Causal Inference on Networks under Continuous Treatment Interference: an application to trade distortions in agricultural markets

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Abstract

Extensive work has been done to assess the role played by externalities in policy interventions. Studies dealing with this issue in experimental settings abound in the literature. Much less attention, however, has been devoted to the case when data comes from observational studies. In this paper, we address this gap by presenting a methodology to draw causal inference in a non-experimental setting subject to interference. Specifically, we develop a generalized propensity score-based estimator that allows estimating both direct and spillover effects of a continuous treatment. In order to showcase this methodology, we investigate whether and how spillover effects shape the optimal level of producers' support in agricultural markets. Our results show that, in this context, neglecting interference may lead to a downward bias when assessing policy effectiveness.

Keywords: Causal inference, networks, continuous treatment interference, agricultural incentives, trade distortions.

JEL Classification: C21, F14, F60, L14, Q17

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1. Introduction

A rigorous evaluation of the effectiveness of a policy or program, as measured by the impact accrued by beneficiaries, determines its success and suggests its potential adoption in different contexts. Knowledge of externalities at play can be crucial in this framework, because decision makers may want to either leverage or reduce them to improve policy effectiveness (Moffitt, 2001). This is for example the case when assessing schooling decisions (Lalive et al., 2009), contrasting criminal involvement (Glaeser et al., 1996), designing health programs (Miguel et al., 2004), or imposing trade restrictions (Giordani et al., 2016).

The presence of externalities poses several challenges to policy evaluations. When neglected, it can hamper their assessment and lead to wrong conclusions. Ideally, spillover effects can be identified using cleverly designed experiments, such as those recently reviewed by Baird et al. (2018). In practice, however, broad-based policy experimentation is often hard to implement and the only viable alternative to assess a policy impact is through observational data. Unfortunately, approaching this analysis with traditional impact evaluation methodologies, such as propensity score matching (PSM) (Dehejia et al., 2002)[1], is not an option when externalities are at play, because these methods rely on the assumption that an agent's treatment will not spillover across other agents (Cox, 1958). This is the assumption of no interference between agents, also called Individualistic Treatment Response (ITR) (Manski, 2013).

In this paper, we address this issue by developing one of the first frameworks in observational studies to evaluate policy interventions in the presence of interference. The mechanism of interference is formalized through an investigation of the agents' interactions, following the raising literature on network studies (Jackson et al., 2017). Modeling agents' interplay as a network presents several advantages. First, it allows to conceive each agent as subject to two treatments, i.e. the individual treatment and the neighborhood treatment. The latter is the area of the network from which spillover effects originates and, once pinpointed, the mechanism of interference determined by agent's interactions can be identified and measured. Second, it provides a formal framework to display the process of the spread of treatment, and potential feedback effects. As a result behavior predictions are typically improved, revealing a variety of mechanisms to leverage social interactions. This is for example the case of Banerjee et al. (2013), who employed social network data in India to identify best practices in a microfinance program, and Beaman (2012) who examines the implications of social networks for the labor market of refugees resettled in the U.S. However, while there is a vast literature on the design, conduct, and analysis of studies for evaluating the efficacy of treatment (Sacerdote, 2014; Athey & Imbens, 2017), very little has been done to properly frame the role of network structures in

[1] Broadly speaking, PSM involves pairing treatment and comparison units that are similar in terms of their observable (pre-treatment) characteristics. Conditioning on these covariates, matching methods yield an unbiased estimate of the treatment impact (Rosenbaum & Rubin, 1983).

causal inference, and the issue of causality is still very much debated in both economic and network analysis (Doreian, 2001; Arpino et al., 2017).

Building on this analytical setting, we design a joint propensity score (JPS), which balances individual and neighborhood covariates across agents under different levels of individual and neighborhood treatment. This approach was first developed by Forastiere et al. (2016), who provided one of the few efforts in the literature to handle interference using observational data. With respect to that study, a key novelty of this paper is represented by the source of treatment considered. While Forastiere et al. (2016) proposes a method to study binary treatment, here we consider a continuous treatment at both individual and neighborhood level.

Following the approach of Aronow & Samii (2017) and Forastiere et al. (2016), in Section 2 of the paper we describe a new framework allowing to i) correct for the bias resulting from both treatment selection and interference and ii) quantify direct and spillover effects of a continuous treatment. Specifically, both direct and spillover effects will be examined as dose-response functions. We showcase the benefits of this methodology through an application to trade distortions in agricultural markets in Section 3. Motivated by the emergence of globally fragmented agri-food production networks and the still very high level of interventions in agricultural markets (Freund and Ozden, 2008; Balié et al., 2018), we investigate whether and how trade network effects shape the optimal level of producers' support. Our results show that neglecting interference may lead to a downward bias when assessing policy effectiveness. Section 4 concludes and draws policy implications.

2. Methodology

We analyze the issue of causal inference when externalities matter and data come from observational studies. In this framework, the agent is subject to two treatments: the individual treatment, directly assigned to the agent, and the neighborhood treatment, received by other agents and to which he is indirectly exposed. This approach was established in the literature on causal inference in the presence of interference in recent years, but only few studies proposed a formalization of it (see Tchetgen Tchetgen & VanderWeele (2012) for a review).

In this paper, we model the presence of interference adopting a social network perspective whereby agents are seen as nodes, and their interactions, mediating spillover effects, are represented as links: i.e., i and j are connected through a link if they interact with each other. Following Aronow & Samii (2017) and Forastiere et al. (2016), a function is used to both map agent's exposure to the treatment that spillover across the network, and locate the area of the network, henceforth *neighborhood*, from which spillovers originate (e.g. friends or commercial partners). This procedure, known as *exposure mapping*, allows to disentangle the effect

of the individual treatment from that resulting from the exposure to the treatment received by other agents located in the neighborhood.

A critical assumption in the literature on causal inference is unconfoundedness (Rubin, 1990). This implies that the treatment can be considered randomly assigned by adjusting for agents' differences in a fixed set of exogenous pre-treatment characteristics. The presence of interference, however, requires an additional assumption, i.e. both the individual and the neighborhood treatments should be unconfounded conditional on covariates. The extension of unconfoundedness to neighborhood treatment is a crucial element of this study, as the process by which agents clump together is often anything but random. This strategic self-selection makes it difficult to compare spillover effects across agents, because they might be non-random, and driven by omitted variables, e.g. agents' characteristics. In these cases, the identification of externalities in a group is hindered by endogenous factors, and a correct agents comparison is obtained by adjusting also for the characteristics featured by either or both the agent and the neighborhood (as identified by the exposure mapping). Only in this way neighborhood treatment can also be considered as random, and any potential confounding effect is offset.

In the next section, we show how to deal with this issue presenting a covariate-adjustment method which yields an unbiased estimate of both the treatment and the spillover effects. The estimator, designed to correct for interference, is based on a joint propensity score (JPS), which balances individual and neighborhood covariates across agents under different levels of individual and neighborhood treatment. As already mentioned, this approach builds on Forastiere et al. (2016), who develop a setting with binary treatment, where a subclassification method adjusts for the individual propensity score and, within each stratum, a model-based approach adjusts the neighborhood propensity score. With respect to that study, our estimator is modified by replacing the subclassification on the propensity score of the binary treatment with a second generalized propensity score (GPS) for continuous treatment. The latter builds on the well-known generalized propensity score approach for continuous treatment proposed by Hirano & Imbens (2004)

2.1. Notation

Let us define a partition of the set of nodes (agents or units) $\mathcal{N}$ around node i as $(i, \mathcal{N}_i, \mathcal{N}_{-i})$, where the set $\mathcal{N}_i$, referred to as the *neighborhood* of agent i [2], has cardinality N_i and contains all nodes j connected to i, and the set $\mathcal{N}_{-i}$ contains all nodes other than i that are not in $\mathcal{N}_i$. For consistency with the literature of social networks (see e.g. Jackson (2010)), the number of neighbors N_i is referred to as the *degree* centrality of agent i. Assume now that agents are embedded in a network, where links register the presence and level

[2]Note that in this framework, i's neighbors, i.e. its direct connections in the network, can be defined both by a social or a spatial criterion.

of interaction between agents. This network can be represented by the adjacency matrix A, with element a_{ij} denoting the strength of connection between agent i and j.

