & Deren, 2000; Özalp, Tanır, Şener, Yazan, & Keskin, 2003) ($A \rightarrow Y$).

Figure 1a displays the associated baseline causal graph $\tilde{G}$. Provided that $\tilde{G}$ is an accurate representation, the first objective is to identify the causal effect of E on Y. Although the identification of the association between E and Y is a rung-one concept (seeing) in the ladder of causation and can be achieved by calculating the expected value of Y given an observation of $E = e$, or $\Pr(Y|E = e)$, the identification of the causal effect of E on Y requires a manipulation or intervention on E, which is a rung-two concept (doing) (Pearl & Mackenzie, 2018). The latter task inquires about the expected value of Y if we make $E = e$ and requires the use of the do-operator that can be written as $\Pr(Y|do(E = e))$. Our aim is to find a strategy that will enable us to remove the do-expression, so that $\Pr(Y|do(E = e)) = \Pr(Y|E = e)$. To obtain $\Pr(Y|do(E = e))$, the causal graph $\tilde{G}$ is modified by performing a surgery (i.e., removing all arrows going into E) using the do-calculus. $\Pr(Y|do(E = e))$ can be inferred in the post-surgery model from the joint distribution of all observables in the pre-surgery model by conditioning on a set of variables or by statistical adjustment (Pearl & Mackenzie, 2018).

Figure 1a shows two backdoor paths from E to Y, that is, any path from E to Y that starts with an arrow pointing to E. These backdoor paths are $E \leftarrow B \rightarrow M \rightarrow F \rightarrow Y$ and $E \leftarrow B \rightarrow M \rightarrow Y$, and they are blocked if and only if B, which is a confounder in the relationship between E and M, is conditioned on. These backdoor paths cannot be blocked by conditioning on B because it is unobservable. Therefore, there is no admissible set of nodes; the implied conditional independences are untestable due to unobservables, and the causal effect of E on Y, either direct or indirect, is not identifiable from $\tilde{G}$.

Even if B had been observable, the causal effect of E on Y would have remained unidentifiable because of a lack of true measure of maternal education (E). To mitigate this problem, we search for a proxy measure that will enable us to replace both B and E in the causal graph $\tilde{G}$. The linguistic barriers (B) imply a lack of knowledge of the Turkish language to the extent of proper expression and understanding of written and verbal communication in maternal and reproductive health. For operational purposes, we shall find a measured factor that will capture maternal education (E) and linguistic barrier (B). A plausible candidate is the female illiteracy (I). Female illiteracy rate is defined as the ratio of the number of illiterate women within a particular age cohort to the total female population in that cohort. The term "illiteracy" also refers to the illiteracy in the official language, Turkish, and the lack of education. Albeit imperfect, it acts as a good proxy for both maternal education (E) and linguistic barrier (B).

The modified causal graph is shown in Figure 1b, where maternal education E and the linguistic barriers B are now captured by female illiteracy I. Notice that all paths connecting I to Y other than the edge $I \rightarrow Y$ in causal graph G are mediated through M, F, C, A, and L. The causal graph G shows that there are no biasing paths and the causal effect of I on Y is identifiable. Two types of causal effects are shown in Figure 1b. The first is the average direct effect (ADE) of I on Y, which is captured by the directed edge $I \rightarrow Y$. The ADE asks the question of what difference in infant mortality would result if one changes the level of female illiteracy while holding the levels of adolescent pregnancy, fertility, consanguinity, and maternal care utilization constant. The second is the indirect effect, which is evaluated along each path other than $I \rightarrow Y$ by taking the product of all the path coefficients along that pathway. The indirect effect answers the question of what change would occur to infant mortality if one changes, say, consanguinity while holding female illiteracy level and other causes constant.

Given the pivotal role of female illiteracy, one may be further interested in the average total effect (ATE) of I on Y. In a linear model that does not involve interactions (moderation), the ATE of I on Y is the sum of the direct and the indirect effect and can be evaluated by taking the sum of products of all the directed paths that emanate from I and reach Y. Since there are no backdoor paths from I to Y in Figure 1b, the admissible set of nodes is an empty set, indicating that no statistical adjustment is necessary to estimate the ATE of I on Y. The ATE estimand of I on Y can be written as follows:

$$\Pr(Y|do(I)) = \Pr(Y|I). \tag{1}$$

To identify the ADE of I on Y, all paths that contain a chain should be blocked. First, conditioning on A will block the path $I \to A \to Y$, but will also unblock the paths $I \to A \leftarrow P \to C \to Y$, $I \to A \leftarrow P \to L \to F \to Y$, $I \to A \leftarrow P \to F \to Y$, and $I \to A \leftarrow P \to M \to Y$ because A is a collider (a node with two incoming arrows) on all four paths and conditioning on colliders opens the path. Since P and L are unobservables that cannot be conditioned on, all the remaining variables, M, F, C, and A, should be blocked by conditioning on them to rectify the collider bias created by conditioning on A. This leaves only the directed edge $I \to Y$ open.

To identify the ADE of A on Y in Figure 1b, all the following backdoor paths between A and Y need to be blocked by conditioning: $A \leftarrow P \to C \to Y$, $A \leftarrow P \to L \to F \to Y$, $A \leftarrow P \to F \to Y$, $A \leftarrow P \to M \to F \to Y$, $A \leftarrow P \to M \to Y$, $A \leftarrow I \to M \to F \to Y$, $A \leftarrow I \to M \to Y$, $A \leftarrow I \to L \to F \to Y$, $A \leftarrow I \to C \to Y$, $A \leftarrow I \to Y$, and $A \leftarrow I \to F \to Y$. Since none of these pathways includes a collider, conditioning on the set of nodes $\{M, F, I\}$ yields the ADE of A on Y. The ADE of M, F, and C on Y can be identified in the same way. Therefore, the causal graph G can be assessed to see if the ADE of all observables in the set $\{I, M, F, C, A\}$ can be identified. Let Ω be a set of nodes such that $\Omega = \{I, M, F, C, A\}$. Then the ADE of Ω on Y is identifiable and is given by the following causal estimand:

$$\Pr(Y|do(\Omega)) = \Pr(Y|\Omega). \tag{2}$$

Equation (2) follows from the second rule of do-calculus with the independence $(\Omega \perp Y)_{G_{\underline{\Omega}}}$. The subgraph, $G_{\underline{\Omega}}$, used in the derivation of the direct effect of Ω on Y is shown in Figure 1c, in which all the outgoing arrows from Ω are stripped out, rendering the set Ω orthogonal to Y.

2.2 | Signed DAG and the monotonic effects

Figure 1d shows a signed causal graph G^S, in which each directed edge is assigned a sign, based on the assessment of section 2.1. Our aim is to determine the sign of the causal effect of every node in the set Ω on Y. Identifying the monotonic effects and the sign of the covariances based on the causal graph G^S also serves as a strategy to check any inconsistencies between the sign of the covariances in the data and those implied by G^S. For this purpose, we invoke the signed DAG analysis (VanderWeele & Robins, 2010). The following definitions and the theorem form the basis of the subsequent five propositions regarding the sign of monotonic effects in causal graph G^S (VanderWeele & Robins, 2010, p: 114, 116, 120):

Definition 1. A variable A exerts a positive monotonic effect on Y if intervening on A is always beneficial or neutral for every unit in the population with respect to Y.

Definition 2. The sign of every directed path in G^S is the product of the signs of the edges that constitute that path.

Definition 3. Two variables A and Y are said to be positively (negatively) monotonically associated if all the directed paths from A to Y are of positive (negative) sign and all common causes C, of A and Y are such that all the directed paths from C to A not through Y are of the same (opposite) sign as all the directed paths from C to Y, not through A.

Theorem 1. If A and Y are positively (negatively) monotonically associated, then $cov(A, Y) \geq 0(cov(A, Y) \leq 0)$.

Proposition 1. Higher female illiteracy (I) will increase the average value of infant mortality (Y).

Proof. The directed path $I \to Y$ in Figure 1d is positive, and all the directed paths $I \to M \to F \to Y$, $I \to M \to Y$, $I \to L \to F \to Y$, $I \to C \to Y$, $I \to A \to Y$, and $I \to F \to Y$ are also of positive sign and I and Y have no common causes. Given that I and Y are positively monotonically associated, $\text{cov}(I, Y) \geq 0$.

