Addendum to Pre-analysis Plan for "Pre-registration, Reporting Guidelines and Publication Patterns in Economics"* to Incorporate Analysis of Spillovers

Fernando Hoces de la Guardia[†] Edward Miguel[‡]
Gufran Pathan[†] Viviane Helena Silva da Rocha[†]
Erik Ø. Sørensen[§] Bertil Tungodden[§]

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1 Introduction

This addendum to the main pre-analysis plan (PAP) of our study describes an analytical approach to test for and quantify potential spillovers in the reporting of study results. The document also provides a brief summary of the research design and discusses the potential scope for spillovers in the study sample.

1.1 Summary of the Research Design

The main pre-analysis plan (PAP) lays out the main analysis for a randomized controlled trial (RCT) designed to examine how different interventions impact the availability of study results and the proportion of null results. The trial is focused on hypotheses drawn from studies registered on the AEA Registry between 2015 and 2017 [Hoces de la Guardia et al., 2024]. In addition to a control arm, the authors of the registered studies were randomly assigned to one of three treatment arms: (T_1) an informational intervention emphasizing the importance of reporting all results, along with an empty results report template; (T_2) the same informational intervention and template, plus a message explaining that our research team had encoded both the main hypotheses and the corresponding publicly available results in a standardized format, presented in a pre-filled results report; and (T_3) the same as T_2, with the added opportunity for research assistance (RA) support (provided by our team) to help locate any missing information.

*Corresponding author: fhoces@berkeley.edu. We thank Katie Baker, Alix Schoback, Akash Shaji, Sarah Stillman, and Michael Walker for providing valuable feedback and assistance on this project.
[†]University of California, Berkeley
[‡]University of California, Berkeley and NBER
[§]NHH Norwegian School of Economics

The two pre-registered primary outcomes of the analysis are the proportion of available results and the proportion of reported null results.

The interventions target the principal investigator (PI) of each study listed on the AEA Registry; the follow-up strategy also involves contacting the study's co-authors, if necessary.[1] The sampling design removes duplicated PIs (i.e., those with more than one registration during the period from 2015 to 2017). However, it does potentially allow for duplicated co-investigators. In practice, co-authors involved in multiple registered studies may be exposed to more than one treatment arm across different studies (or they may be exposed to the same intervention multiple times). Specifically, after our study team sent follow-up emails to co-authors in the first batch of studies in the sample, it became clear to us that a non-trivial number of co-authors who were involved in multiple studies could receive emails related to different treatment arms.[2]

This document discusses the potential for such spillovers across co-authors and studies, and outlines adjustments to the main estimation strategy to estimate such effects. This addendum has been written and filed (on the AEA Registry) while baseline data collection for the RCT is still ongoing.

2 Scope for Potential Spillovers

2.1 Characterizing the Network of Studies and Researchers

To characterize the network of studies and researchers, we focus on the initial 500 studies that meet the inclusion criteria for our sample.[3] Figure 1 displays the distribution of the number of authors per study in the study sample, as defined in the data entered into the AEA Registry. The average number of authors is 2.7 and the median is 3. The total number of unique authors across all studies is 1111.

[1] A follow-up email is sent to a study's PI if they do not respond to the first email. If there is no response to this follow-up message, a second follow-up (the third email overall) is sent to all researchers listed in the registration.

[2] The RCT was implemented in batches. Batch 1 consisted of 140 studies, and this is followed by subsequent batches of approximately 50 studies each.

[3] This is more than our initial goal of 400 studies in the analysis sample to account for exclusions and the archiving of materials for studies in the control group, as described in the main PAP.