Let now $Z_i \in \mathscr{Z}$ be the continuous variable representing the treatment level for agent i. We refer to Z_i as the *individual treatment*. We then let $Y_i \in \mathscr{Y}$ be the observed agent's outcome, and $\mathbf{Z}$ and $\mathbf{Y}$ the corresponding vectors. For each unit i, the partition $(i, \mathscr{N}_i, \mathscr{N}_{-i})$ defines the following partitions of the treatment and outcome vectors: $(Z_i, \mathbf{Z}_{\mathscr{N}_i}, \mathbf{Z}_{\mathscr{N}_{-i}})$ and $(Y_i, \mathbf{Y}_{N_i}, \mathbf{Y}_{N_{-i}})$. Then, let $\mathbf{X}_i \in \mathscr{X}$ be a vector of K covariates for agent i. This represents the set of exogenous pre-treatment variables used to offset counfounding factors (unconfoundedness). It is worth noting that whenever the selection of neighborhood treatment is driven by the characteristics featured by both the agent and the neighborhood, unconfoundedness is achieved by decomposing $\mathbf{X}_i$ into two subvectors: individual covariates, denoted with $\mathbf{X}_i^{ind} \in \mathscr{X}^{ind}$, and neighborhood covariates, denoted with $\mathbf{X}_i^{neigh} \in \mathscr{X}^{neigh}$. While $\mathbf{X}_i^{ind}$ denotes the set of individual-level covariates, such as economic and social characteristics, $\mathbf{X}_i^{neigh}$ may include two types of neighborhood-level covariates: i) variables representing the structure of the neighborhood $\mathscr{N}_i$ (e.g. the degree N_i, the centrality, the reciprocity, the topology, etc.), and ii) variables representing the composition of the neighborhood $\mathscr{N}_i$, i.e., aggregational covariates summarizing individual attributes of nodes j.[3]

2.2. Potential outcomes and neighborhood interference

An essential element of the causal inference literature is the definition of potential outcomes, i.e., the potential value of the outcome variables that an agent would have under the different levels of the treatment (Imbens & Rubin, 2015). When no interference is at work, agent's potential outcome is assumed to depend only on the agent's own treatment. This assumption is referred to as Stable Unit Treatment Value Assumption (SUTVA) (Rubin, 1980), and standard PSM methods relies on this assumption to draw correct inference. Under interference however, we further consider the level of treatment received by the agent's neighborhood. Formally, this generalization of the potential outcomes violates SUTVA and requires the introduction of a new assumption.

In principle, the outcome observable at node i depends on the entire treatment assignment vector $\mathbf{Z}$ and can be written as $Y_i(\mathbf{Z})$. As pointed out by Rubin (1986), this potential outcome is well defined only if the following assumption holds:

Assumption 1 (No Multiple Versions of Treatment (Consistency)). *The mechanism used to assign treatments does not matter and assigning the treatments in a different way does not constitute a different*

[3]Specifically, this function takes the form of $h_i(\mathbf{X}_{\mathscr{N}_i}^{ind})$, where $\mathbf{X}_{\mathscr{N}_i}^{ind}$ is a $|\mathscr{X}^{ind}| \times N_i$-matrix collecting all the neighbors' individual covariates and $h_i(\cdot)$ is a function $h_i : (\mathscr{X}^{ind})^{N_i} \to \mathscr{H}_i$ summarizing the matrix $\mathbf{X}_{\mathscr{N}_i}^{ind}$ into a vector of dimension $|\mathscr{H}_i| < |\mathscr{X}^{ind}| \times N_i$. However, for the sake of simplicity, in the empirical application that follows we will only make use of the first type of neighborhood-level covariates.

treatment.

$$Y_i = Y_i(\boldsymbol{Z})$$

The consistency assumption is the first component of SUTVA (Rubin, 1980, 1986).

The second component of SUTVA rules out the presence of interference between units, assuming that the potential outcome of unit i only depends on the individual treatment Z_i. Given the incompatibility of this requirement with our framework, here we relax the no interference assumption and allow the existence of interference on a network. Formally, we replace the assumption of no interference with a new local interference assumption within the neighborhood.

Assumption 2 (First-Order Interference). *The potential outcome of agent i depends on the level of individual treatment, i.e., Z_i, and the level of treatment received by connected agents, i.e., $\boldsymbol{Z}_{\mathcal{N}_i}$:*

$$Y_i(\boldsymbol{Z} = \boldsymbol{z}) = Y_i(\boldsymbol{Z}_{\mathcal{N}_i} = \boldsymbol{z}_{\mathcal{N}_i})$$

Assumption 2 states that interference acts only within the immediate neighborhood: i.e. agent's direct connections in the network. Building on this assumption, we can formalize the dependence of agent i's outcome from the treatments received by neighboring agents through a specific summarizing function $g_i : \mathcal{Z}^{N_i} \to \mathcal{G}_i$, defined by Aronow & Samii (2017) as *exposure mapping* function. Specifically:

Assumption 3 (First-Order Interference with Exposure Mapping). *Given a function $g_i : \mathcal{Z}^{N_i} \to \mathcal{G}_i$, $\forall\, \boldsymbol{Z}_{\mathcal{N}_{-i}}, \boldsymbol{Z}'_{\mathcal{N}_{-i}}$ and $\forall\, \boldsymbol{Z}_{\mathcal{N}_i}, \boldsymbol{Z}'_{\mathcal{N}_i}$ such that $g_i(\boldsymbol{Z}_{\mathcal{N}_i}) = g_i(\boldsymbol{Z}'_{\mathcal{N}_i})$, the following equality holds:*

$$Y_i(Z_i, \boldsymbol{Z}_{\mathcal{N}_i}, \boldsymbol{Z}_{\mathcal{N}_{-i}}) = Y_i(Z_i, \boldsymbol{Z}'_{\mathcal{N}_i}, \boldsymbol{Z}'_{\mathcal{N}_{-i}})$$

Assumption 3 is most consolidated in the recent literature (Van der Laan, 2014) and in combination with Assumption 1 is referred to as Stable Unit Treatment on neighborhood Value Assumption (SUTNVA) (Forastiere et al., 2016).

Now let $G_i = g_i(\mathbf{Z}_{\mathcal{N}_i})$ and refer to G_i as *neighborhood treatment*. Depending on the exposure function $g_i(\cdot)$, G_i can be either univariate or multivariate. Here, as in Forastiere et al. (2016), we consider neighborhood treatments of the form

$$G_i = \frac{1}{N} \sum_j a_{ij} Z_j \tag{1}$$

where a_{ij} is the entry of matrix A registering the level of interaction between i and j. A desirable feature of Assumption 3, with G_i defined as in (1), is that it allows for the existence of heterogeneous network effects, since the level of first-order interference depends on the position of the agent in the network. An interesting property of this formulation is that it can be used to take into consideration both direction and intensity of

the interaction between agents (i.e. weighted directed networks).

Given this framework, each node can be represented as subject to two treatments: the *individual treatment*, Z_i, and the *neighborhood treatment*, G_i. It follows that potential outcomes can be indexed by a joint treatment: $Y_i(z,g)$. This – which is the simplified expression for $Y_i(Z_i = z, G_i = g)$ – can be seen as the potential outcome for node i under treatment z, in a situation where agent i is exposed to the neighborhood treatment g through connected agents.

Because of the continuous individual treatment and the bivariate joint treatment, the potential outcome of unit i $Y_i(z,g)$ as a function of the individual treatment z and the neighborhood treatment g can be seen as a dose-response function. We define the marginal mean of the potential outcome $Y_i(z,g)$, for each value of z and g as the average dose-response function (aDRF), denoted by $\mu(z,g)$. Formally, let

$$\mu(z,g) = E[Y_i(z,g)] \tag{2}$$

which can be marginalized to get the univariate average dose-response functions

$$\mu^Z(z) = \int_g E[Y_i(z,g)]f(g)dg \quad \text{and} \quad \mu^G(z) = \int E[Y_i(z,g)]f(z)dz \tag{3}$$

where $f(g)$ and $f(z)$ are the observed marginal distributions of the neighborhood and individual treatments. The univariate average dose-response functions allow to define direct effects of the treatment as any comparison $\tau(z,z') = \mu^Z(z) - \mu^Z(z')$, or as the first derivative of the average dose-response function $\tau(dz) = \frac{d\mu^Z(z)}{dz}$. Similarly, spillover effects can be defined as the difference between the average potential outcome corresponding to two different levels of the neighborhood treatment g and g': $\delta(g,g') = \mu^G(g) - \mu^G(g')$, or as the first derivative of the average dose-response function $\delta(dg) = \frac{d\mu^G(g)}{dg}$.

Following the assumption of interference through exposure mapping defined by $g_i(\cdot)$, the identification of the dose-response function $\mu(z,g)$ relies on the unconfoudedness assumption of the joint treatment, that is the conditional independence between the joint treatment and potential outcomes: $Y_i(z,g) \perp\!\!\!\perp Z_i, G_i \mid \mathbf{X}_i, \forall z, g, \forall i$ (Forastiere et al., 2016).

2.3. Joint Propensity Score-Based Estimator

As previously mentioned, in this paper we adopt a JPS-based estimator of the average dose-response function $\mu(z,g)$, which extends the propensity score method proposed by Forastiere et al. (2016) to the case of continuous individual treatment. We define the *joint propensity score* $\psi(z;g;x)$ as the probability of being subject to direct treatment z and being exposed to a weighted average of the treatments if the agent's

connections equal to g, given characteristics $\mathbf{X}_i = x$:

$$\begin{aligned}\psi(z;g;x) &= P(Z_i = z, G_i = g|\mathbf{X}_i = x) \\ &= P(G_i = g|Z_i = z, \mathbf{X}_i = x)P(Z_i = z|\mathbf{X}_i = x)\end{aligned} \tag{4}$$

We denote the probability of neighborhood treatment at level g conditional on a specific value z of the individual treatment, and on the vector of covariates $\mathbf{X}_i$: i.e., $P(G_i = g|Z_i = z, \mathbf{X}_i = x)$, with $\lambda(g;z;x)$ and refer to it as *neighborhood propensity score*. Similarly, we denote the probability of individual treatment at level z conditional on covariates: i.e., $P(Z_i = z|\mathbf{X}_i = x)$, with $\phi(z;x)$, and we refer to it as the *individual propensity score*. Given the factorization of the *joint propensity score* into the product of the *individual propensity score* and *neighborhood propensity score* (4), we could use a generalized propensity score approach (Hirano & Imbens, 2004) on both propensity scores to adjust for confounding covariates $\mathbf{X}_i$.