Proposition 2. Higher maternal care utilization (*M*) will decrease the average value of infant mortality (*Y*).

Proof. The directed path $M \to Y$ and $M \to F \to Y$ are negative and for the common causes of M and Y, which are I and P, the directed path from P to M not through Y ($P \to M$) is negative and of opposite sign to all the directed paths from P to Y not through M ($P \to A \to Y$, $P \to C \to Y$, $P \to F \to Y$, and $P \to L \to F \to Y$). For the second common cause I, the directed path from I to M not through Y ($I \to M$) is negative and of opposite sign to all the directed paths from I to Y not through M ($I \to Y$, $I \to A \to Y$, $I \to C \to Y$, $I \to L \to F \to Y$, and $I \to F \to Y$). Given that M and Y are negatively monotonically associated, $\text{cov}(M, Y) \leq 0$.

Proposition 3. Higher fertility (*F*) will increase the average value of infant mortality (*Y*).

Proof. The directed path $F \to Y$ is positive and for the common causes of F and Y, which are M, I and P, the directed path from M to F not through Y ($M \to F$) is negative and of same sign to the directed path from M to Y not through F ($M \to Y$). For the second common cause I, all the directed paths from I to F not through Y ($I \to F$ and $I \to L \to F$) are positive and of same sign as all the directed paths from I to Y not through F ($I \to Y$, $I \to M \to Y$, $I \to A \to Y$, and $I \to C \to Y$). For the third common cause P, all the directed paths from P to F not through Y ($P \to F$, $P \to L \to F$, and $P \to M \to F$) are positive and of same sign as all the directed paths from P to Y not through F ($P \to A \to Y$, $P \to C \to Y$, and $P \to M \to Y$). Given that F and Y are positively monotonically associated, $\text{cov}(F, Y) \geq 0$.

Proposition 4. Higher adolescent pregnancy (*A*) will increase the average value of infant mortality (*Y*).

Proof. The directed path $A \to Y$ is positive and for the common causes of A and Y, which are I and P, the directed path from P to A not through Y ($P \to A$) is positive and of same sign as all the directed paths from P to Y not through A ($P \to M \to Y$, $P \to C \to Y$, $P \to F \to Y$, and $P \to L \to F \to Y$). For the second common cause I, the directed path from I to A not through Y ($I \to A$) is positive and of same sign as all the directed paths from I to Y not through A ($I \to Y$, $I \to M \to Y$, $I \to C \to Y$, $I \to F \to Y$, and $I \to L \to F \to Y$). Given that A and Y are positively monotonically associated, $\text{cov}(A, Y) \geq 0$.

Proposition 5. Higher consanguineous unions (*C*) will increase the average value of infant mortality (*Y*).

Proof. The directed path $C \to Y$ is positive and for the common causes of C and Y, which are I and P, the directed path from P to C not through Y ($P \to C$) is positive and of same sign as all the directed paths from P to Y not through C ($P \to M \to Y$, $P \to A \to Y$, $P \to F \to Y$, and $P \to L \to F \to Y$). For the second common cause I, the directed path from I to C not through Y($I \to C$) is positive and of same sign as all the directed paths from I to Y not through C ($I \to Y$, $I \to M \to Y$, $I \to A \to Y$, $I \to F \to Y$ and $I \to L \to F \to Y$). Given that C and Y are positively monotonically associated, $\text{cov}(C, Y) \geq 0$.

One can assess the accuracy of the causal graph G (Figure 1b) by (1) testing the implied conditional independences; (2) comparing the sign of the monotonic effects obtained from G^S against those indicated by the data, and (3) introducing a confounder or a collider bias into the empirical model to test if such bias would manifest itself in the estimations as implied by the causal graph. These assessments should be considered as falsification tests of the accuracy of the causal graph G or G^S. Unfortunately, (1) is inadmissible because the conditional independences are

untestable due to the unobservables L and P in the model. However, (2) and (3) can be assessed empirically. These two falsification tests are conducted in Section 3.1 and in Section A of the Appendix, respectively.

2.3 | Latent exposure-outcome confounder

So far, the causal graph G did not account for latent confounders that cause both I and Y. If such unobservables exist in the model, the causal effect of I on Y ($I \rightarrow Y$) is not identifiable without additional assumptions. This situation is given in the causal graph G^Z, as shown in Figure 1e. The bidirected dashed edge between I and Y in Figure 1e indicates a latent common cause that confounds the relationship between I and Y. For instance, income might be one such latent confounder that affects both female illiteracy and infant mortality. The latent confounder indicates that there is a backdoor path from I to Y ($I \leftarrow ----- \rightarrow Y$) and that it cannot be blocked since the confounder is unobservable. When the backdoor adjustment is not possible, one can obtain the causal effect of I on Y by an adjustment with an **IV**-admissible set $\{Z\}$. For Z to be **IV**-admissible, the following conditions should hold:

Condition 1. The set $\{Z\}$ is d-separated from Y in G_I.

Condition 2. The set $\{Z\}$ is d-connected to I.

Condition 3. No node in the set $\{Z\}$ is a descendant of I.

Conditions 1 and 2 are satisfied by the rules of d-separation (Verma & Pearl, 1988), which state that Z and Y are independent from each other given I and that there is a directed edge $Z \rightarrow I$ and therefore Z and I are d-connected (not d-separated). Condition 3 is also satisfied because Z is not a child or a descendant of I. Note that Z would not be an admissible **IV** if one conditions on the set $\{M,F,C,A\}$ because doing so would violate condition 3 that additionally states that no nodes in the set $\{M,F,C,A\}$ are children or descendants of I.

Let the edge $Z \rightarrow I$ be α and the edge $I \rightarrow Y$ be β. From the causal graph G^Z, the causal effect of Z on I, that is α, can be estimated by the slope of a regression of I on Z. The causal effect of Z on Y can be estimated in the same way because Z and Y have no common causes and $Z \rightarrow I \leftarrow ----- \rightarrow Y$ is blocked by the collider I. Therefore, the causal effect of Z on Y is simply the product of the size of the edges that constitute the path $Z \rightarrow I \rightarrow Y$: $\alpha\beta$. Therefore the causal effect of I on Y, that is $\beta = \frac{\alpha\beta}{\alpha}$.

A credible application of an **IV** strategy requires an instrument that complies with the graphical assumptions of **IV** as shown in Figure 1e. The vexing part of this strategy is to find an instrument that satisfies all three conditions. In search for an IV, restrictions on data availability made it not possible to replace the causal instrument Z with a measured one. For example, two such causal instrument candidates are ethnicity or ethnic composition and income inequality, but data on these measures do not exist.

The solution is to search for a surrogate instrument K as shown in Figure 1f. In causal graph G^K, the gray filling of the node Z indicates that it is an *unmeasured causal instrument* with a directed edge $Z \rightarrow I$, whereas K is a *proxy or surrogate instrument* that is connected to I through $K \leftarrow Z \rightarrow I$ because the backdoor path from K to I is open due to Z being unobservable (Hernán & Robins, 2020) and K is d-separated from Y in G_I. Therefore, K also satisfies all three conditions above. A potential candidate from the literature on electoral studies meets the graphical assumptions of an **IV** and is a good fit for the surrogate instrument, K. The vote share of People's Democratic Party (HDP) of the June 2015 legislative election, obtained from the Turkish Supreme Election Council, is considered as a proxy instrument for female illiteracy rate.[4] Other instrument candidates were also considered. However, every potential

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[4]HDP is a leftist, pro-minority political party in Turkey whose founding principles uphold participatory democracy, egalitarianism, and the rights of minorities. HDP receives the highest share of votes in the Eastern and Southeastern regions, a cluster of historically underdeveloped regions that inhabit a significant Kurdish population.

candidate other than the HDP vote share in June 2015 elections was either unrelated or weakly related to the exposure (e.g., valid vote share or voter turnout rate) or strongly related to the exposure but was also directly related to the outcome or to the unobservables of the model (e.g., male illiteracy rate and spatial lag of female illiteracy rate), rendering them invalid.

