The Lead-Crime Hypothesis: A Meta-Analysis*

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Abstract

Does lead pollution increase crime? We perform the first meta-analysis of the effect of lead on crime by pooling 529 estimates from 24 studies. We find evidence of publication bias across a range of tests. This publication bias means that the effect of lead is overstated in the literature. We perform over 1 million meta-regression specifications, controlling for this bias, and conditioning on observable between-study heterogeneity. When we restrict our analysis to only high-quality studies that address endogeneity the estimated mean effect size is close to zero. When we use the full sample, the mean effect size is a partial correlation coefficient of 0.11, over ten times larger than the high-quality sample. We calculate a plausible elasticity range of 0.22-0.02 for the full sample and 0.03-0.00 for the high-quality sample. Back-of-envelope calculations suggest that the fall in lead over recent decades is responsible for between 36%-0% of the fall in homicide in the US. Our results suggest lead does not explain the majority of the large fall in crime observed in some countries, and additional explanations are needed.

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1. Introduction

Homicide rates spiked and then fell in a consistent pattern across many western countries in the 20th century (figure I). In the US alone the homicide rate has halved since the 1980s, when it was as high as the road fatality rate is today. In other countries the falls are not so great in magnitude, but still amount to many lives saved. If the causes of this fall were known, many more deaths and trauma could potentially be prevented.

Is lead pollution responsible? Lead is a toxic metal linked to harmful health and behavioural outcomes (see section 2). Studies have pointed to falling lead levels in the environment as a cause of the falls in homicide, and as a factor in crime rates in general. The reduction in lead pollution is largely due to falling emissions from leaded gasoline (figure II), but also due to less lead pollution from water pipes, paint, food, and soil. However, the rise and fall pattern in figure I is by no means uniform. Further, Buonanno *et al*. (2011) show that while total crime has behaved similarly to homicide in the US, it has not in Europe (figure III). Alternative hypotheses for the observed fall in crime in some countries range from falling poverty levels (Rosenfeld and Fornango, 2007, and Messner, Raffalovich, and Mcmillan, 2001), to demographic transition, where an ageing population is less likely to be victimised by or engage in crime (Fox, 2005, chap. 9; Baumer, Rosenfeld, and Wolff, 2012), increased/better policing or incarceration (Levitt, 1996, 1997, 2004; Marvell and Moody, 1996; and Corman and Mocan, 2000), to more controversial hypothesis such as legalized abortion reducing the number of children born into "adverse home environments" (Donohue and Levitt, 2001, 2019; Buonanno et al., 2011). Tcherni-Buzzeo (2019) provides a recent summary of potential causes.

Against this background, our paper conducts the first meta-analysis of the effect of lead on crime. We systematically review the literature and, using the procedural guidance from Havránek et al. (2020), construct a dataset of estimates converted to comparable effect sizes. We perform tests for publication bias and find that the effect of lead on crime is overstated in the literature due to this bias. Furthermore, we find substantial between-study heterogeneity in our sample. We therefore use meta-regression to estimate an average effect size accounting for both publication bias and the observable between-study heterogeneity.

We take into account model uncertainty in our meta-regression analysis by estimating over 1 million specifications, using every combination of our covariates on both the full sample and several subsamples. We then plot the distributions of the estimated average effect size of lead on crime. Our main finding is that there are substantial differences between the average effect size when we use the full sample, and when we use only the high-quality study designs that address endogeneity. The mean partial correlation coefficient in the full sample, evaluated at sample averages, is 0.11, while the equivalent for the high-quality sample is 0.01, almost zero. Similarly, the sample of studies that have crime in an area as the focus of analysis have a larger mean effect size compared to that of studies which focus on individual behaviour. Conversely, we do not find evidence of differences for the effect of lead on different types of crime when we use homicide, violent, and non-violent crime samples.

Finally, we convert the estimated mean partial correlation coefficients to elasticities. Our calculations give a plausible elasticity range of 0.22-0.02 for the full sample mean effect and 0.03-0.00 for the high-quality sample that addresses endogeneity. We conduct back-of-envelope calculations which imply the reduction in lead pollution may be responsible for between 36%-3% of the fall in homicide in the US when the full sample elasticity is used, but only between 5%-0% when the high-quality sample elasticity is used. Our findings suggest that, while there is a possibility that the effect of lead may be substantial, it is not responsible for the majority of the fall in crime. Therefore, other explanations require further investigation.