In this framework, the observed data, $\{Y_i, Z_i, G_i, \mathbf{X}_i\}$, can be used to predict for each unit the potential outcome $Y_i(z,g)$ at a specific level of joint treatment. It is then possible to obtain the average dose-response function $\mu(z,g)$ by averaging the potential outcomes over all units, $\widehat{\mu}(z,g) = \frac{1}{N}\sum_{i=1}^{N}\widehat{Y}_i(z,g)$.

2.4. Estimation Procedure

In what follows, we outline the estimating procedure for the average dose-response function $\mu(z,g)$.

We first posit models for the individual treatment, the neighborhood treatment and the outcome. Consider the following general models:

$$Z_i \sim f^Z(\mathbf{X}_i;\theta^Z) \tag{5}$$

$$G_i \sim f^G(Z_i, \mathbf{X}_i;\theta^G) \tag{6}$$

$$Y_i(z,g) \sim f^Y(z,g,\phi(z;\mathbf{X}_i),\lambda(z;g;\mathbf{X}_i);\theta^Y) \tag{7}$$

According to the models in (5), (6), and (7), the estimation procedure requires the following steps:

1. Estimate the parameters θ^Z and θ^G of the models for the individual propensity score $\phi(z;\mathbf{X}_i)$ and the neighborhood propensity score $\lambda(z;g;x)$;

2. Use the estimated parameters in Step 1 to predict for each unit $i \in N$ the actual individual propensity score $\widehat{\Phi}_i = \phi(Z_i;\mathbf{X}_i)$ and the actual neighborhood propensity score $\widehat{\Lambda}_i = \lambda(G_i;Z_i;\mathbf{X}_i)$, that is, the probabilities for unit i of receiving the individual treatment Z_i and being exposed to the neighborhood treatment G_i, where Z_i and G_i are the values that were actually observed;

3. Combining the observed data $\{Y_i, Z_i, G_i, \mathbf{X}_i\}$, and the predicted propensity scores $\widehat{\Phi}_i$ and $\widehat{\Lambda}_i$, estimate the parameters θ^Y of the outcome model in (7);

4. For each level of the joint treatment $(Z_i = z, G_i = g)$, predict for each unit $i \in \mathcal{N}$ the individual and the neighborhood propensity score corresponding to that level of the treatment, i.e., $\phi(z; \mathbf{X}_i)$ and $\lambda(z; g; \mathbf{X}_i)$, and use these predicted values to impute the potential outcome $Y_i(z, g)$.

5. To estimate the average dose-response function $\mu(z, g)$, for each level of the joint treatment take the average of the potential outcomes over all units

$$\widehat{\mu}(z, g) = \frac{1}{N} \sum_{i=1}^{N} \widehat{Y}_i(z, g)$$

Standard errors and 95% confidence intervals can be derived using bootstrap methods (Efron, 1979; Forastiere et al., 2016).

3. Empirical application

The analysis of trade distortions in agricultural markets provides an interesting environment to illustrate how our methodology works in practice for two main reasons.

First, over the last century, the agricultural sector has been subjected to some of the most heavy-handed governmental interventions which accounted for an estimated 70 percent of the global welfare cost in 2004 (Anderson et al., 2010). Despite the successful conclusion of the Uruguay Round Agreement on Agriculture (URAA) in 1994 that helped to reduce distortions, countries still prefer to regulate agricultural markets and subsidize farmers (Carter and Steinbach, 2018). The reason is that preventing losses in a sector with such significant presence (in terms of output, employment, etc.) may loom large in the government's objective function (Marvel and Ray, 1983; Trefler, 1993; Freund and Ozden, 2008).[4]

Second, following the emergence of the so-called agri-food global value chains (GVCs), agricultural markets in different countries have become dramatically interconnected (Johnson & Noguera, 2017). As Balié et al. (2018) points out, global linkages in agriculture and food sectors have been increasing over time thereby challenging the way policy makers establish their trade policies.

As a matter of fact, policy interventions which, in the first place were aimed at shielding the domestic market from unfavorable events in foreign economies, have eventually created spillover effects, thus exacer-

[4]For instance, in Africa the agriculture sector still generates about 25% of the gross domestic product (GDP), or 50% if we look at the broader agribusiness sector, and involves roughly 65% of the local population (Balié et al., 2018).

bating tensions in the world market (Gouel, 2016; Bayramoglu et al., 2018; Beckman et al., 2018).[5] This makes harder for governments to achieve their redistribution objectives, particularly when considering food security issue. The latter has been newsworthy after the 2008 food crisis, as riots erupted in many developing countries, and received particular attention from academics and policy makers ever since (Larochez-Dupraz and Huchet-Bourdon, 2016; Magrini et al., 2017).

Motivated by these issues, we investigate whether and how the increasing interconnected structure of food and agriculture markets mitigates or exacerbates the effects of agricultural distortions on food security. While there is an intense debate over the effects that openness to trade in the primary sector has had on consumers' welfare, such as, on income during a famine in India (Burgess and Donaldson, 2010), on health in Mexico (Giuntella et al., 2017) and on labor market distortions (Tombe, 2015), to the best of our knowledge, there are few investigations into the causal relationship between trade distortions and food security (Magrini et al., 2017) but none takes into account spillover effects.

3.1. Data

The empirical analysis requires four sets of data: i) the treatment variable measuring the level of distortions, ii) a set of observed country's characteristics, iii) an outcome measuring food security, and iv) a network describing the trade linkages between countries.

Following Anderson and Nelgen (2012a,b), here we assess the intensity of distortions using the Nominal Rate of Assistance (NRA), which measures the percentage by which government policies have raised (lowered) gross returns to farmers above (below) what they would be without the government's intervention. NRA is used in the guise of treatment (i.e. set i). It is pivotal to testing our methodology in that it is a continuous measure accounting for both traditional policy instruments (such as tariffs, export subsidies, and import quotas), and the additional measures untamed by the URAA such as trade remedies.[6] For ease of interpretation, we shift the support of the treatment (NRA + 1), which is known as Nominal Assistance Coefficient (NAC). Therefore, for any given country, a NAC>1 signals the presence of a support for agricultural producers, while a NAC<1 indicates disincentives in the agricultural sector. Figure 1 shows that, while richer countries are decreasing their policy support to farmers, developing countries (DC) switched from taxing agricultural production to applying protectionist measures, often exceeding the level of support provided by OECD countries (Swinnen et al., 2012).[7]

[5] Giordani et al. (2016) document the existence of a "multiplier effect" that can take place if either the country imposing the restricting measure is a large exporter of the good (i.e., it has market power in the global market), as its export volumes fall, or if many small exporters apply such measures.

[6] It is worth mentioning, however, that policies such as direct subsidies and input taxes, contribute only marginally to these distortions. Trade policies, such as export bans and import tariffs, instead still account for 60% of the NAC at the global level (Anderson et al., 2013).

[7] Anderson and Nelgen (2012a,b) provide a detailed explanation on the method used to develop the NRA and on the

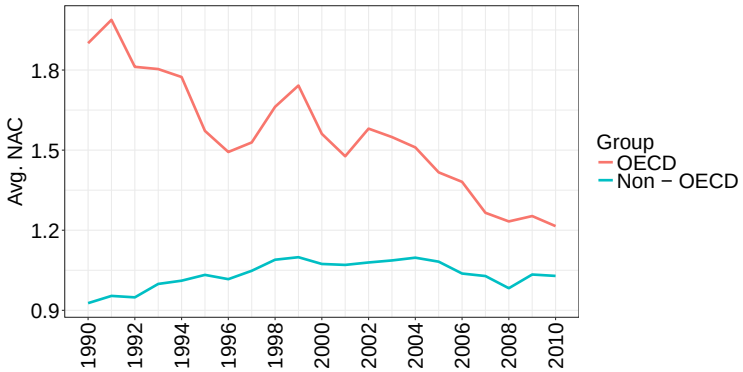

Figure 1: NAC values in OECD (red) and Non-OECD (blue) countries from 1990 to 2010

The covariates selected to explain the level of distortions (i.e., set *ii*) are borrowed from the agricultural and trade policy literature (Anderson et al., 2013; Magrini et al., 2017). Specifically, we consider: real per capita GDP and its squared and cubic terms to control for trade behaviors and its non-linearities; total population and its squared term as a proxy of the country size; per-capita arable land and the agricultural total factor productivity growth index to assess the country's relative agricultural comparative advantage; the ratio of food imports to total exports, net food exports, and absolute (positive and negative) percentage deviations from the trend in international food prices as a measure of country's access to, dependence from and position in the global market; the international food price volatility index to capture country's response to changes in price levels. Finally, we include a dummy capturing the effect of the food crisis of 2007-2008 and a set of regional dummies to control for unobservable characteristics of African, Asian, European transition, Latin American, and high income countries.