3 | EMPIRICAL STRATEGY

We use a mix of regional (Nomenclature of Units for Territorial Statistics 1—NUTS-1) and provincial (NUTS-3) cross-sectional data, covering 81 provinces, averaged over the period of 2009–2017. The NUTS-1 and NUTS-3 regional maps of Turkey are shown in Figure 2.

3.1 | Data and sample

Infant mortality rates (Y), under-20 birth ratio (A), age-specific fertility rate (F), and female illiteracy rate (I) are retrieved from Turkish Statistical Office (Turkstat, 2018).[5] All of these variables are measured at the NUTS-3 level for the period 2009–2017. Consanguineous marriages (C) and maternal care utilization rates (M) are measured at the NUTS-1 level for the years 2006, 2011, 2016 and 2008, 2013, respectively, and are retrieved from the Turkish Family Structure Survey (TFSS, 2017).

Table 1 displays the descriptive statistics. The average infant mortality rate in the sample period is 11.08 deaths per 1000 live births. However, its geographic distribution, shown in Figure 3a, is spatially uneven with Southeastern Anatolia exhibiting the highest (15.36 deaths per 1000 live births) and the Istanbul region exhibiting the lowest rates (8.69 deaths per 1000 live births). A similar geographic skewness persists for female illiteracy rates among women between 25 and 44 years of age, which is about 14 times higher in the Eastern and Southeastern Anatolia regions in the sample period, compared to the Eastern Marmara and Aegean regions that have the lowest female illiteracy rates in the country (Figure 3b). The geographic distribution of under-20 birth ratio and fertility rates are shown in Figures 3c and 3d. The eastern parts of the Northeastern and Central East Anatolia are characterized by provinces with very high under-20 birth ratios and under-20 fertility rates. On the contrary, the Eastern Marmara region and part of the eastern Black Sea are characterized by provinces with low under-20 birth ratios and low fertility rates.

Figure 3e and f, respectively, shows the geographic distribution of consanguineous marriages and maternal care utilization rate. The geographic distribution of the share of consanguineous marriages in Figure 3e shows a clustering of very high rates of consanguineous unions in the southeastern region that gradually diminishes from the east to the west. A similar pattern is observed for maternal care utilization rates. There exists a clear east–west dichotomy showing that the Northeastern and the Central Eastern Anatolia regions have the lowest rates of maternal care utilization, followed by the Southeast Anatolia and the western regions that have the highest rates of utilization.

A comparison of the sign of monotonic effects from the causal graph G^S and those obtained from the data is given in Table 2. The predicted sign of the covariances between Y and each observable in the set $\Omega = \{I, M, F, C, A\}$ in the causal graph G^S is entirely consistent with the sign of the covariances obtained from the data.

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[5]Infant mortality rate, under-20 birth ratio, and age-specific fertility rate are defined as $\frac{D}{N} \times 1000$, $\frac{X}{N} \times 1000$, and $\frac{X}{N'} \times 1000$, respectively, where D is the number of deaths under 1 year of age, X is the number of births by women under the age of 20, N' is the female population under 20 years of age, and N is the total number of live births of newborns. Female illiteracy rate is defined as the ratio of illiterate women between 25 and 44 years of age to the female population between 25 and 44 years of age. This age interval is chosen based on the period in which a woman is considered fertile (see Section C of the Appendix for an expanded definition). An individual is said to be illiterate if she cannot read and write in the official language, no matter how proficient she is in her native language. Therefore, a woman who has not received formal education but who can read and write in the official language would be classified as literate (no degree), regardless of whether she can read and write in her native language, if different from the official language. Female illiteracy rate includes all women between the ages of 25 and 44, regardless of whether the individual is a mother because of a lack of data on motherhood. It also includes women who are not mothers and excludes women who are mothers but are older than 44. An overwhelming proportion of women in the 25–44 age group are also mothers, and the excluded women who have recently become mothers (with a child under 1 year of age) but who are also older than 44 are a very small proportion of all mothers in Turkey. Therefore, the inclusion of women who are not mothers and the exclusion of women who are mothers but are above the age of 44 should not impinge upon the assignment of female illiteracy as a good proxy for maternal education in Turkey.

(a) NUTS-1 Regions (12 regions)

(b) NUTS-3 Regions (81 provinces)

FIGURE 2 Regions of Turkey

TABLE 1 Descriptive statistics

	Mean (s.d.)	Min.	Max.
Health outcome			
Infant mortality rate (Y)	11.08 (2.52)	7.28	18.13
Maternal education			
Female illiteracy rate (I)	4.43 (6.03)	0.79	25.49
Birth and fertility			
Under-20 birth ratio (A)	74.88 (26.04)	30.64	140.03
Under-20 fertility rate (F)	7.62 (3.13)	2.90	17.19
Healthcare utilization			
Maternal care utilization rate (M)	90.43 (7.20)	76.75	96.55
Cultural factors			
Consanguineous marriages (C)	24.75 (9.62)	6.83	43.1

Notes: N = 81. All figures except maternal care utilization and consanguineous marriages are measured at NUTS-3 (81 provinces) level and averaged over the 2009–2017 period. Maternal care utilization is measured at NUTS-1 (12 regions) level and averaged over the years 2008 and 2013. Consanguineous marriages are measured at NUTS-1 (12 regions) level and averaged over the years 2006, 2011, and 2016. *, **, and *** denote the statistical significance at 10, 5, and 1%, respectively.
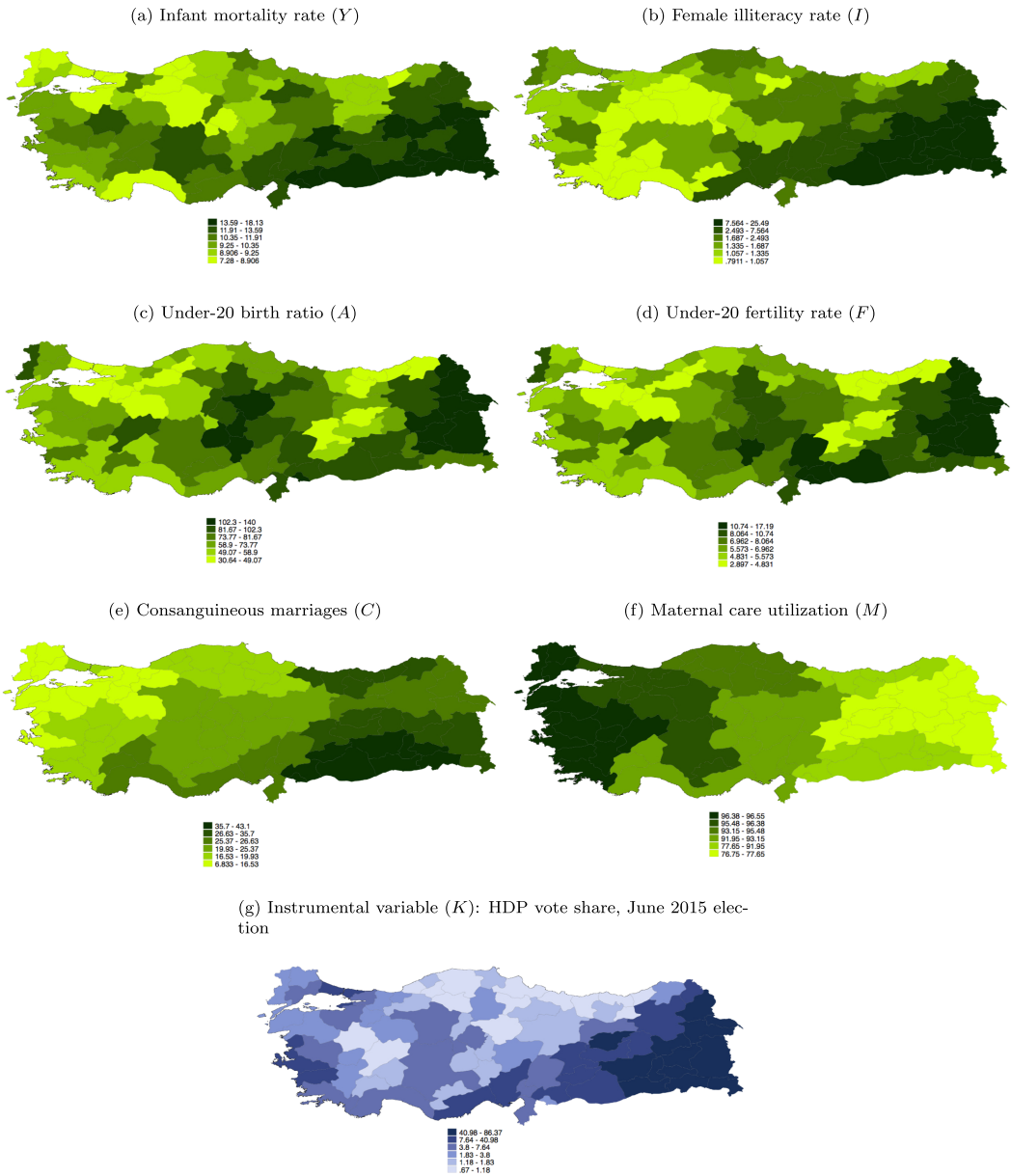
(a) Infant mortality rate (Y)