2. Lead and Crime

Lead has been part of the human environment for a long time. It was used in cosmetics, paint, and as coinage in ancient China (Schafer, 1956). Similar uses were recorded in ancient Egypt, India, and across the Bronze Age world (Needleman, 1992). The sweet taste of lead acetate meant that the Roman Empire and later medieval Europe used lead to sweeten wine, cider, and food (Lessler, 1988). The Romans had many other uses for lead, using it for cooking utensils, pottery, and water pipes (Hernberg, 2000). Indeed, Roman use of lead was prodigious, with estimates from Greenland artic ice putting the increase in atmospheric lead pollution at around 4000 metric tons a year at its peak

around 2000 years ago (Hong *et al*. 1994). This is equivalent to the UK's lead pollution emissions in the mid-1980s, when leaded gasoline had not yet been phased out.

Lead is a useful but toxic metal. At high levels of exposure even adults will experience lead poisoning. Acute lead poisoning is rare but can kill quickly. Chronic poisoning can still kill and is associated with abdominal pain, organ failure, tumours, and exhaustion, amongst other symptoms (WHO, 2010a). Although chronic lead poisoning in adults still happens, and appears to affect behaviour, it is primarily the long-term lead exposure of children that is thought to influence crime rates.

Children are especially vulnerable to lead pollution. Children not only absorb more lead per unit body weight than adults, but, as the brain and nervous system are still developing, lead has more harmful long-term effects even at low levels (WHO, 2010b). Lead is chemically similar to calcium[4]. Calcium is important for cell growth, and synaptic functioning, as well as a myriad of other body processes (Sanders *et al*., 2010). Therefore, lead is particularly harmful to the developing brain and nervous system, and thus in the womb and early infancy are the worst time to be exposed to lead (WHO, 2010b).

The causal chain of lead to crime starts with the biological changes it induces at this young age. The mechanism for these changes is laid out in Sanders *et al*. (2010), and there is an array of evidence for lead's negative effects. These include impaired nerve conduction (Sindhu and Sutherling, 2015), damaged myelination in the nerve system (Brubaker *et al*., 2009), impeded brain development (Lanphear, 2015), and reduced brain matter (Cecil *et al*., 2008).

The next link in the chain is from biological change to behavioural change in later life. Meta-analyses have found that lead exposure is associated with aggressiveness and other conduct problems (Marcus, Fulton, & Clarke, 2010), lower IQ (Schwartz, 1994), and impaired cognitive functioning (Vlasak et al., 2019, and Seeber et al, 2002).

The final link is from behavioural changes to an increased propensity to commit crime. There are several possible mechanisms. Needleman pioneered research on lead exposure and aggressiveness (1996), suggesting it is linked to violent crime in particular. In

[4] They both convert easily to ions with 2+ charge.

contrast, Denno (1990) and Fergusson, Boden and Horwood (2008) argue that the link is through lower education outcomes, leading to worse life outcomes, which causes increased criminality. This mechanism is consistent with Becker's (1968) economic theory of crime, where lower opportunity cost makes crime relatively more attractive, and suggests lead would show a stronger link to property crime than violent crime. A third mechanism was proposed by Gottfredson and Hirschi (1990), where lack of self-control, combined with opportunity, causes higher crime rates. Lead has been associated with increases in impulsivity (Winter and Sampson, 2017), and so may cause an increase in crime through this process. If this mechanism were true we might expect increases in violent crime, non-violent crime, or both. Separating the different types of crime may help identify which, if any, mechanism lead acts through. However, whilst a range of mechanisms have been laid out linking lead in the environment to the propensity to commit crime, the strength of this link is a matter of empirical enquiry. The main objective of this paper is to quantify the strength of this link from the range of empirical work reported to date. To do this, we use meta-analysis.

3. Data

Meta-analysis data collection begins by specifying the criteria which studies must fulfil to be accepted into the analysis.

The criteria we chose were:

1. The explanatory variable must be some quantitative measure of lead exposure.
2. Outcome variable must measure crime in some way (i.e. not other types of behaviour such as aggressiveness or depression).
3. Must have original estimates, i.e. no review papers.
4. Must have estimates that can be combined into a meta-analysis.[5]
5. Be published before December 2019.
6. Study must be available in English.

[5] By which we mean they must be convertible to a common estimate such as a partial correlation coefficient. See discussion below.

We then undertook a systematic literature review for papers on Web of Science, PubMed, and Google Scholar in 2019. We also searched on NBER and REPEC for working papers to include as much "grey" literature as possible. The keyword combinations used were:

("lead", or "lead" AND "pollution", or "lead" AND "poisoning", or "lead" AND "exposure", or "lead" AND "blood", or "lead" AND "air", or "lead" AND "paint", or "lead" AND "water")

Combined with:

("crime" or "conviction" or "arrest" or "jail" or "prison")

After searching, papers were screened to see if they fulfilled the criteria, as laid out in the PRISMA[6] flow diagram (figure IV). A review and description of the studies included is given in the appendix.