Food security (i.e., set *iii*) is measured as the level of food availability, i.e. the supply of food commodities in kilo-calories per person. Accordingly, consumers are better off when this measure is maximized. As robustness checks, we also analyze food access, food utilization and food stability, proxied by the depth of food deficit, the prevalence of anemia among children aged under five and per capita food supply variability, respectively (CFS, 2009).[8]

Finally, the network (i.e., set *iv*) is built using the value of bilateral agri-food trade in each given year (Figure 2).[9] Summary statistics are reported in Table A.2. For additional information on data sources, see

interpretation of NAC.

[8]Consequently, consumers are better off when the measure of food stability is maximized, while food access and utilization are minimized.

[9]The time window considered goes from 1990 to 2010. Furthermore, it is worth emphasizing that in Magrini et al. (2017), to avoid the risk of interference, the countries most likely to generate or be affected by policy spillovers, i.e. the top global exporters and importers, namely the United States, Germany, France, Italy, Spain, the Netherlands, Belgium, China, Brazil, Canada, Japan and the UK are excluded from the analysis. By contrast, we keep these countries, as it is our interest to account for interference. As a result, we end up with a sample of 74 countries (see Figure A.6).

Table A.3 in Appendix.

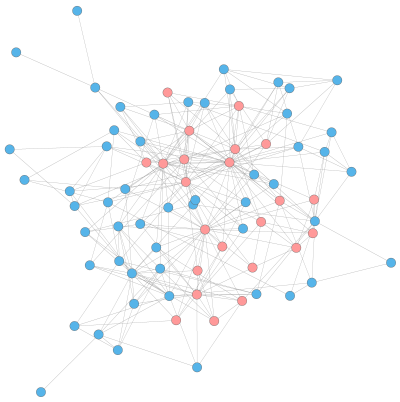

Figure 2: International Trade Network in 2010. Only top 5 incoming flows are displayed for OECD (red) and Non-OECD (blue) countries

3.2. Model Setup

The general form of the JPS presented in section 2 allows to test several economic mechanisms underlying externalities. For the purpose of this empirical application, we assume that the extent to which the level of distortions of a country j affects country i depends on the value of its bilateral agri-food exports, normalized by the average world trade value. Consequently, equation 1 is as follows:

$$G_i = \frac{1}{NS}\sum_j a_{ij}Z_j \qquad \text{where} \quad S = \frac{\sum_k \sum_j a_{kj} I(a_{kj} \neq 0)}{\sum_k \sum_j I(a_{kj} \neq 0)} \tag{8}$$

In addition, we leverage the information provided by the observation of the network structure over time, i.e. $\{A^{1990}, \ldots, A^{2010}\}$, by condensing the relations described in A^t matrices in a block-diagonal fashion. As a result, we obtain the square adjacency matrix A where the generic element $a_{ij} = 0$ if i and j: i) refer to the same node, or ii) have no relation at time t, or iii) indicate observations at different points in time.[10]

Following the estimating procedure for the average dose-response function of Section 2.4, in this empirical application, we first apply a zero-skewness Box-Cox transformation (Box and Cox, 1964) to the vector of treatment, $Z^\star = (Z^k - 1)/k$, [11] and assume the following normal model for $Z^\star$:

$$Z^\star \sim N(\alpha_Z + \beta_Z^T \mathbf{X}_i, \sigma_Z) \tag{9}$$

[10] This requires independence between observations across years, a common assumption in most spatial applications (Anselin et al., 2008).

[11] Note that k is chosen so that the skewness of the transformed variable is zero.

where X_i contains the the set of covariates relative to agent i (i.e., $\mathbf{X}_i^{ind}$). [12]

Similarly, we assume the following model for the neighborhood treatment G_i

$$G_i \sim N(\alpha_G + \beta_G^T \mathbf{X}_i + \beta_{GZ}^T Z_i, \sigma_G) \tag{10}$$

where G_i follows a normal distribution with mean $\alpha_G + \beta_G^T \mathbf{X}_i$ and variance σ_G.

Finally, we can postulate a normal model for the outcome given the propensity scores:

$$Y_i(z,g) \sim N\big(q\big(z,g,\phi(z;\mathbf{X}_i),\lambda(z;g;\mathbf{X}_i)\big),\sigma_Y\big) \tag{11}$$

where $q(\cdot)$ is the sum of cubic polynomials and their interactions. We also include in $q(\cdot)$ an interaction term between the country NAC and the network NAC. This allows the direct effect of national policies to vary depending on the policies implemented in partner countries, and the spillover effects to vary depending on the country NAC.

3.3. Results

Table 1 reports the estimated parameters of the models for the individual treatment Z_i (Direct NAC) and the neighborhood treatment G_i (Network NAC). The set of covariates used in these models, i.e, $\mathbf{X}_i$, coincides with those described in Section 3.1.

Propensity scores are instrumental to fit the outcome model. Nonetheless, the estimated parameters already provide relevant insights on the relationship between country characteristics, the direct NAC and the level of NAC in partner countries. According to Table 1, a country with a high per capita GDP is likely to provide higher support to its agricultural sector (second column) and have commercial partners with low NAC (third column). Moreover, the table shows that a comparative advantage in the agricultural sector, as measured by per capita arable land, has a negative effect on the intensity of both national and neighborhood policy interventions. Specifically, results suggests that when countries present a high comparative advantage, they tend to increase the taxation of revenues, both at a local (second column) and at a neighborhood level (third column). A U-shaped relationship is then found between the level of country NAC and its dimension, as proxied by population size, while the opposite is true for the network NAC. When the local demand is either very low or very high, governments tend to increase their support to stimulate agri-food production (second column). Partner countries' NAC instead rises with the country's size, although at a decreasing rate, to take advantage of its dimension (third column). Furthermore, we find evidence of a higher level of NAC for those countries which heavily rely on imports, while those with higher values for net food exports tend

[12] The implicit assumption is that self-selection mechanisms are driven by the characteristics of the network structure, but not by neighborhood characteristics.

Table 1: Individual and neighborhood propensity scores (Dep Vars: Direct NAC and Network NAC)

	$\phi(z;x^z)$ (Eq. 5)	$\phi(g;z;x^g)$ (Eq. 6)
(l1.ln) pc real gdp	0.139 (0.352)	-9.847^{***} (3.121)
(l1.ln) pc real gdp^2	-0.020 (0.043)	1.109^{***} (0.379)
(l1.ln) pc real gdp^3	0.001 (0.002)	-0.037^{**} (0.015)
(l1.ln) pc arable land	-0.046^{***} (0.006)	-0.111^{**} (0.055)
(l1.ln) pop	-0.133^{*} (0.069)	1.524^{**} (0.610)
(l1.ln) pop^2	0.004^{**} (0.002)	-0.033^{*} (0.018)
(l1) agr. tfp	-0.001^{***} (0.0003)	0.002 (0.003)
(l1) food imp/tot exp	0.009 (0.023)	-0.410^{**} (0.206)
(l1) food imp/tot exp^2	0.008 (0.005)	-0.162^{***} (0.045)
(l1) net exp	-0.017^{***} (0.002)	0.117^{***} (0.018)
(l1) pos dev food	-0.174 (0.141)	-1.768 (1.254)
(l1) neg dev food	-0.262^{**} (0.131)	-2.580^{**} (1.165)
food volatility	-2.926^{***} (1.095)	-21.886^{**} (9.731)
food crisis	-0.033^{*} (0.017)	-0.091 (0.154)
Z		-1.229^{***} (0.205)
Constant	0.754 (1.191)	11.913 (10.562)
Observations	930	930
R^2	0.538	0.464
Adjusted R^2	0.529	0.453
Residual Std. Error	0.125 (df = 911)	1.107 (df = 910)
F Statistic	58.978^{***} (df = 18; 911)	41.488^{***} (df = 19; 910)
Regional dummies	Yes	Yes

Note: $^{*}p<0.1$; $^{**}p<0.05$; $^{***}p<0.01$. l1 stands for one year lag.

to provide less protection (second column). The opposite is true for their partner countries (third column). NAC is also negatively correlated with both positive and negative deviations of international prices from their trend and food volatility suggesting that, when price spikes, governments tend to stabilize their domestic markets by imposing restrictions on exports and lowering protection on imports. The negative and significant coefficient on food crisis confirms the pattern whereby the support is lower during food crisis years. Finally, the direct NAC is negatively correlated with the network NAC, suggesting that, country characteristics being held constant, the higher the level of support in the country the lower will be that of partner countries.