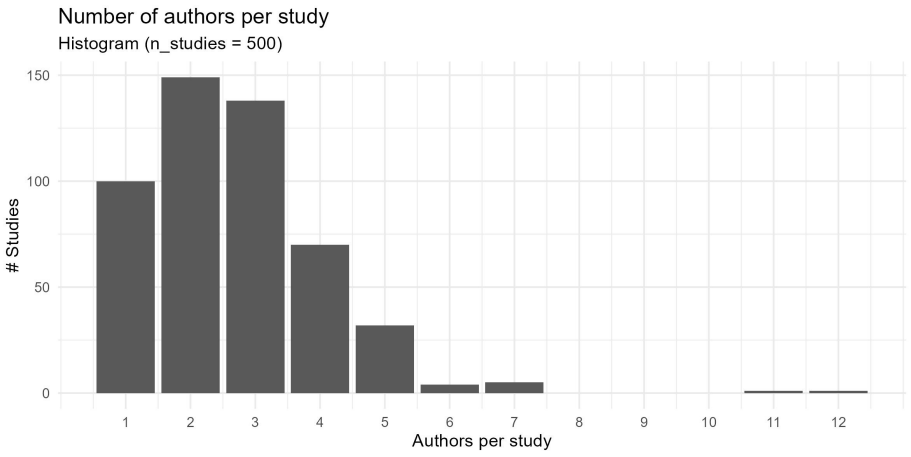

Figure 1: Histogram of number of authors per study on the AEA Registry during 2015-2017

Figure 2 presents the distribution of the number of co-authors per researcher in the study sample, i.e., the total number of other scholars that a given researcher is connected to in the AEA Registry by virtue of being listed as coauthors during the period 2015-2017 (our study period). The average number of co-authors per researcher is 2.7 and the median is 2.

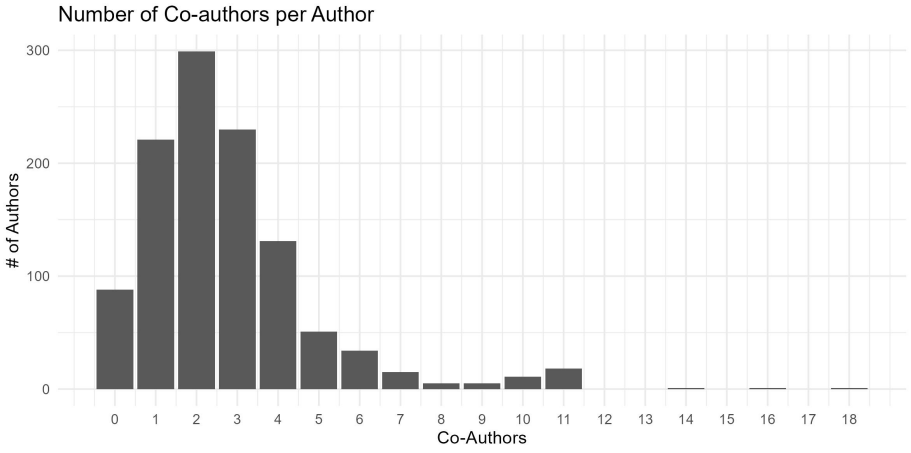

Figure 2: Distribution of the number of co-authors linked to an author in the study sample on the AEA Registry during 2015-2017

Of the 1111 authors in the sample, approximately 934 appear in only one study.

All connections across studies are thus driven by the 177 authors who are present in multiple studies, and their participation ranges from 2 to 4 studies listed on the AEA Registry during the 2015 to 2017 period.

Figure 3 presents the network graph for all the studies in the main sample. 50% of studies on the registry do not share any co-authors with other studies in the sample period and are therefore not subject to the type of potential spillovers that is our primary focus. These studies are represented by the individual dots on the left hand side of the figure. Of the remaining 50% of studies that do share co-authors with other studies in the sample, most share co-authors with one or two other studies on the registry (71.6% of this group of studies). The most "connected" study using this metric shares co-authors with 7 additional studies during the 2015-2017 period.

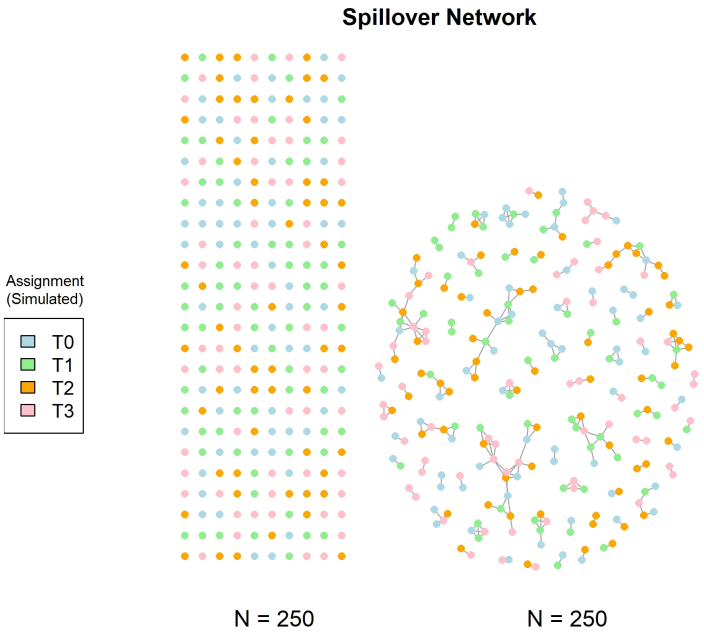

Figure 3: Network Of Studies in the Sample, from the AEA Registry during 2015-2017, showing connections by co-authors. *Note:* Treatment arm assignment from study 141 and onward is simulated in this figure (since the randomization has not yet been carried out at the time of writing), which means that the colors of the nodes will change once the final randomization is available for studies beyond Batch 1.