The vast majority of the studies identified in the literature review did not fulfil criteria one or two and therefore did not estimate the lead-crime relationship. These were then filtered out at the screening stage. 31 papers did estimate the lead-crime relationship, but 7 of these could not be converted into comparable effect sizes, failing criterion four. Criterion four is needed because estimates must be combined in a meta-analysis. Estimates are made comparable by converting into a common metric, such as the partial correlation coefficient (PCC). Most regression coefficients and simple correlations can be converted into PCCs easily. Odds ratios and standardised mean differences can also be converted into PCCs. However, five papers used risk ratios (Boutwell *et al*., 2016; Boutwell *et al*., 2016; Haynes *et al*., 2011; Stretesky and Lynch, 2001; and Write *et al*., 2008). Risk ratios can be converted into odds ratios, which can then be converted to PCCs, but need a base rate risk to do so. It was not possible to infer a base rate risk from the data available in the papers. Therefore, these papers did not fulfil criteria four and were excluded at the eligibility stage. One other paper (Masters and Coplan, 1999) contained charts but not enough information to make PCCs and was excluded. Similarly, Denno (1990) did not have enough information to use the estimates. No papers were excluded based on criteria six, but search terms were only in English. This left 24 papers in the final meta-analysis dataset.

[6] PRISMA is Preferred Reporting Items for Systematic Reviews and Meta-Analyses. It is a standard across sciences for how to report any process where a systematic literature search with filtering is performed.

We organised accepted papers into a dataset following the guidelines for meta-analysis in economics in Havránek *et al*. (2020). Every paper gave multiple estimates for the effect of lead on crime. Meta-analyses tend to either select one estimate from each study as a "representative" estimate; or take all estimates and account for the potential clustering of estimates from the same study. Both are defensible. Taking all estimates means more information available for the meta-analysis. Representative estimates, on the other hand, may be less biased. For example, a researcher may show a simple OLS estimate before giving reasons for why it will be biased. They then go on to use their preferred method of estimation, which attenuates this bias. In most of our results we use all estimates, but as a robustness check we also test our results by using one representative estimate from each study. There was not always a clear representative estimate from each study. Therefore, choosing the estimates involves some subjective judgement. We tried to choose results mentioned in the abstract or as the main result. In general, we chose representative estimates which were less specific (i.e., totals preferred to subsample male/female, white/black results etc.), and results which had more covariates.

In the full sample, there are 529 estimates from the 24 studies. The dataset forms an unbalanced panel, with each estimate being an observation and observations grouped by study. The studies included span across a variety of disciplines including economics, sociology, medicine, epidemiology, and criminology.

Study effect sizes were then converted to the common effect size using PCCs. See the appendix for more details of how PCCs and their standard errors are calculated. Conversion is necessary because both lead and crime are measured in different ways in each paper, and therefore must be converted to be comparable. PCCs tend to be the common metric used in economic meta-analyses (see for example Doucouliagos (1995), Efendic and Pugh (2009), and Valickova, Havranek, and Horvath (2015)).

PCCs measure the correlation between two variables holding other variables in the model constant. Their sizes are not intuitive. They have no unit and cannot be interpreted quantitively in a meta-analysis with varied measurements of outcome (Doucouliagos, 2011). However, as they are bounded from -1 to 1, they do offer a sense of the magnitude and direction of an effect. In a survey of economic effect sizes Doucouliagos (2011) offers the following rough guidelines: 0.07-0.17 is a small effect size, 0.18-0.33 is a moderate

one, and above 0.33 a large one. For most of the paper, we follow this taxonomy, but a small effect combined with a large absolute change in a variable can still mean it is significant for welfare. Therefore, in section 4.5 we convert our main PCC estimates to elasticities using some additional data and assumptions to give a measure of the welfare impacts of our estimates.

Table I presents the mean, median and weighted average PCC for each study (with weights being equal to the precision, 1/standard error). It also includes some information on the characteristics of each study.

4. Methods

4.1 General Approach[7]

Let θ_j be an effect size of interest in study *j*. Study *j* uses some method to estimate θ_j and these we denote as $\hat{\theta}_{ij}$, for estimate *i* of study *j*. Researchers are often interested in both how close $\hat{\theta}_{ij}$ is to θ_j, and in how useful θ_j would be in predicting results from a similar event or study. This can be interpreted as the degree of external validity of a study.