Following steps 2 and 3, we predict the individual and neighborhood propensity scores, i.e. the probability of observing the actual level of NAC in a country and in its partner countries given country characteristics. Propensity scores are then used to estimate the conditional expectation of the outcome given by the model (7).[13] The estimated coefficients are reported in Table A.6 in the Appendix while, for the sake of space, in the remaining of this section we focus on the analysis of the dose-response functions. These are obtained following steps 4 and 5 of the methodology for each pair of values of the direct NAC *(z)* and network NAC *(g)*. That is, we first predict the probability of observing these values for each country, then we use the individual

[13]Since we make use of nonlinear functions of the individual and network NAC, model (7) implies a nonlinear functional form on the direct and network NAC.

and neighborhood propensity scores to predict the country-level outcomes corresponding to *Y(z,g)*. The dose-response function is given by the average outcomes across all countries against the values of direct NAC *(z)* and network NAC *(g)*.

We first report in Figure 3 the marginal aDRF for the food availability outcome when neglecting interference, i.e., when we do not include neither the network NAC nor the neighborhood propensity score in the outcome model. Then, in Figure 4 we report the marginal aDRF $\mu^Z(z)$ (left panel) and $\mu^G(z)$ (right panel) when interference is taken into account. By comparing the aDRF $\mu^Z(z)$ and the coefficients of the outcome models (Tables A.5 and A.6) neglecting or not interference, we obtain meaningful information on the extent and direction of the bias when considering spillover effects.[14] Specifically, Figure 3, which represents the average dose-response function of the NAC neglecting interference, shows that the highest level of food availability is registered when NAC is about 48%, i.e. when governments provide a limited support to their agricultural sector. Moreover, marginal benefits are also obtained by eliminating residual taxation and moving to limited support, i.e. when NAC values range from 0.9 to 1.48. By contrast, negative effects are produced in cases of strong incentives (i.e., NAC higher than 1.48) or disincentives (i.e., NAC lower than 0.9). This first result shows that: i) in line with Anderson et al. (2013), taxing agricultural producers to obtain additional resources for investment in more dynamic sectors comes at a cost of lower food availability and therefore welfare[15]; ii) a strong support of the primary sector may result in a protection of inefficient domestic producers or crop varieties which, through a lower productivity, negatively affects welfare (Tombe, 2015).

Then, by comparing the above result with that of Figure 4, we observe that, when ignoring interference, the impact of national policies ($\phi(z;\mathbf{X}_i^z)$) is overestimated by about 20%. The left panel of Figure 4, which represents the average dose-response function of the direct NAC $\mu^Z(z)$ when interference is taken into account and marginalized over, shows indeed that the highest benefit in terms of food supply is registered when NAC value is equal to 1.67. That is to say, in order to be effective in an interconnected world, domestic policies may require additional efforts.

Finally, the dose-response estimator allows us to assess also the spillover effects of policy interventions in partner countries. The right panel of Figure 4, representing the average dose-response function of the network NAC $\mu^G(g)$, shows that as a result of the emergence of agri-food GVCs, it is crucial to take into account partner policies when determining the optimal level of a domestic intervention, as these can either

[14] In the Appendix we further present: Table A.5; Figures A.8, A.11 and A.14; the bivariate aDRF $\mu(z,g)$ (Figures A.9, A.12 and A.15,); and Figures A.7, A.10 and A.13.

[15] Anderson et al. (2013) shows that taxation affects both producers and consumers. For producers, it reduces profits and incentives to respond to market signals. For consumers, if taxation discourages farming activity, it can negatively affects demand for farm labor and wages for unskilled workers in both farm and non-farm jobs.

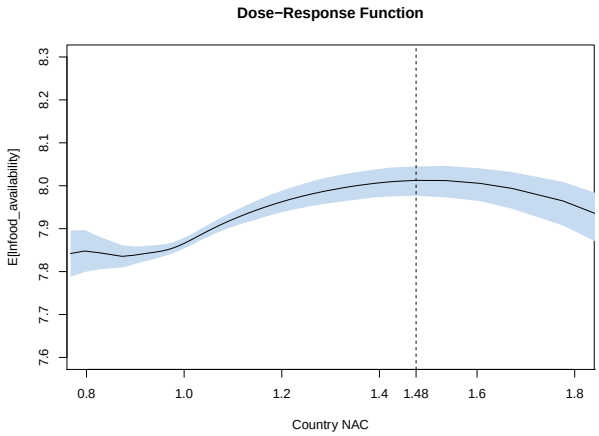

Figure 3: Dose-response function $E[Y_i(z)]$ of direct NAC on food availability (log scale) neglecting interference

boost or counteract the effect of local measures. Specifically, we observe that higher levels of domestic food supply are reached when trading partners provide incentives to their own agricultural producers. This is straightforward as we bear in mind that a producer support may boost exports and therefore food availability in the importing (target) country.

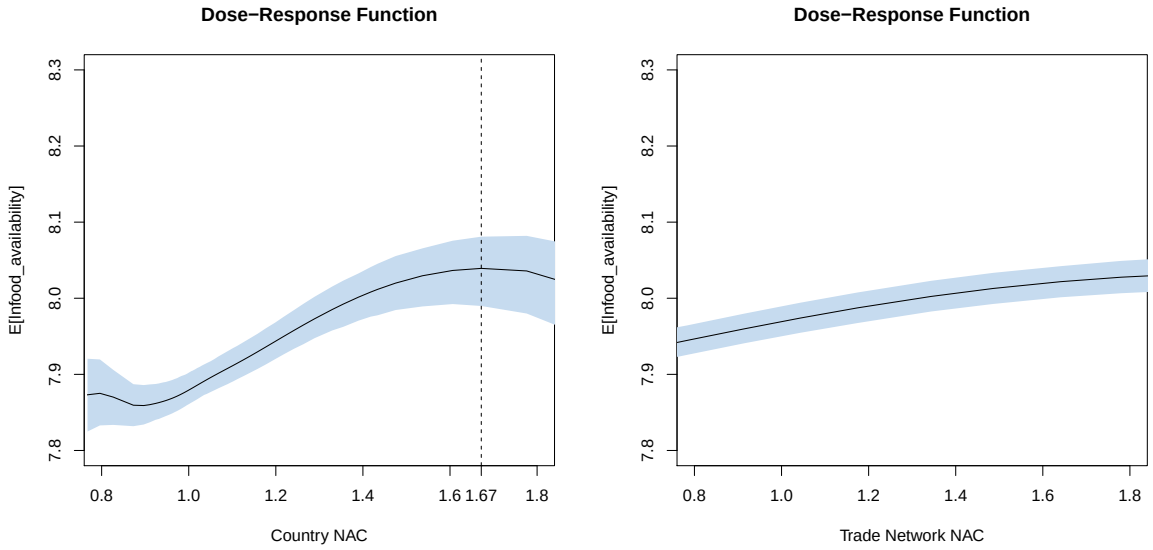

Figure 4: Marginal dose-response function $\mu^Z(g)$ of direct NAC (left) and marginal dose-response function $\mu^G(z)$ of network NAC (right) on food availability (log scale)

The effect of the correlation between domestic and foreign distortions - mediated by the trade network - is made clearer when we look at Figure 5, which represents the bivariate aDRF $\mu(z, g)$. Even if governments were able to maximizes their objective functions in order to reach the highest level possible of welfare, policies

implemented in partner countries may shift the supply of food above the desired level.

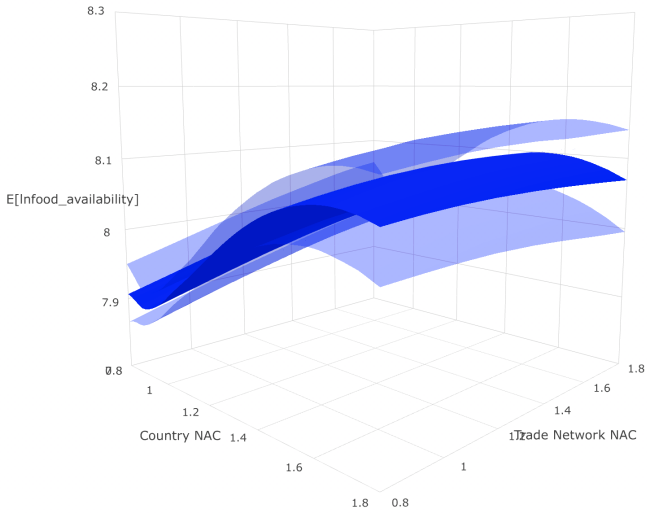

Figure 5: Average dose-response function $\mu(z, g)$ of direct NAC and network NAC on food availability (log scale)

Similar results are found for another dimension of food security, namely food utilization (Figure A.8). Consumers are better off for minimum levels of the intervention (i.e. when NAC is about 1.36), but again this optimal level of support is underestimated when considering interference. As for food stability (Figure A.11), interference does not appear as a significant source of concern for the domestic government, as the highest possible benefit is reached when the support is about 75% in both cases. Finally, the results relative to food access are less reliable due to wider confidence intervals (Figure A.14).

4. Conclusions

Causal inference in observational studies has often neglected the presence of interference, which has proven to be pervasive in economics and social sciences. By developing a JPS extended to the case of continuous treatment, this paper provides a methodology to evaluate policies when externalities matter.