(b) Female illiteracy rate (I)

(c) Under-20 birth ratio (A)

(d) Under-20 fertility rate (F)

(e) Consanguineous marriages (C)

(f) Maternal care utilization (M)

(g) Instrumental variable (K): HDP vote share, June 2015 election

FIGURE 3 Geographic distribution of infant mortality, observable causes, and the instrumental variable
Notes: All variables except HDP vote share, maternal care utilization, and consanguineous marriages are measured at NUTS-3 (81 provinces) level and averaged over the 2009–2017 period. Maternal care utilization is measured at NUTS-1 (12 regions) level and averaged over the years 2008 and 2013. Consanguineous marriages are measured at NUTS-1 (12 regions) level and averaged over the years 2006, 2011, and 2016. HDP vote share data come from the June 2015 legislative election.

3.2 | Patterns of spatial dependence

The causal graph *G* implies the following linear models, respectively, for the ATE of *I* on *Y* and for the ADE of all *k* observable causes in the set Ω = {*I,M,F,C,A*} on *Y*:

TABLE 2 Sign of monotonic effects and covariances

	Sign of covariance from G^S	Covariance from data
Maternal education		
Female illiteracy rate (I)	$cov(I, Y) \geq 0$	$cov(I, Y) = 10.798$
Birth and fertility		
Under-20 birth ratio (A)	$cov(A, Y) \geq 0$	$cov(A, Y) = 33.659$
Under-20 fertility rate (F)	$cov(F, Y) \geq 0$	$cov(F, Y) = 4.887$
Healthcare utilization		
Maternal care utilization rate (M)	$cov(M, Y) \leq 0$	$cov(M, Y) = -10.918$
Cultural factors		
Consanguineous marriages (C)	$cov(C, Y) \geq 0$	$cov(C, Y) = 17.807$

$$Y_i = \mu_0 + \beta_0 I_i + u_{0i}, \tag{3}$$

$$Y_i = \mu_1 + \Omega_{ik}\gamma_k + u_{1i}. \tag{4}$$

The generalized spatial versions of equations (3) and (4) can be compactly written, respectively, as:

$$Y_i = \mu_0 + \rho \mathbf{W}Y_i + \beta_0 I_i + \mathbf{W}I_i\theta + v_{0i} \text{ where } v_{0i} = \lambda\mathbf{W}v_{0i} + \epsilon_{0i}, \tag{5}$$

$$Y_i = \mu_1 + \rho \mathbf{W}Y_i + \Omega_{ik}\gamma_k + \mathbf{W}\Omega_{ik}\theta_k + v_{1i} \text{ where } v_{1i} = \lambda\mathbf{W}v_{1i} + \epsilon_{1i}, \tag{6}$$

where v_{0i} and v_{1i} are the respective error terms, Ω_{ik} is the kth covariate for province i, and $\mathbf{W}$ is a $n \times n$ spatial weight matrix with elements w_{ij} indicating the relative connectivity between provinces i and j, and ρ and λ indicate the average strength of these effects conditional on $\mathbf{W}$. Two commonly used spatial weight matrices are a *row-normalized first-order queen contiguity* matrix whose elements are one if two provinces have a common border and zero otherwise and a *spectral inverse distance* spatial weight matrix where the elements w_{ij} of the matrix $\mathbf{W}$ contain the inverse of the distance between the centroid of provinces i and j, and is divided by its largest characteristic root (i.e., spectral normalization). When $\rho = \theta = 0$, the **SEM** is obtained where the interaction effects among the error terms in equations (5) and (6) represent the case where the unobservable determinants of infant mortality rates are spatially autocorrelated (Elhorst, 2014). When $\theta = \lambda = 0$, the spatial lag model (**SAR**) is obtained and the spillovers or externalities arise from the outcome. When $\lambda = 0$, the spatial Durbin model (**SDM**) is obtained, allowing for global spillovers in observables. Finally, when $\rho = 0$, the spatial Durbin error model (**SDEM**) is obtained, allowing for local spillovers in observables.

Intuitively, there are no reasons to expect that female illiteracy rate or infant mortality of the neighboring province j would directly affect infant mortality in province i. Rather, there might be unaccounted or omitted and spatially clustered factors that are more likely to give rise to a residual interaction effect (i.e., **SEM**) than any other specification. An example of such lurking unobservables is income, which is expected to be spatially autocorrelated with the eastern and southeastern regions having a much lower per capita income level than the rest of Turkey and western regions (Marmara and the Aegean regions) having higher-than-average per capita income levels. Therefore, throughout all global spatial models, we set $\rho = \theta = 0$, leading to a **SEM** and perform a number of tests to assess if the **SEM** can be constrained to ordinary least squares (**OLS**).[6]

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[6]The validity of other spatial specifications to augment the robustness of the choice of SEM is explored in Section B of the Appendix.

3.3 | Instrumental variables

The geographic distribution of the proxy or the surrogate instrument K is shown in Figure 3g. It clearly shows that the HDP vote share of June 2015 election is highly and positively correlated with female illiteracy rate, albeit there are outliers such as Izmir and Istanbul where HDP vote share is high but female illiteracy rate is low.

The causal graph G^K implies the following linear models for the effect of K on I (first stage) and I on Y (second stage), respectively:

$$I_i = \mu + \alpha K_i + u_i, \tag{7}$$

$$Y_i = \mu + \beta \hat{I}_i + v_i, \tag{8}$$

where $\hat{I}_i$ is the predicted value of female illiteracy from the first stage. In observational studies, one can use the non-causal association between the *proxy instrument* K and I in the denominator of the **IV** estimand (Hernán & Robins, 2020 p:197). With a continuous proxy instrument K, an exposure I, and an outcome Y, the **IV** estimand is $\beta_{IV} = \frac{Cov(K,Y)}{Cov(K,I)}$.

3.4 | Patterns of spatial heterogeneity

Global models of Section 3.2 assume spatial homogeneity in the processes and compute a single statistic that represents the average relationship between mortality and its predictors (i.e., the relationship is stationary across space). The geographically weighted regression (GWR) of Brunsdon, Fotheringham, and Charlton (1996), Fotheringham, Charlton, and Brunsdon (1996), Brunsdon, Fotheringham, and Charlton (1998), and Fotheringham, Brunsdon, and Charlton (2002) relaxes the assumption of spatial homogeneity and allows the parameter estimates of a regression to vary locally to account for the case that provinces may differ from each other not only in terms of infant mortality rates but also in terms of its causes. Therefore, the observable determinants of mortality may have different effects on different geographies. However, **GWR** constrains the local relationships within each model to vary at the same spatial scale.

A recent extension of the **GWR**, called multiscale **GWR** or **MGWR**, allows each relationship in the model to vary at a unique spatial scale and therefore computes covariate-specific bandwidths as opposed to a single bandwidth (Fotheringham, Yang, & Kang, 2017; Yu et al., 2020). This less-restrictive extension minimizes overfitting, reduces bias in the parameter estimates, and mitigates concurvity (Oshan, Li, Kang, Wolf, & Fotheringham, 2019). The **MGWR** counterparts of equations (5) and (6) take the form:

$$Y_i = \beta_{bw}(u_i, v_i) I_i + \varepsilon_i, \tag{9}$$

$$Y_i = \sum_k \eta_{bwk}(u_i, v_i) \Omega_{k,i} + \varepsilon_i, \tag{10}$$

where Y is infant mortality rate, I is the female illiteracy rate, $\Omega_{k,i} = \{I, M, F, C, A\}$ is the kth covariate at location i, $\beta_{bw}(u_i, v_i)$ and $\eta_{bwk}(u_i, v_i)$ are the locally varying coefficients where β_{bw} indicates the bandwidth used for the calibration of the conditional relationship between female illiteracy rate and mortality, $\eta_{bwk}(u_i, v_i)$ are the locally varying coefficients indicating the bandwidth used for the calibration of the kth conditional relationship, (u_i, v_i) is the $x - y$ coordinate of the ith location, and ε_i is the Gaussian error at location i.