2.2 Potential for Spillovers By Treatment Status

There are at least two types of channels through which a study can be exposed to other treatments. First, **direct exposure** occurs when a researcher in study i is also a co-author in another study j in our sample. Based on our follow-up strategy, this researcher may receive two emails with different treatments, one for study i and a follow-up for study j. (This could occur if two prior emails to the main principal investigator had gone unanswered, triggering an email to all co-authors on each registration.) Second, **indirect exposure** may occur if studies with different author groups share a co-author on a third study (or even if information flows through shared venues such as departments, editorial teams, or workshops).

We allow spillover effects to depend on both the treatments a researcher is exposed to through co-author networks and the study's own assigned treatment status. We first define the direct exposure that each study is subject to. If we denote the set of authors in study i as A_i, and M_{as} as an indicator taking on a value of 1 if author a is part of study s, we can define the following variables:

$$\begin{aligned} S_{ik} &= \sum_{a \in A_i} \sum_{s \neq i} M_{as} \times T_{sk}, \\ S_i &= \sum_{k=1}^{J} S_{ik}, \\ P_i &= \sum_{k=0}^{J} S_{ik}. \end{aligned}$$

Here S_{ik} is the number of times that co-authors of study i are exposed to other studies in arm k (with $k \in \{0, 1, 2, 3\}$, and the control arm indexed $k = 0$); T_{sk} is an indicator variable for study s being in treatment k; S_i is the number of times co-authors of study i that are exposed to any treatment arm (T_1, T_2 or T_3), and P_i is a variable that captures the total degree to which co-authors of study i appear in other studies in our sample (registered on the AEA Registry during 2015-2017), including the control arm. This defines direct exposure to each treatment arm and to any treatment arm (S_{ik} and S_i, respectively) and allows us to condition on the size of the author network that a study is part of (P_i) in the analysis, as described below.

To illustrate this definition of direct exposure, Figure 4 presents a simplified example of how studies can be exposed to each other. In this example, lower case letters are used to represent authors, and studies are connected at most by one author only: studies 1 and 3 have one exposure each while study 2 has two exposures. Study 4 has no direct exposures, its connection with study 3 is only through a study (5) that is not in our sample (i.e., a study that does not have a registration or is registered outside of the sample period).

This example illustrates why it is convenient to initially focus on direct exposures. If the indirect connection between study 1 and study 3 through study 2 were to be counted as an exposure, this would open up the possibility for additional connections out of sample as well (here, between study 3 and 4). Direct and indirect exposures are qualitatively different: in the former case, the exposure consists of a single author directly

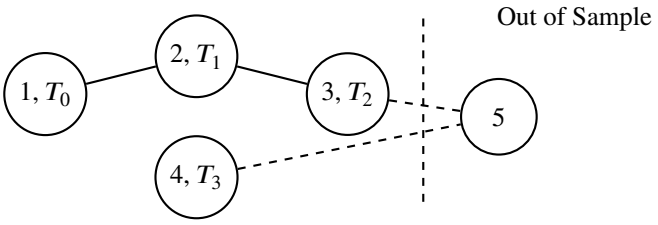

Figure 4: Illustrative example of a network of authors

Note: We assume the following authorships between studies (indexed by i) and authors $(b, c, \ldots, h)$, in different treatments T_j:

$$
\begin{aligned}
&i = 1 : A_1 = \{b, c, d\}, T_0; \\
&i = 2 : A_2 = \{b, e\}, T_1; \\
&i = 3 : A_3 = \{e, f\}, T_2; \\
&i = 4 : A_4 = \{g, h\}, T_3; \\
&i = 5 : A_5 = \{g, f\}, \text{not in sample.}
\end{aligned}
$$