Specifically, we develop a generalized propensity score-base estimator that corrects for the bias resulting from both treatment selection and continuous treatment interference, balancing individual and neighborhood covariates across units under different levels of individual treatment and of exposure to neighbors' treatment.

The empirical relevance of our methodology is illustrated through the evaluation of the effects of distortions in agricultural markets. Our results show that, on the one hand, policy interventions matter and they have a non-linear impact on food security. In particular, both a local excessive taxation and support for the primary sector is detrimental for food availability. On the other hand, the average direct effect that

is estimated in the existing literature underestimates the optimal level of producers' support. Interference indeed accounts for a bias of some 20% of the optimal support.

The correlation between local and foreign policies - mediated by the trade network - points to new directions of research. For example, this finding may provide interesting insights to assess the indirect effects of policy changes coordinated at the supranational level, as in the case of the Single Farm Payment implemented in 2003 under the Common Agricultural Policy of the European Union (EU). This policy intervention consisted in detaching farmers' income payments from the production of specific crops, in order to increase the flexibility of farm decision making and ultimately reduce the level of EU intervention. Thus, the framework provided in this paper would have helped in assessing its indirect consequences on EU partner countries.

However, although this paper makes a concrete step forward in causal inference studies accounting for network structures, a serious limitation lies on the assumption that limits spillover effects to first-order network neighbors. While this simplifying assumption is common in the literature, the plausible presence of higher-order externalities might bias the estimates of both direct and first-order spillover effects. Moreover, disentangling different orders of these effects can be of interest in many social contexts. Possible extensions in this direction are a promising avenue for future research.

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Appendix A. Figures and Tables

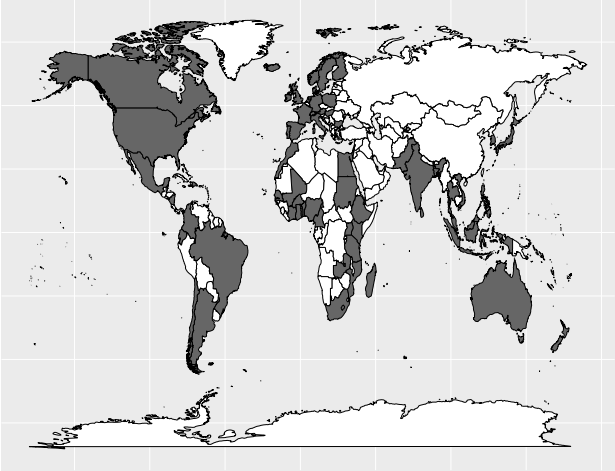

Figure A.6: Map of the World. Countries included in the sample are indicated by the color grey

Table A.2: Summary statistics of outcomes, covariates and treatment variable

	Variable	Mean	St. Dev.	Max	Min
Outcomes	food availability	2,772.66	453.82	3,522	2,046
	food access	143.16	76.39	289.00	22.00
	food utilization	36.08	22.15	79.00	10.50
	food variability	36.79	14.58	70.00	16.00
Covariates	pc real gdp	13,883.05	18,126.58	91,617.28	302.13
	pc arable land	0.32	0.39	2.81	0.03
	pop (/100)	680,600.84	1,585,192.15	12,309,806.91	3,174.14
	agr tfp	113.28	15.97	180.44	83.47
	food imp/tot exp	0.13	0.18	1.94	0.01
	net exp	1.66	2.38	24.61	0.00
	pos dev food	0.01	0.03	0.15	0.00
	neg dev food	0.05	0.04	0.12	0.00
	food volatility	0.01	0.00	0.02	0.00
	food crisis	0.10	0.29	1.00	0.00
Treatment	NAC (Z)	1.14	0.26	2.21	0.77
Network	Trade value	0.92	1.50	10.27	0.00

Table A.3: Data sources

	Variable	Source
Outcome	food access	World Bank - World Development Indicators
	food availability	FAO - Food Balance Sheets
	food utilization	World Bank - World Development Indicators
	food variability	FAOSTAT
Covariates	pc real gdp	World Bank - World Development Indicators
	pc arable land	World Bank - World Development Indicators
	pop	World Bank - World Development Indicators
	agr tfp	United States Department of Agriculture - Economic Research Service
	food imp/tot exp	FAOSTAT
	net exp	FAOSTAT
	pos dev food, neg dev food, food volatility	World Bank - GEM Commodity Price Data
	Regional group dummies	World Bank dataset (Anderson and Nelgen, 2012b)
	food crisis	Authors calculation
Treatment	NAC (Z)	World Bank dataset (Anderson and Nelgen, 2012b)
Network	trade value	FAOSTAT (https://fao.org/)

Table A.4: NAC distribution by country

Country	Mean	St. Dev.	Max	Min	Country	Mean	St. Dev.	Max	Min
ARG	0.87	0.11	1.00	0.70	KEN	1.01	0.09	1.16	0.69
AUS	1.02	0.02	1.06	1.00	KOR	2.28	0.35	2.78	1.52
AUT	1.42	0.23	1.82	1.07	LKA	1.03	0.11	1.19	0.85
BEL	1.30	0.12	1.54	1.10	LTU	1.16	0.26	1.65	0.55
BEN	0.99	0.02	1.01	0.93	LVA	1.18	0.27	1.73	0.55
BFA	0.98	0.03	1.02	0.90	MAR	1.65	0.15	1.98	1.45
BGD	0.93	0.12	1.06	0.63	MDG	1.00	0.10	1.24	0.90
BGR	0.95	0.14	1.18	0.64	MEX	1.14	0.13	1.41	0.85
BRA	1.01	0.09	1.11	0.80	MLI	0.98	0.02	1.02	0.94
CAN	1.21	0.09	1.43	1.08	MOZ	1.04	0.06	1.24	0.95
CHE	2.77	0.83	4.32	1.48	MYS	0.99	0.05	1.06	0.87
CHL	1.07	0.03	1.10	1.01	NGA	1.00	0.08	1.22	0.87
CHN	1.03	0.13	1.27	0.71	NIC	0.90	0.08	1.05	0.73
CIV	0.76	0.05	0.82	0.68	NLD	1.39	0.17	1.62	1.08
CMR	0.99	0.01	1.01	0.96	NOR	2.68	0.61	3.67	1.63
COL	1.17	0.10	1.34	0.96	NZL	1.02	0.01	1.06	1.00
CZE	1.21	0.12	1.48	1.07	PAK	0.96	0.08	1.12	0.78
DEU	1.39	0.20	1.79	1.07	PHL	1.19	0.14	1.41	0.87
DNK	1.34	0.18	1.70	1.06	POL	1.18	0.14	1.60	0.98
DOM	1.07	0.15	1.43	0.76	PRT	1.27	0.11	1.44	1.08
ECU	0.93	0.14	1.22	0.70	RUS	1.14	0.19	1.42	0.55
EGY	0.97	0.07	1.10	0.84	SDN	0.82	0.29	1.47	0.31
ESP	1.28	0.14	1.56	1.08	SEN	0.98	0.12	1.23	0.83
EST	1.10	0.18	1.41	0.62	SVK	1.24	0.12	1.43	1.07
ETH	0.86	0.19	1.27	0.50	SVN	1.56	0.29	2.06	1.09
FIN	1.58	0.47	2.54	1.07	SWE	1.46	0.30	2.13	1.07
FRA	1.37	0.22	1.88	1.06	TCD	0.99	0.01	1.01	0.96
GBR	1.42	0.21	1.88	1.09	TGO	0.98	0.02	1.00	0.93
GHA	0.98	0.03	1.05	0.92	THA	1.00	0.06	1.14	0.90
HUN	1.20	0.12	1.45	1.07	TUR	1.25	0.11	1.43	1.01
IDN	1.01	0.11	1.20	0.78	TZA	0.85	0.15	1.12	0.50
IND	1.08	0.11	1.26	0.88	UGA	0.96	0.08	1.02	0.76
IRL	1.57	0.26	2.05	1.08	UKR	0.93	0.15	1.13	0.54
ISL	2.76	0.86	4.94	1.62	USA	1.12	0.04	1.18	1.04
ITA	1.29	0.15	1.57	1.07	ZAF	1.05	0.07	1.21	0.93
JPN	2.08	0.25	2.72	1.68	ZMB	0.94	0.00	0.94	0.94
					VNM	1.13	0.15	1.32	0.88

Note: Country names are denoted using ISO3 Code.