The **MGWR** estimates a separate regression and uses a different weighting for each observation. Observations of closer locations have more influence on each other than observations that are spatially apart (Tobler, 1970). The bandwidth determines the way each observation is weighted and the way these weights decline with distance. An adaptive bandwidth selects a different bandwidth for each location so that all regression points have the same

number of nearest neighbors. The bandwidth is chosen by minimizing the small sample bias-corrected Akaike information criterion (AIC). An adaptive bi-square kernel is chosen so that the kernel bandwidth increases (decreases) in areas where data points are sparse (plenty). In contrast, a fixed bandwidth implies a greater likelihood that some local calibrations will be based on only a few data points. As a result, the distribution of local estimates will exhibit greater variation, and therefore larger standard errors (Fotheringham et al., 2002).

The adaptive bi-square kernel is $\mathbf{W}_{ij} = \begin{cases} \left[1 - \left(\frac{d_{ij}}{G_i} \right)^2 \right]^2 & \text{if } d_{ij} < G_i \\ 0 & \text{otherwise,} \end{cases}$ where d_{ij} is the distance between locations i and j, and G_i is the distance from point i to its Mth nearest neighbor where M is the optimal number of nearest neighbor (Fotheringham et al., 2017).[7]

4 | RESULTS

4.1 | Global results

4.1.1 | Total and direct effects

The results of the **SEM** estimates for the ATE of female illiteracy rate on infant mortality are given in columns (1)–(3) of Table 3.[8] Based on the causal graph G, the parameter estimate on female illiteracy rate captures the total effect (direct+indirect). Column (1) of Table 3 estimates equation (5) using a first-order, row-normalized queen contiguity spatial weight matrix.

Upon preliminary inspection, the geographic distribution of the residuals obtained from a **OLS** regression of female illiteracy rate on infant mortality clearly shows a clustering of positive and negative residuals and a mild spatial autocorrelation in the errors (residual Moran's I [z-value]: 0.451 [6.579]).[9] The total impact of female illiteracy (direct+indirect) on infant mortality is statistically distinguishable from zero at conventional test levels, suggesting that a 1-percentage point increase in female illiteracy rate increases infant mortality by 0.245 deaths per 1000 live birth of newborns.

As a robustness check, column (2) of Table 3 uses an *inverse distance* spatial weight matrix in *lieu* of a contiguity-based spatial weight matrix. The normalization procedure proposed by Elhorst (2001) and Kelejian and Prucha (2010) is followed, where each element w_{ij} of the pre-normalized inverse distance matrix is divided by its largest characteristic root. While the average strength of spatial dependence in the errors conditional on $\mathbf{W}$ is much stronger than those using a contiguity-based matrix, the ATE of female illiteracy in infant mortality upon the use of inverse distance weighting is 0.286 deaths per 1000 live birth of newborns and still statistically distinguishable from zero. Column (3) of Table 3 assesses the robustness of our estimates reported in column (1) to other functional forms. For this purpose, equation (5) is re-estimated in a full-logarithmic specification and the parameter estimate on female illiteracy captures elasticity. Again, the elasticity estimate of 0.141 is statistically distinguishable from zero at conventional test levels.

The results of the **SEM** estimates for the ADE on infant mortality rates are given in columns (4)–(6) of Table 3. Based on causal graph G, the parameter estimate of each observable captures the direct effect of that variable on mortality rate. A particular problem with the estimation of ADEs is that under-20 birth ratio and fertility rates have been found to be extremely correlated with each other ($\hat{\rho} = 0.97$). Preliminary **OLS** estimations also showed very high

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[7]The **MGWR** 2.0 software is developed by Z. Li, T. Oshan, S. Fotheringham, W. Kang, L. Wolf, H. Yu, and M. Sachdeva, available at Arizona State University, School of Geographical Sciences & Urban Planning, Spatial Analysis Research Center (SPARC): https://sgsup.asu.edu/sparc/multiscale-gwr.

[8]All global spatial regression analyses were performed using Stata/MP 15.0 (Stata Corp.).

[9]The global Moran's I statistic shows the extent of spatial autocorrelation and ranges between −1 and +1, where −1 indicates the perfect spatial randomness (i.e., checkerboard pattern) and +1 indicates the perfect spatial clustering.

TABLE 3 SEM estimates of the total and direct effects of observable causes on mortality rates

| | Infant mortality rate (Y) | | | | | | Male suicide rates |
| | Average total effects (ATE) | | | Average direct effects (ADE) | | | |
Outcome variable	Linear (1)	Linear (2)	Logarithmic (3)	Linear (4)	Linear (5)	Logarithmic (6)	Linear (7)
Constant	9.953***(0.531)	8.827***(1.239)	2.256***(0.043)	9.364***(4.130)	7.755***(3.586)	2.154 (1.540)	11.674***(5.076)
Maternal Education							
Female illiteracy rate (I)	0.245***(0.052)	0.286***(0.040)	0.141***(0.027)	0.115***(0.054)	0.093***(0.047)	0.073**(0.034)	-0.056 (0.067)
Birth and Fertility							
Under-20 birth ratio (A)	-	-	-	-0.040 (0.204)	-0.170 (0.191)	-0.001 (0.018)	0.325 (0.243)
Under-20 fertility rate (F)	-	-	-	0.658***(0.247)	0.767***(0.221)	0.055**(0.023)	-0.181 (0.299)
Healthcare utilization							
Maternal care utilization rate (M)	-	-	-	-0.016 (0.041)	-0.0005 (0.036)	-0.073 (0.325)	-0.050 (0.050)
Cultural factors							
Consanguineous marriages (C)	-	-	-	0.106***(0.033)	0.123***(0.028)	0.157***(0.059)	-0.030 (0.041)
Spatial dependence							
λ (spatial error)	0.686***(0.090)	0.914***(0.061)	0.635***(0.099)	0.477***(0.123)	0.737***(0.178)	0.489***(0.121)	0.557***(0.112)
% ATE direct	-	-	-	46.94	32.52	51.77	-
Pseudo R^2	0.5031	0.5031	0.5377	0.6817	0.6908	0.6503	0.1597
Log pseudo-likelihood	-145.26	-153.98	52.539	-137.21	-139.85	57.827	-151.02
Akaike Information Criterion (AIC)	298.53	315.96	-97.077	290.41	295.70	-99.654	318.04

(Continues)

TABLE 3 (Continued)

| Outcome variable | Infant mortality rate (Y) | | | | | | Male suicide rates |
| | Average total effects (ATE) | | | Average direct effects (ADE) | | | |
	Linear (1)	Linear (2)	Logarithmic (3)	Linear (4)	Linear (5)	Logarithmic (6)	Linear (7)
Variance Inflating Factor (VIF)	-	-	-	2.18	2.18	2.39	-
Wald test ($\lambda = 0$)	57.77***	226.91***	40.87***	14.99***	17.09***	16.17***	24.61***
LR test (OLS vs. SEM)	31.69***	14.25***	24.41***	8.25**	2.96	8.84**	-
Spatial weight matrix	contiguity	distance	contiguity	contiguity	distance	contiguity	contiguity

Notes: The unit of observation is the province. $N = 81$. All variables except maternal care utilization and consanguineous marriages are measured at NUTS-3 (81 provinces) level and averaged over the 2009-2017 period. Maternal care utilization is measured at NUTS-1 (12 regions) level and averaged over the years 2008 and 2013. Consanguineous marriages are measured at NUTS-1 (12 regions) level and averaged over the years 2006, 2011 and 2016. Total effect refers to all directed edges that emanate from female illiteracy (I) and reach mortality (Y) in the causal graph G in figure 1b, while the direct effects refer to each of the directed edges $I \rightarrow Y$, $A \rightarrow Y$, $F \rightarrow Y$, $C \rightarrow Y$ and $M \rightarrow Y$. Contiguity and distance respectively refer to a row-normalized first-order queen contiguity and a spectral inverse distance spatial weight matrices. LR test (OLS vs. SEM) reports the chi-square for the null hypothesis of no spatial dependence. Robust standard errors in parentheses. *, ** and *** denote statistical significance at the 10, 5 and 1 percent respectively.

individual variance inflating factors (VIFs) of 31.03 and 25.38 with a mean VIF of 12.79. To mitigate multicollinearity bias and to include both variables in the model to obtain the ADE based on the implications of the causal graph G, the least invasive procedure is to orthogonalize both variables before the estimations. This yielded a mean VIF of 2.18 in column (4) that is drastically lower and considered safe.