Consequently, our exposure variables are calculated as:

$$
\begin{array}{llll}
S_{11} = 0, & S_{12} = 1, & S_{13} = 0, & S_{14} = 0, \\
S_{21} = 1, & S_{22} = 0, & S_{23} = 1, & S_{24} = 0, \\
S_{31} = 0, & S_{32} = 1, & S_{33} = 0, & S_{34} = 0, \\
S_{41} = 0, & S_{42} = 0, & S_{43} = 0, & S_{44} = 0,
\end{array}
$$

$$
S_1 = 1, \quad S_2 = 1, \quad S_3 = 1, \quad S_4 = 0,
$$

$$
P_1 = 1, \quad P_2 = 2, \quad P_3 = 1, \quad P_4 = 0.
$$

Note that $S_2 = 1$ and $S_2 \neq P_2$, since one link from study 2 is to the control arm (T_0).

observing emails sent to them regarding two different studies, while the latter allows for the possibility that their behavior is influenced by communication with a broader set of collaborators. This second, indirect form of exposure to the treatment through networks of coauthors is interesting and potentially influential. Yet since there are multiple ways in which such indirect exposure could be defined or operationalized (including possibly for studies outside of our main sample period of 2015-2017) we do not pre-specify the analysis of spillover effects of indirect exposure here, and instead leave this for future exploratory analysis.

To begin investigating the potential scope for spillovers by assigned treatment status (via direct exposure), we have simulated the treatment assignment of the additional 360 studies beyond Batch 1 that are yet to be assigned to treatment arms, as noted above, and consider them in addition to the Batch 1 studies.

Table 1 presents the distribution of potential spillovers by assigned arm, distinguish-

ing between no exposure, exposure to the same-arm treatment through another study, and to a different-arm treatment through another study. The simulation indicates that half of the 500 studies (specifically, 250 studies) will not have any co-author that is involved in another study in our sample. The simulation also suggests that the types of exposure are likely to be largely balanced across treatment arms, as expected given the randomized design (but different-arm potential spillovers are slightly underrepresented in the T_3 treatment arm in this simulation exercise, by chance).

Table 1: Exposure Status by Arm Assignment

	Arm Assigned				
Exposure Status	**T0**	**T1**	**T2**	**T3**	**Total**
No exposure	60	68	59	63	**250**
Same-arm exposure	8	7	7	14	**36**
Different-arm exposure	57	51	59	47	**214**
Total	**125**	**126**	**125**	**124**	**500**

Note: Arm Assignment is simulated for studies not in Batch 1.

Table 2 presents the distribution of the potential spillovers by both the treatment arm to which a study is assigned (the columns) and to the arms it is exposed to through co-author networks (the rows). Specifically, each row represents a unique combination of exposures to the four treatment arms through the co-author network, where each sub-index denotes an indicator for each of the four intervention arms (i.e., from left to right: control T_0, T_1, T_2 and T_3). For example, the row denoted by S_{0101} includes all studies that have been exposed through co-authors to T_1 and T_3 (with no exposure to studies in the control arm or in T_2). As the categories are mutually exclusive, the total in each row adds up to the number of studies in each treatment arm.

Table 2: Exposure Combinations by Arm Assignment

	Arm Assigned				
Spillover Combination	**T0**	**T1**	**T2**	**T3**	**Total**
S_{0000}	60	68	59	63	**250**
S_{0001}	13	6	10	14	**43**
S_{0010}	10	11	7	8	**36**
S_{0011}	3	2	1	6	**12**
S_{0100}	9	7	13	3	**32**
S_{0101}	2	0	3	2	**7**
S_{0110}	5	1	2	4	**12**
S_{0111}	5	4	2	1	**12**
S_{1000}	8	9	9	11	**37**
S_{1001}	0	2	5	1	**8**
S_{1010}	0	6	3	3	**12**
S_{1011}	0	2	0	3	**5**
S_{1100}	9	1	3	2	**15**
S_{1101}	0	3	4	0	**7**
S_{1110}	1	3	4	2	**10**
S_{1111}	0	1	0	1	**2**
Total	**125**	**126**	**125**	**124**	**500**

Note: Arm Assignment is simulated for studies not in Batch 1. The $S_{e_0 e_1 e_2 e_3}$ is the count of studies that are exposed to treatments k, with e_k being indicators for exposure to treatment k.