Table A.5: Coefficients of the outcome models neglecting interference

	Food Availability	Food Utilization	Food Variability	Food Access
z	2.418^{***} (0.817)	-5.855^{*} (2.986)	-10.145^{***} (2.337)	-7.443 (12.357)
z^2	-1.251^{**} (0.596)	2.915 (2.203)	7.850^{***} (1.712)	9.389 (9.641)
z^3	0.158 (0.137)	-0.277 (0.512)	-1.836^{***} (0.394)	-3.282 (2.427)
$\phi(z;\mathbf{X}_i^z)$	-0.327^{***} (0.079)	0.386 (0.281)	0.670^{***} (0.223)	0.959^{**} (0.463)
$\phi(z;\mathbf{X}_i^z)^2$	0.090^{**} (0.045)	0.141 (0.172)	-0.198 (0.126)	0.054 (0.272)
$\phi(z;\mathbf{X}_i^z)^3$	-0.018^{**} (0.008)	0.005 (0.033)	0.032 (0.023)	0.004 (0.045)
$z*\phi(z;\mathbf{X}_i^z)$	0.151^{***} (0.019)	-0.797^{***} (0.074)	-0.234^{***} (0.061)	-1.212^{***} (0.259)
Constant	6.703^{***} (0.342)	6.857^{***} (1.239)	7.290^{***} (0.974)	6.333 (4.980)
Observations	930	952	952	571
R^2	0.353	0.445	0.038	0.092
Adjusted R^2	0.348	0.441	0.031	0.081
Residual Std. Error	0.134 (df = 922)	0.499 (df = 944)	0.399 (df = 944)	0.686 (df = 563)
F Statistic	71.953^{***} (df = 7; 922)	108.282^{***} (df = 7; 944)	5.337^{***} (df = 7; 944)	8.143^{***} (df = 7; 563)

Note: *p<0.1; **p<0.05; ***p<0.01

Table A.6: Coefficients of the outcome models

	Food Availability	Food Utilization	Food Variability	Food Access
z	1.617^{**} (0.705)	-2.086 (2.365)	-9.490^{***} (2.553)	4.853 (11.918)
z^2	-0.799 (0.518)	0.371 (1.759)	7.334^{***} (1.884)	-0.923 (9.292)
z^3	0.106 (0.120)	0.187 (0.413)	-1.720^{***} (0.437)	-0.620 (2.336)
$\phi(z;\mathbf{X}_i^z)$	-0.243^{***} (0.065)	0.155 (0.213)	0.684^{***} (0.224)	0.629 (0.447)
$\phi(z;\mathbf{X}_i^z)^2$	0.072^{*} (0.037)	0.200 (0.130)	-0.241^{*} (0.126)	0.148 (0.258)
$\phi(z;\mathbf{X}_i^z)^3$	-0.012^{*} (0.007)	-0.026 (0.025)	0.037 (0.023)	-0.016 (0.043)
$z*\phi(z;\mathbf{X}_i^z)$	0.081^{***} (0.017)	-0.483^{***} (0.060)	-0.157^{**} (0.064)	-1.034^{***} (0.255)
g	0.121^{***} (0.029)	-0.612^{***} (0.092)	0.074 (0.097)	-0.972^{**} (0.400)
g^2	-0.016^{**} (0.006)	0.125^{***} (0.022)	0.015 (0.018)	0.194 (0.120)
g^3	0.001^{***} (0.0004)	-0.011^{***} (0.002)	-0.002 (0.002)	-0.039^{**} (0.018)
$\lambda(g;z;\mathbf{X}_i^g)$	0.138 (0.688)	-0.836 (2.198)	0.248 (1.801)	2.244 (2.361)
$\lambda(g;z;\mathbf{X}_i^g)^2$	2.634 (3.469)	13.051 (12.028)	5.202 (8.074)	2.689 (6.408)
$\lambda(g;z;\mathbf{X}_i^g)^3$	-10.098^{*} (5.344)	-14.153 (19.842)	-8.396 (11.199)	-5.127 (5.342)
$g*\lambda(g;z;\mathbf{X}_i^g)$	0.122^{***} (0.045)	-0.664^{***} (0.151)	-0.320^{***} (0.119)	-0.574^{***} (0.189)
$z*g$	-0.044^{***} (0.014)	0.188^{***} (0.045)	-0.025 (0.053)	0.941^{***} (0.316)
Constant	7.056^{***} (0.294)	4.846^{***} (0.985)	6.677^{***} (1.067)	0.843 (4.833)
Observations	930	952	952	571
R^2	0.578	0.693	0.071	0.206
Adjusted R^2	0.571	0.688	0.056	0.185
Residual Std. Error	0.109 (df = 914)	0.373 (df = 936)	0.394 (df = 936)	0.646 (df = 555)
F Statistic	83.476^{***} (df = 15; 914)	140.568^{***} (df = 15; 936)	4.770^{***} (df = 15; 936)	9.610^{***} (df = 15; 555)

Note: *p<0.1; **p<0.05; ***p<0.01

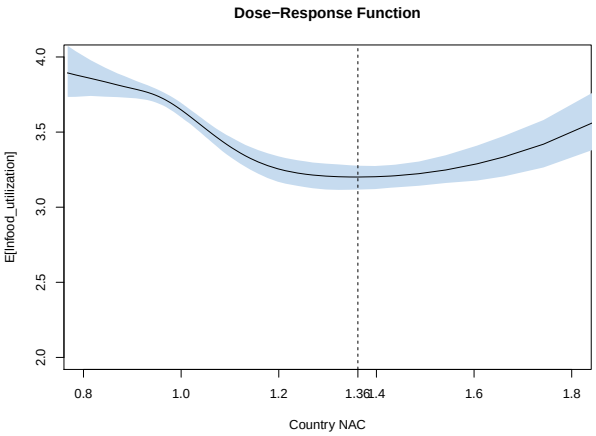

Figure A.7: Dose-response function $E[Y_i(z)]$ of direct NAC on food utilization (log scale) neglecting interference

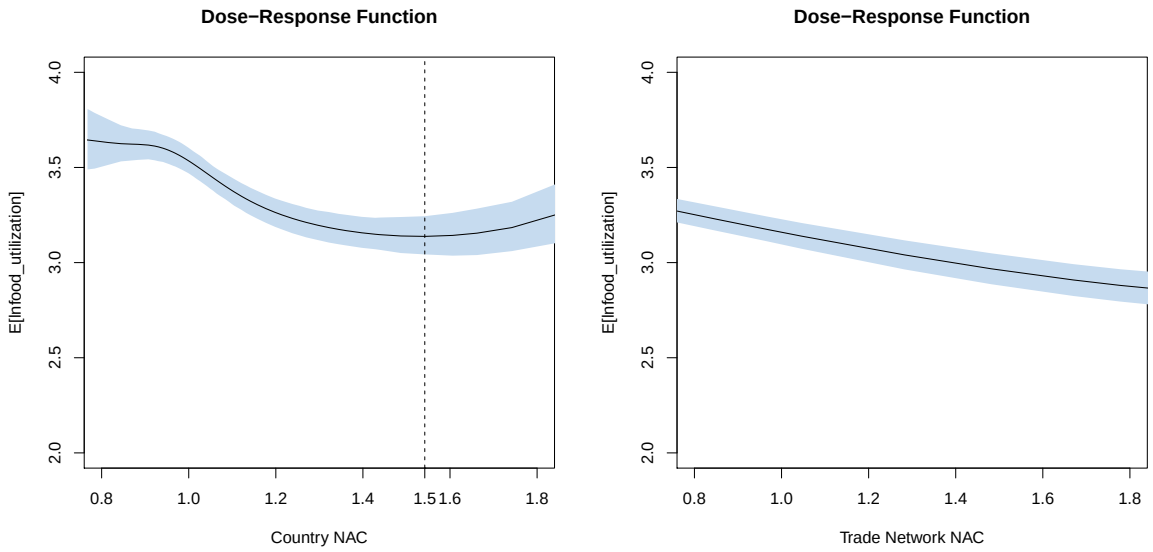

Figure A.8: Marginal dose-response function $\mu^Z(g)$ of direct NAC (left) and marginal dose-response function $\mu^G(z)$ of network NAC (right) on food utilization (log scale)

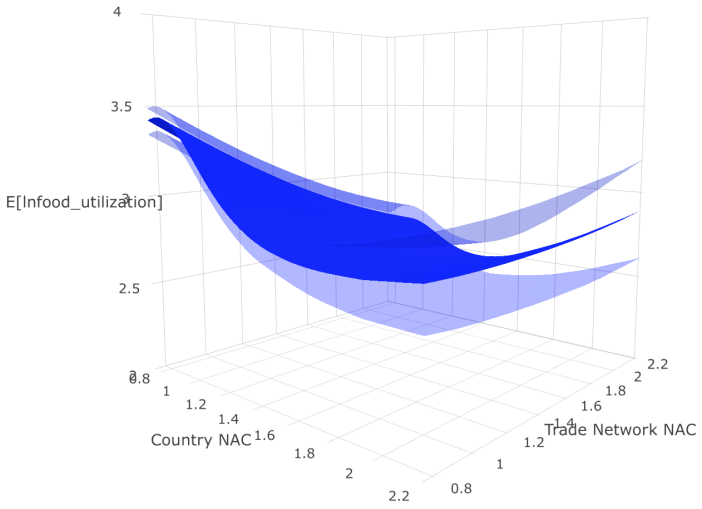

Figure A.9: Average dose-response function $\mu(z, g)$ of direct NAC and network NAC on food utilization (log scale)