Upon the inclusion of the remaining factors, the parameter estimate on female illiteracy rate in column (4), relative to column (1), drops from 0.245 to 0.115 but remains statistically significant at conventional test levels. This indicates that about 47% (0.115/0.245) of the total effect of female illiteracy is the direct effect, whereas the remaining 53% is the indirect effect through birth and fertility measures, healthcare utilization, and cultural factors. From column (4), the other two most important direct causes of higher infant mortality are fertility rates and the share of consanguineous marriages whose effects are statistically distinguishable from zero at conventional test levels.

Columns (5) and (6) report the **SEM** estimates using a spectral inverse distance spatial weight matrix and a logarithmic model, respectively. Again, although the average strength of spatial dependence conditional on **W** is much stronger using an inverse-distance matrix, the magnitude and the statistical significance of the ADE of female illiteracy in infant mortality upon the use of inverse distance weighting is slightly lower at 0.093 deaths per 1000 live birth of newborns and still statistically distinguishable from zero. This indicates that, relative to column (2), about 33% of the ATE of female illiteracy on infant mortality is the direct effect, whereas the remaining 67% is the indirect effect. In the full-logarithmic model reported in column (6) of Table 3, about 52% (0.073/0.141) of the total effect of female illiteracy on infant mortality is the direct effect, whereas the remaining 48% is the indirect effect through birth and fertility measures, healthcare utilization, and cultural factors. The linear hypothesis testing for the spatial error ($\lambda = 0$) term and the LR test, respectively, suggests that the residual interaction effects are individually statistically and significantly distinguishable from zero at conventional test levels and that the **SEM** cannot be constrained to **OLS** for any of the models in Table 3 except column (5). The size of the AIC and the log-pseudo likelihood also favor the **SEM**.

The final exercise assesses the robustness of our estimates to a spurious outcome. For this purpose, we assess the impact of female illiteracy on an outcome that is known to be unaffected by any of the observables. Our confidence in the causal effects would be severely undermined if the parameter estimates turn out to be statistically distinguishable from zero at conventional test levels. The reason is that if a regression of female illiteracy rate on a spurious outcome yields a statistically significant effect, then one can conclude that the effect of female illiteracy rate on infant mortality may also have been the results of the statistical fluke and not of a true effect. For this purpose, we collected data on provincial male suicide rates, averaged over the 2009–2017 period. If female illiteracy is truly unrelated to male suicide rates, its parameter estimate should be statistically indistinguishable from zero at conventional test levels. Column (7) of Table 3 reports the results. Expectedly, none of these factors has any statistically distinguishable impact on male suicide rates.

Overall, the results of Table 3 show that irrespective of the functional form or of the specification of the spatial associations, female illiteracy has a statistically significant and positive direct effect on infant mortality.[10] Depending on the functional form and the spatial weight matrix used, about 33–52% of the total effect is the direct effect, whereas the remaining is the indirect effect. Along with female illiteracy, fertility rates and consanguineous marriages stand out as important factors of infant mortality. The robustness checks show that both the ATE and the ADE are robust to spurious outcome, to functional form, and to the choice of the spatial weight matrix.

4.1.2 | Estimates of IV

Based on causal graph 1f, Table 4 shows the **IV** estimation results with a set of diagnostics on endogeneity, weak identification, and instrument validity. Column (1) of Table 4 reports the first-stage results of a non-spatial regression of female illiteracy on the excluded instrument, HDP vote share of the June 2015 legislative election, and columns (2) and (3), respectively, report the parameter estimate on the predicted values of female illiteracy rate from the first-

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[10]Section C of the Appendix conducts a robustness check using an expanded version of female illiteracy rate, defined as the ratio of illiterate women between the ages 14 and 49 to the female population between the ages 14 and 49.

TABLE 4 IV estimates of the causal effect of female illiteracy on infant mortality

Outcome variable	Female illiteracy(I) First-stage (1)	Infant mortality rate(Y) Second-stage IV (2)	SEM-IV (3)
Constant	0.858***(0.149)	9.820***(0.253)	9.897***(0.564)
Maternal education			
Female illiteracy rate (I)	-	0.286***(0.023)	0.258***(0.072)
Proxy instrument			
HDP vote share (K)	0.220***(0.017)	-	-
Spatial dependence			
λ (spatial error)	-	-	0.699***(0.101)
Wald test ($\lambda = 0$)	-	-	47.76***
First-stage F statistic	174.95	-	-
Underidentification test	-	16.138 [0.0001]	-
Weak identification test	-	430.738	-
Anderson-Rubin statistic	-	16.44 [0.0001]	-
Endogeneity test	-	1.56 [0.2116]	-

Notes: The unit of observation is the province. $N = 81$. Female illiteracy rate is instrumented by the HDP vote share of the June 2015 legislative election. The underidentification test reports the Kleibergen-Paap rk LM statistic and the p-value for the null hypothesis that the equation is underidentified (i.e., the excluded instruments are irrelevant) (Kleibergen & Paap, 2006). The weak identification test reports the Cragg-Donald Wald F-statistic and the p-value for the null hypothesis that the equation is weakly identified (Cragg & Donald, 1993). Weak identification test critical values for 10 and 15% maximal IV size are 16.38 and 8.96, respectively (Stock & Yogo, 2005). Anderson-Rubin test statistic reports the weak identification-robust inference Wald statistic and the p-value for the null hypothesis that the coefficient of female illiteracy rate is zero (Anderson & Rubin, 1949). The endogeneity test reports the chi-square and the p-value for the null hypothesis that female illiteracy rate is exogenous. SEM models use a row-normalized first-order queen contiguity spatial weight matrix. Robust standard errors in parentheses and p-values in brackets. *, **, and *** denote the statistical significance at 10, 5, and 1%, respectively.

stage regression, using a non-spatial **IV** and a **SEM-IV** (second stage). Both the first- and the second-stage **IV** regressions exclude measures of birth and fertility, healthcare utilization, and consanguinity for the fact that the inclusion of these measures violates condition 3 of the graphical assumptions of an **IV**.

The first-stage F statistic, as a suggested measure to assess the explanatory power of the excluded instrument, is well above 10, indicating that the instrument is not weak (Bound, Jaeger, & Baker, 1995; Staiger & Stock, 1997). The diagnostic test results indicate that the instrument is relevant (correlated with female illiteracy) and also indicate that the null hypothesis that female illiteracy is exogenous cannot be rejected at conventional test levels. The non-spatial **IV** model indicates that female illiteracy exerts a positive causal effect on infant mortality rate by a factor of 0.286 deaths per 1000 live birth of newborns.

Column (3) of Table 4 accounts for spatial dependence in the errors and performs a generalized spatial two-stage least squares (**GS2SLS**) estimation of a model of global spillovers in unobservables (i.e., **SEM**). The causal effect of female illiteracy is statistically distinguishable from zero at conventional test levels and indicates an increase in infant mortality rate by a factor of 0.258 deaths per 1000 live births of newborns.

4.2 | Local results

The assumption of spatial homogeneity is relaxed by estimating a **MGWR** that computes a unique parameter estimate for every province and for every causal variable along with female illiteracy rate. In all summary statistics for

the locally varying coefficients reported in Tables 5 and 6, the bandwidths are covariate-specific and the significance of the parameter estimates have been adjusted using the procedure proposed by da Silva and Fotheringham (2016). All surface of estimates are reported at 1% significance level.