3 Estimation strategy

The main econometric specification registered in the main pre-analysis plan is the following difference-in-differences regression:

$$Y_{it} = \mu_i + \delta_1 I_{t=1} + \sum_{j=1}^{J} \tau_j \left(T_{ij} \times I_{t=1}\right) + \varepsilon_{it}, \quad t = 0, 1. \tag{1}$$

In this expression, Y_{it} denotes the outcome (e.g., the proportion of available results for study i at time t); T_{ij} is an indicator for study i being in treatment arm j; $t = 0$ indicates that it is measured in the pre-intervention period, while $t = 1$ is post intervention; and $I_{t=1}$ is an indicator for the $t = 1$ period. There are $J + 1$ arms (i.e., the three treatment arms and the control arm), and the effects are relative to the left-out control arm ($j = 0$). The specification contains fixed effects at the study level (μ_i) and a time effect for the post-intervention period ($\delta_1 I_{t=1}$).

We augment this specification to incorporate the possibility of spillovers, allowing them to vary depending on both the type of exposure through the co-author network (k), and on the study's own assigned treatment arm (j). We consider direct exposure through the coauthor network, as described above. This leads to the following spillover estimation equation:

$$\begin{aligned} Y_{it} = \mu_i + \delta_1 I_{t=1} + \sum_{j=1}^{J} \tau_j \left(T_{ij} \times I_{t=1}\right) \\ + \sum_{j=0}^{J} \sum_{k=0}^{J} \beta_{jk} (T_{ij} \times I_{t=1} \times S_{ik}) \\ + \sum_{j=0}^{J} \psi_j (T_{ij} \times I_{t=1} \times P_i) + \varepsilon_{it}, \\ t = 0, 1. \end{aligned} \tag{2}$$

To increase the precision of the estimates, we consider the following restrictions on parameters, which we view as reasonable (as discussed further below):

1. No spillover effect from being exposed to control studies through the co-author network: $\beta_{j0} = 0$, for all $j \in \{0, 1, 2, 3\}$.

2. The spillover effect on studies in each arm j does not depend on the arm k to which the study is exposed through the co-author network: $\beta_{jk} = \beta_{j*}$, for all $j \in \{0, 1, 2, 3\}$ and $k \in \{1, 2, 3\}$.

3. The spillover effect on studies in all treatment arms (T_1, T_2, T_3) is the same: $\beta_{jk} = \beta_{*k}$ for all $j \in \{1, 2, 3\}$.

The common informational and encouragement component of all arms (T_1, T_2, T_3) appears to be the most readily transferable element from one study to another through the co-author network. The additional components of T_2 and T_3—the pre-filled results

report and research assistance (RA) support, respectively—are study-specific and thus appear less likely to spillover onto other studies. For studies in control (T_0), spillovers could take the form of partial exposure to standardized reporting practices and exposure to the informational message on the importance of reporting all results, while for studies in T_1, T_2, and T_3, the exposure could serve as a reminder of the same message. We will be able to test whether these assumptions do in fact hold in the estimation of the more general equation above.

These restrictions combined imply a constant spillover effect for the control arm (T_0), $\beta_{0k} = \beta_0$ for all $k \in \{1, 2, 3\}$. They imply another constant spillover effect for studies in the treatment arms ($T1$, $T2$, and $T3$), of $\beta_{jk} = \beta_*$ for all $k \in \{1, 2, 3\}$ and $j \in \{1, 2, 3\}$.

These assumptions and parameterizations simplify the 16 β_{jk} parameters from the general equation above to those presented in Table 3:

	Arm Exposure			
Arm Assigned	$k = 0$	$k = 1$	$k = 2$	$k = 3$
$j = 0$	0	β_0	β_0	β_0
$j = 1$	0	β_*	β_*	β_*
$j = 2$	0	β_*	β_*	β_*
$j = 3$	0	β_*	β_*	β_*

Table 3: Parameter Estimates under Restrictions 1, 2 and 3.

For treatment arms, the spillover effects can be understood as an effect for the control arm (β_0) plus an additional effect for those in the three treatment arms, reflecting either complementarity or substitution with the direct effect of the interventions. We adopt the following parameterization:

$$\beta_* = \beta_0 + \theta,$$

where $\theta > 0$ indicates complementarity and $\theta < 0$ substitution between treatment assignment and exposure through the co-author network. With this notation, the resulting regression specification can be expressed as:

$$\begin{aligned} Y_{it} = \mu_i + \delta_1 I_{t=1} + \sum_{j=1}^{J} \tau_j \left(T_{ij} \times I_{t=1}\right) \\ + \beta_0 (S_i \times I_{t=1}) + \theta (S_i \times I_{t=1}) \times I_{T_{i0} \neq 1} \\ + \sum_{j=0}^{J} \psi_j (T_{ij} \times I_{t=1} \times P_i) + \varepsilon_{it}, \quad t = 0, 1. \end{aligned} \tag{3}$$

4 Hypotheses

In this section, we describe the hypothesis tests that we will carry out.