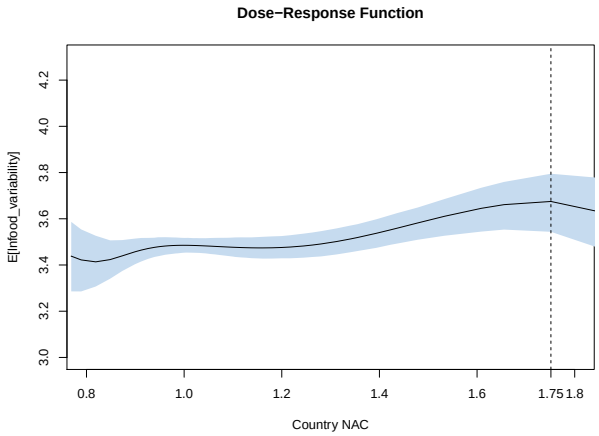

Figure A.10: Dose-response function $E[Y_i(z)]$ of direct NAC on food variability (log scale) neglecting interference

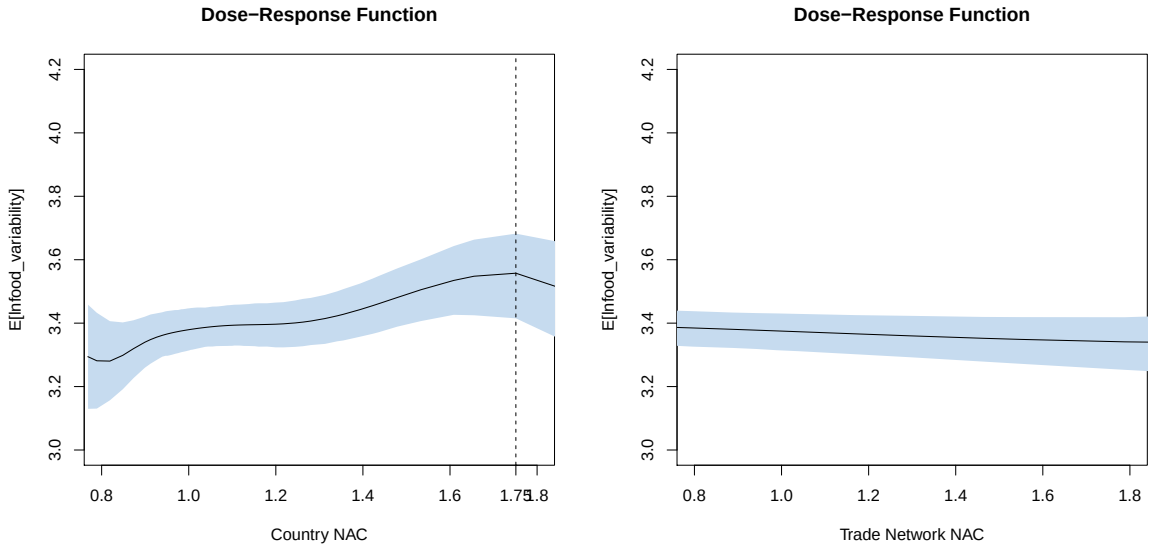

Figure A.11: Marginal dose-response function $\mu^Z(g)$ of direct NAC (left) and marginal dose-response function $\mu^G(z)$ of network NAC (right) on food variability (log scale)

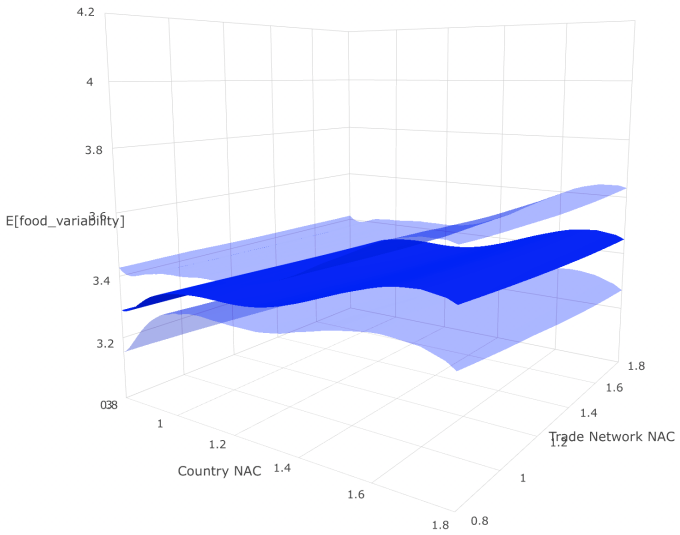

Figure A.12: Average dose-response function $\mu(z, g)$ of direct NAC and network NAC on food variability (log scale)

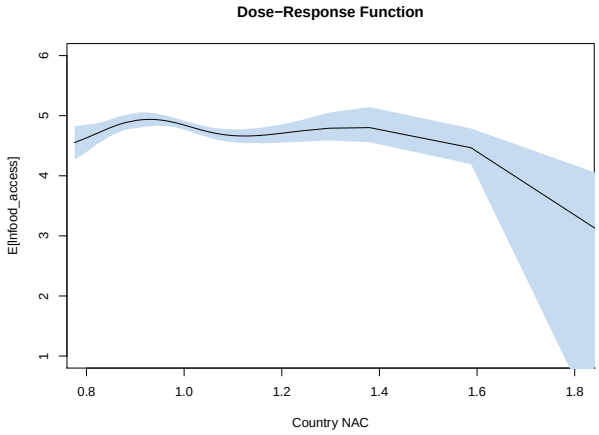

Figure A.13: Dose-response function $E[Y_i(z)]$ of direct NAC on food access (log scale) neglecting interference

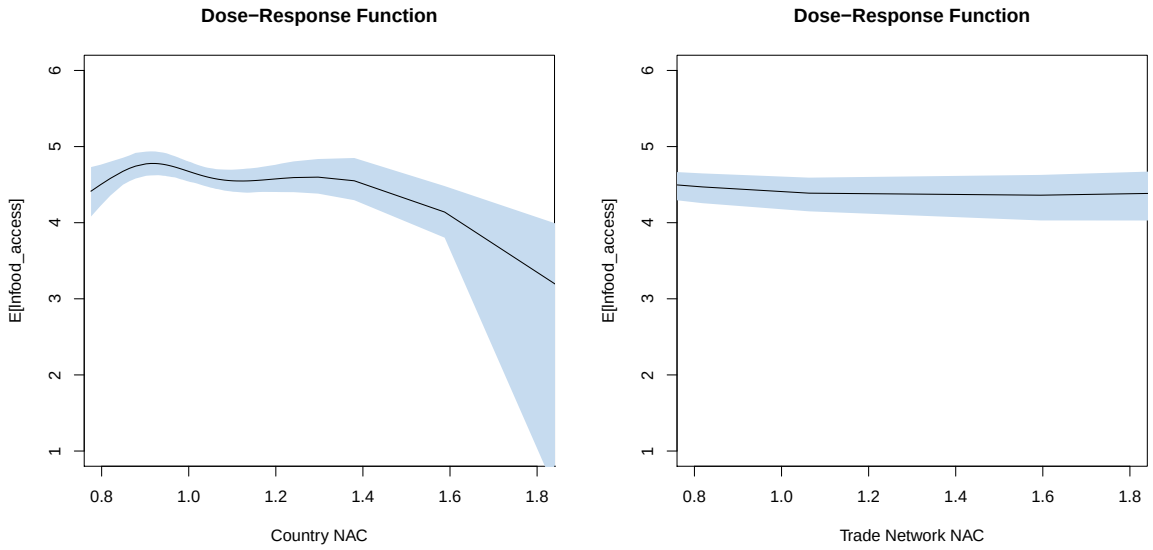

Figure A.14: Marginal dose-response function $\mu^Z(g)$ of direct NAC (left) and marginal dose-response function $\mu^G(z)$ of network NAC (right) on food access (log scale)

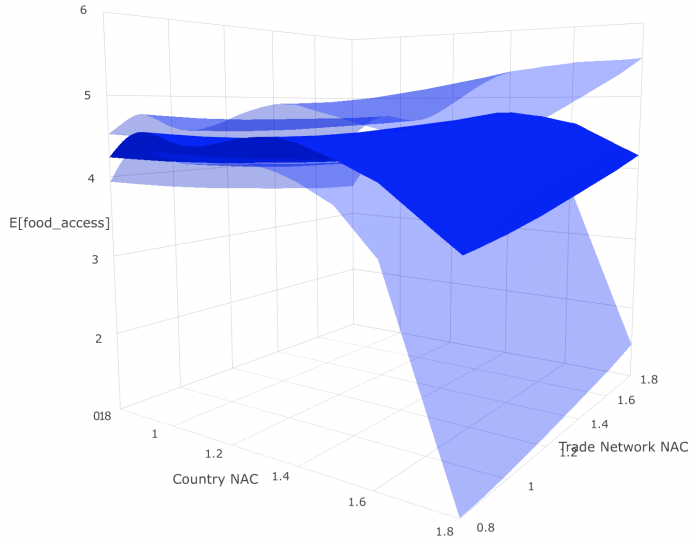

Figure A.15: Average dose-response function $\mu(z, g)$ of direct NAC and network NAC on food access (log scale)