TABLE 5 Geographic variability of MGWR estimates, total effects

Panel A: Summary for local coefficients						
			Outcome: Infant mortality rate(Y)			
	Bandwidth	Adj. α (95%)	Mean (s.d.)	Min.	Max.	Spatial variability p-value
Intercept	43	2.571	0.043 (0.256)	−0.316	0.642	0.000
Maternal education						
Female illiteracy rate (I)	80	2.074	0.661 (0.065)	0.586	0.778	0.015
Panel B: Model diagnostics						
	AIC		Adj. R^2	Residual Moran's I		
OLS	179.53		0.497	0.125***[8.646]		
MGWR	154.22		0.646	0.046***[3.639]		

Notes: ***denotes the statistical significance at 1% level. Z-scores in brackets. The residual Moran's I uses a spectral inverse distance spatial weight matrix. The Monte Carlo spatial variability test reports the p-value for the null hypothesis that the spatial variability in the local estimates arises by chance.

TABLE 6 Geographic variability of **MGWR** estimates, direct effects

Panel A: Summary for local coefficients						
			Outcome: Infant mortality rate(Y)			
	Bandwidth	Adj. α (95%)	Mean (s.d.)	Min.	Max.	Spatial variability p-value
Intercept	80	2.074	−0.067 (0.026)	−0.115	−0.013	0.602
Maternal education						
Female illiteracy rate (I)	80	2.047	0.211 (0.016)	0.174	0.270	0.736
Birth and fertility						
Under-20 birth ratio (A)	43	2.527	−0.055 (0.111)	−0.299	0.089	0.105
Under-20 fertility rate (F)	80	2.155	0.385 (0.031)	0.334	0.429	0.523
Healthcare utilization						
Maternal care utilization rate (M)	48	2.374	0.017 (0.080)	−0.119	0.222	0.222
Cultural factors						
Consanguineous marriages (C)	43	3.437	0.421 (0.180)	0.117	0.792	0.005
Panel B: Model diagnostics						
	AIC		Adj. R^2	Residual Moran's I		
OLS	149.20		0.675	0.036***[2.971]		
MGWR	142.81		0.733	−0.006 [0.416]		

Notes: ***denotes the statistical significance at 1% level. Z-scores in brackets. The residual Moran's I uses a spectral inverse distance spatial weight matrix. The Monte Carlo spatial variability test reports the p-value for the null hypothesis that the spatial variability in the local estimates arises by chance.
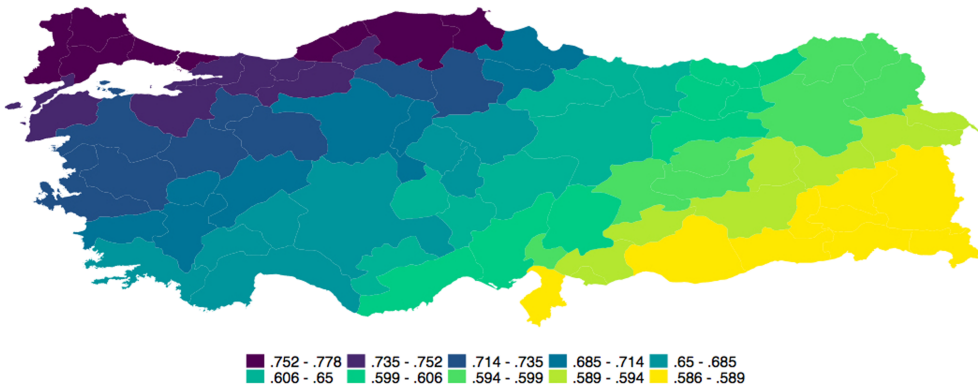
4.2.1 | Total and direct effects

The geographic distribution of the **MGWR** results for the local total effects is mapped in Figure 4 along with the summary for local coefficients in Panel A of Table 5. All local coefficient estimates of female illiteracy rate are positive and range between 0.586 and 0.778 with a mean of 0.661, indicating spatially monotonically positive effects.

Figure 4a maps the surface of estimates for the local total effects of female illiteracy rates on infant mortality. Expectedly, higher female illiteracy is associated with spatially monotonically increasing infant mortality rates throughout Turkey's provinces, with stronger effects concentrated in Marmara region and parts of the Western Black Sea. The spatial variability test reported in Panel A of Table 5 shows that the null hypothesis that the spatial variability of the local estimates has arisen by chance can be safely rejected.

Figure 5 maps the surface of estimates for the local direct effects on infant mortality along with the summary for local coefficients in Panel A of Table 6. The last column in Panel A of Table 6 indicates that only the spatial variability of the local estimates of the share of consanguineous marriages on infant mortality is attributable to the inherent processes. Again, the summary statistics for the local coefficients indicate that consanguinity exerts positive monotonic effects, ranging between 0.117 and 0.792. The surface of estimates of the local direct effects of

(a) Local total effects of female illiteracy on infant mortality

.752 - .778   .735 - .752   .714 - .735   .685 - .714   .65 - .685
.606 - .65   .599 - .606   .594 - .599   .589 - .594   .586 - .589

(b) Local R^2

.783 - .801   .73 - .783   .679 - .73   .655 - .679   .619 - .655
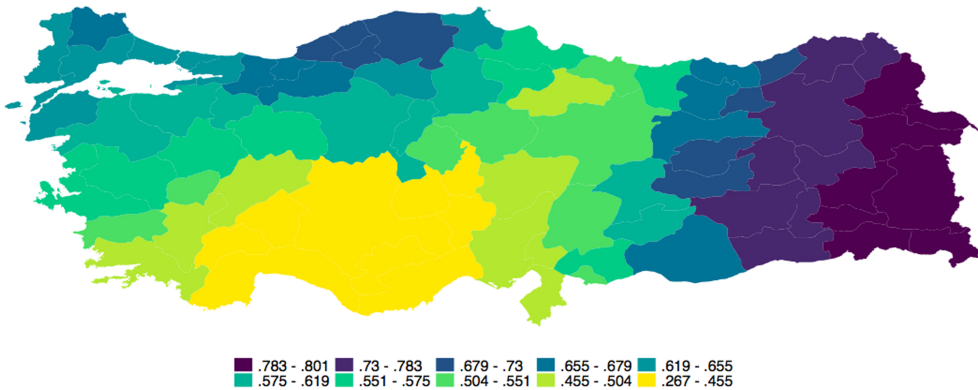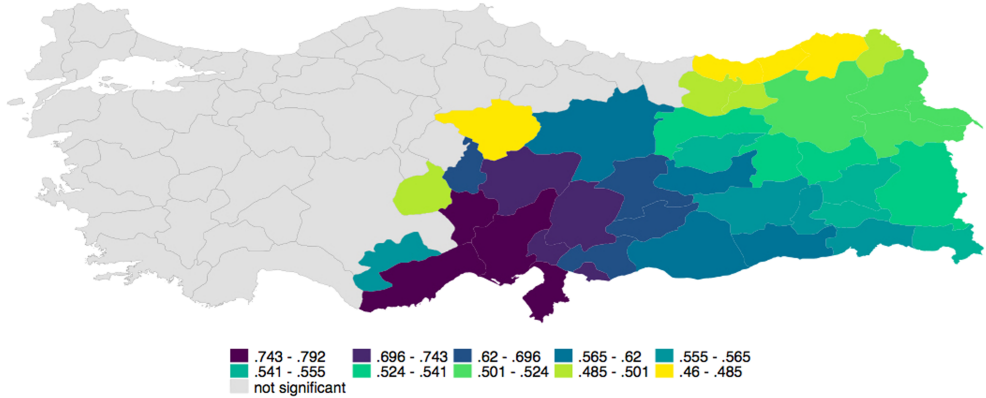.575 - .619   .551 - .575   .504 - .551   .455 - .504   .267 - .455

FIGURE 4   Spatial distribution of **MGWR** estimates, total effects
Notes: All locally varying estimates are statistically significant at 1% level.