We will first estimate a somewhat less restrictive model to test for the hypothesis of any spillovers in the co-author network. To do so, we impose restriction #1 on the more general regression specification (equation 2) and estimate the following regression model:

$$Y_{it} = \mu_i + \delta_1 I_{t=1} + \sum_{j=1}^{J} \tau_j \left(T_{ij} \times I_{t=1}\right) + \sum_{j=0}^{J} \sum_{k=1}^{J} \beta_{jk} (T_{ij} \times I_{t=1} \times S_{ik}) + \sum_{j=0}^{J} \psi_j (T_{ij} \times I_{t=1} \times P_i) + \varepsilon_{it}, \quad t = 0, 1. \quad (4)$$

H1 - There are no spillover effects from exposure through the co-author network to any of the treatment arms (T_1, T_2, T_3) onto studies in any of the arms (T_0, T_1, T_2, T_3):

$$\text{H1}_0 : \beta_{jk} = 0 \text{ for all } j \in \{0, 1, 2, 3\} \text{ and } k \in \{1, 2, 3\}. \quad (5)$$

The alternative hypothesis ($H1_A$) is that at least one coefficient is different from zero.

To estimate the magnitudes of the spillovers, we plan to focus on the more restrictive regression specification (equation 3) to improve statistical power. We plan to test the following hypotheses:

H2 - There is no spillover effect through the co-author network from exposure to the treatment arms (T_1, T_2, T_3) onto the control arm (T_0):

$$\text{H2}_0 : \beta_0 = 0, \quad \text{H2}_A : \beta_0 \neq 0. \quad (6)$$

H3 - There is no spillover effect through the co-author network from exposure to the treatment arms (T_1, T_2, T_3) onto the treatment arms (T_1, T_2, T_3):

$$\text{H3}_0 : \beta_0 + \theta = 0, \quad \text{H3}_A : \beta_0 + \theta \neq 0. \quad (7)$$

H4 - Exposure to treatment (any of T_1, T_2, T_3) through the co-author network and assignment to a treatment arm (any of T_1, T_2, T_3) are substitutes:

$$\begin{aligned} \text{H4}_0 &: \quad \theta \leq 0, \quad \text{(Substitutes)} \\ \text{H4}_A &: \quad \theta > 0. \quad \text{(Complements)} \end{aligned} \quad (8)$$

Following the main pre-analysis plan (PAP) [Hoces de la Guardia et al., 2024], each hypothesis will be tested for two primary outcomes: the fraction of hypotheses available (Y_{1i}), and the fraction of null results reported (Y_{3i}) per study.

We consider H1, H2 and H4 to be the primary hypotheses. We will test H3, but view it as a secondary hypothesis since it is closely related to the results of H2 and H4 taken together.

Unlike the main registration, we do not view heterogeneity tests and subgroup effects as primary tests in the spillover analysis, due to concerns over statistical power. We view any such heterogeneity tests in the spillover analysis as exploratory.

The results of the spillover analysis may affect the interpretation of the main prespecified analysis. In particular, if the null hypothesis of no spillover effect is rejected (Hypothesis H1), then we will also report the total treatment effect in each intervention arm as the sum of the direct effects specified in the main PAP (the τ_j terms) plus the estimated spillover coefficients (in equation 3) times the average values of the exposure measures.

Additionally, we may perform exploratory analyses with indirect exposure measures discussed in Section 2.2 and test each of the above hypotheses for the indirect measures. As these indirect exposure effects appear likely to be weaker than the spillovers from direct exposure, we will likely only carry out this exploratory analysis of indirect exposure effects if there is evidence of spillovers from direct exposure.

References

Fernando Hoces de la Guardia, Edward Miguel, Viviane Silva da Rocha, Gufran Pathan, Erik Ø. Sørensen, and Bertil Tungodden. Pre-registration, reporting guidelines and publication patterns in economics. AEA RCT Registry, June 2024. URL `https://doi.org/10.1257/rct.13755-1.0`.