(a) Local direct effects of consanguinity on infant mortality
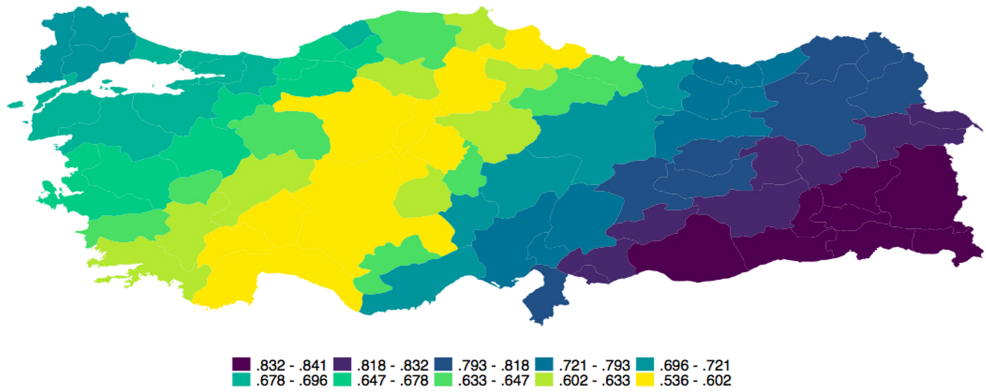

(b) Local R^2

FIGURE 5 Spatial distribution of **MGWR** estimates, direct effects
Notes: All locally varying estimates are statistically significant at 1% level.

consanguinity, shown in Figure 5a, points out that the largest statistically significant effects are concentrated in the eastern Mediterranean and the adjacent provinces of the Southeastern Anatolia, a region known for extremely high rates of consanguineous unions (25–43% of all marriages).

4.2.2 | Model diagnostics

Panel B of Tables 5 and 6 assesses the model performance of **MGWR** over **OLS** by a comparison of AIC; the adjusted **R**^{2} and the residual Moran's I (Anselin, 1995; Anselin, Bera, Florax, & Yoon, 1996; Anselin, 1998; Moran, 1950). From Table 5, the AIC of the **MGWR** is about 14% lower than that of the **OLS** regression suggesting that the global regression is inadequate. On the contrary, the adjusted **R**^{2} for the **MGWR** model is conspicuously higher than their respective **OLS** counterpart. The residual Moran's I displayed in Panel B of Tables 5 and 6 compares the extent of spatial autocorrelation in the residuals of **OLS** against the **MGWR** and is computed using a *spectral inverse distance* weighting. For the local total effects, the residual spatial autocorrelation persists upon estimating a **MGWR** model yet they are extremely small. For the local direct effects, on the contrary, the residual spatial autocorrelation is positive but extremely weak for the **OLS** to begin with and disappears upon estimating a **MGWR** model.

The surface of estimates of the local R^2 is shown in Figure 4b for the local total effects and in Figure 5b for the local direct effects. They show that the female illiteracy rate alone or in combination with the remaining observable causes explains up to 80% of the variation in infant mortality rates in the bordering provinces of the far Northeastern and far Central-Eastern Anatolia regions. On the contrary, consanguinity, as a direct cause, explains up to 84% of the variation in infant mortality in the Southeastern and Central-Eastern Anatolia regions.

5 | CONCLUSION

This study attempts to identify the causal effects of female illiteracy, birth and fertility, healthcare utilization, and consanguinity on infant mortality in Turkey with a focus on the patterns of spatial dependence and spatial heterogeneity in the processes. There are only a handful of questions we can answer, and many more remain unanswered. The diversity of the questions are limited because of our adherence to the statistical implications of the patterns of relationships among the causes of infant mortality and to the requirements for credible inference entailed by the conceptual framework. With this motivation, a set of causal graphs of the measured and unmeasured causes of infant mortality are built. The conceptual framework indicates that an intervention on female education through a policy change would translate into a significant drop in infant mortality rates, not only directly but also through its impact on birth and fertility, healthcare utilization, and cultural factors.

The global spatial regressions show that female illiteracy plays a central role in the containment of rising infant mortality rates. The significant association between female illiteracy and infant mortality is robust to functional form, spurious outcome, and alternative specification of the spatial weight matrix, reinforcing the view that these effects are likely to represent associations beyond correlations.

Through estimating a **MGWR** to address spatial heterogeneity in the processes, the locally varying impact of female illiteracy on infant mortality rates is found to be stronger in the neighboring provinces of Marmara and the Western Black Sea regions. From Figure 3a, these regions also have the lowest levels of infant mortality in the nation. On the contrary, the locally varying impact of consanguinity on infant mortality rates is found to be stronger in the neighboring provinces of the Mediterranean and the Southeastern Anatolia regions, a region characterized by an already high rate of infant mortality and consanguineous unions. The clustering of the spatially varying effects in these provinces emphasizes location-specific policies to reduce infant mortality. The high explanatory power of the **MGWR** in the Eastern and Southeastern Anatolia confirms that female education and consanguinity are important causes of high infant mortality rates in these regions. The analysis suggests that reducing illiteracy through continuous education in mother and child care may have a large impact on the containment of infant mortality rates, especially in the Kurdish-populated Eastern and Southeastern Anatolia regions where female illiteracy and infant mortality rates are the highest in the nation.

This study has several limitations. First, due to a lack of data on maternal education and linguistic barriers in Turkey, female illiteracy is used as a proxy variable for these unobservables. Although it is deemed to be a good proxy, the downside is that since female illiteracy is not a perfect measure for maternal education, inferences should not be drawn regarding the impact of maternal education on infant mortality and the impact of female illiteracy may not capture the true effect due to this imperfection. Second, data on maternal care utilization and consanguineous marriages were available only at the NUTS-1 (12 regions) level. Using aggregate data at a geographic scale coarser than NUTS-3 (81 provinces) hides province-level spatial variation within regions, which is likely to have reflected itself in the magnitude of the parameter estimates.

A third limitation is that the causal interpretation of the direct effects requires fairly strong assumptions. Control must be made for the exposure (I) and outcome (Y) confounding (Assumption 1); control must be made for the mediator (M, F, C, A) and outcome (Y) confounding (Assumption 2); control must be made for the exposure (I) and mediator (M, F, C, A) confounding (Assumption 3); and the mediator and outcome confounder should not be affected by the exposure (Assumption 4) (VanderWeele, 2016). Assumptions 1 and 3 are necessary only for the causal interpretation

of the total effect. On the contrary, mediation additionally requires Assumption 2. While the causal graph G in Figure 1b satisfies the first three assumptions, the causal graph G^K in Figure 1f specifically deals with the violation of Assumption 1 and the fourth assumption needed for standard estimates to be interpreted as direct effects is partially satisfied,[11] there may be unaccounted latent confounders between the mediators and the outcome that are absent in the causal graph G. If such confounders exist, this would violate Assumption 2 and sensitivity analyses would have to be conducted to assess how strong the mediator-outcome confounder should be in order for the reversion of our conclusions regarding the average causal mediation effects (ACME) (i.e., failure to reject the null hypothesis that ACME = 0). The sensitivity analysis of unmeasured confounding becomes even more challenging if there are global or local spillovers in the (un)observables in the model. This study did not address such a challenge. Therefore, our results should be interpreted in the shadow of a potential violation of Assumption 2.

Fourth, the choice of aggregate versus individual-level data involves a trade-off. As much as regional analysis with aggregate data allows us to study spatial effects and locality, using individual-level data might be convenient to study causal mediation effects if one is willing to sacrifice spatial aspects. With the choice of individual-level data, the study design should be suitable to collect data on potential mediator-outcome confounders to avoid pitfalls in inference. Given the breadth of questions on mother and child care, nutrition, fertility, family planning, and socioeconomic status, the quinquennial national population surveys conducted by *Hacettepe University, Institute of Population Studies* have the potential to identify causes and effects at the cost of ignoring the role of geography.

CONFLICT OF INTEREST

The author declares to have no conflict of interest.

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[11]Assumptions 1 and 3 are trivially satisfied since there is no incoming arrow to I in causal graph G. For mediators M, C, A, control must be made for I and P. For mediator F, control must be made for I, P, M. P is the only unobservable node in this set and cannot be controlled for. However, controlling for A in the association between M and Y or F and Y or C and Y blocks the confounding effect of P because A is in the pathway $P \rightarrow A \rightarrow Y$ and P does not directly affect Y. Therefore, Assumption 2 is satisfied by adjusting for A. Assumption 4 is satisfied for the mediators M, C, A, L but not for the mediator F because M is a confounder between F and Y and M is also affected by I.

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