If θ_j is a draw from some distribution with a likelihood function $\psi(\cdot\,|\Theta)$ such that $\theta_j \sim \psi(\cdot\,|\Theta)\ \forall\ j$, then there exists some parameter(s) Θ which can give information about a new draw θ_{j+1} from that distribution. It is the parameters contained in Θ that are estimated in a meta-analysis. There may be several parameters of interest, but in practice meta-analyses usually estimate two: θ, the mean of the distribution, and the variance τ^2. This is because meta-analyses tend to impose the assumption $\theta_j \sim N(\theta, \tau^2)\ \forall\ j$ in the interests of efficient estimation. Even if this is not the true shape of the distribution McCulloch and Neuhaus (2011) show, both in theory and simulation, that maximum likelihood estimates are robust to different distributions of θ_j around θ. If we also assume, as the individual studies themselves usually do, that $\hat{\theta}_{ij}$ follows a

[7] This sections owes much to the excellent expositions in Meager (2019), Rubin (1981), and Röver (2018). Much of their explanation deals with Bayesian methods but works equally well for non-Bayesian methods up to the point we arrive at.

normal distribution with mean θ_j and variance σ_j^2 , then this leads to the normal-normal hierarchical model of Rubin (1981):

(1) $$\theta_j \sim N(\theta, \tau^2)\ \forall\ j$$

(2) $$\hat{\theta}_{ij} \sim N\left(\theta_j, \sigma_j^2\right)\ \forall\ i \text{ and } \forall\ j$$

(3) $$\hat{\theta}_{ij} \mid \theta, \sigma_j^2, \tau^2 \sim N\left(\theta, \sigma_j^2 + \tau^2\right)\ \forall\ i \text{ and } \forall\ j$$

where the last expression follows from the previous two but is expressed in marginal form, as in Röver (2018). This marginal form can be further extended to be conditional on observable variables, common across the $\hat{\theta}_{ij}$'s, as we do in our meta-regression analysis.

The variance of the effect size distribution $\tau^2$is a crucial measure of how useful aggregation of estimates will be. If τ^2 is zero, then all studies are estimating the exact same effect and it is only the study variances that affect how well they can predict θ_{j+1}. This we call the common effect model following the Rice, Higgins, and Lumley (2018) terminology. As τ^2 grows larger, aggregation becomes less useful. $\tau^2 \to \infty$ represents an "apples and oranges" comparison where meta-analysis should never be undertaken.

4.2. Between-Study Heterogeneity

We begin investigating between-study heterogeneity in effect sizes by plotting each study's weighted average PCC along with their 95% confidence intervals in figure V. Recall that the PCC is a statistical measure of the common variance between lead and crime after accounting for other factors and is bounded between -1 and 1. Only two of the studies have negative average PCCs. There are 13 studies with an average PCC of 0-0.1 with some degree of overlap in confidence intervals (one measure of how much heterogeneity there is between studies). 9 studies have PCCs of 0.18-0.94, with less overlap in intervals. The two Nevin studies have particularly strong effect sizes and no overlap with other studies.

If in (3) τ^2 is zero and the common effects model is true, then a consistent estimator of θ is a weighted average with the weights equal to $1/\sigma_j^2$. The estimate variances σ_j^2 are not observed. Instead, we use $\hat{\sigma}_{ij}^2$, the estimated variance from each study estimate. If $\tau^2 \neq 0$ then some estimated $\hat{\tau}^2$ is needed for the weighted average (see appendix for details on common and random effects estimation). We show the common and random effects estimates at the bottom of figure V. The common effects point estimate is 0.01 and the random effects 0.18. The difference between them illustrates that between-study heterogeneity is important, as the lower the estimated heterogeneity between studies, the closer the random effects estimate will be to the common effects. A Cochran's Q test (Cochran, 1954) strongly rejects the null of no between-study heterogeneity, with a p-value of 0.0001.

It is unlikely that the only source of this heterogeneity is the random, unobservable variances σ_j^2 and τ^2. (3) can be extended to be conditional on a $1 \times K$ vector of variables $\boldsymbol{x}_{ij}$. In this case the study specific estimates θ_j are a function of this variation in $\boldsymbol{x}$ and we have the conditional distribution:

$$\hat{\theta}_{ij} \mid \sigma_j^2, \tau^2, \boldsymbol{x_{ij}}, \boldsymbol{\beta} \sim N\left(\boldsymbol{x}_{\boldsymbol{ij}}'\boldsymbol{\beta}, \sigma_j^2 + \tau^2\right) \forall\, i \text{ and } \forall\, j \tag{4}$$

If these variables are observable, we can include them in our estimation. To investigate sources of observable between-study heterogeneity, table II splits the data into sub-samples, based on common characteristics. These characteristics are also used as covariates in the meta-regression analysis and described fully in section 4.4. We then compare three measures of between-study heterogeneity for each sample, $\hat{\tau}^2$, $\hat{I}^2$, and $\widehat{H}^2$. For each of these measures, the higher they are, the higher the estimated between-study heterogeneity. $\widehat{H}^2$ and $\hat{I}^2$ are sensitive to the number of estimates and the variation in the standard error of those estimates. $\hat{I}^2$ tends to 100 as the number of studies included increases. $\hat{\tau}^2$ is an estimate of the variance of the effect size distribution in (3) using the DerSimonian-Laird (1986) method and is less sensitive to the number of studies but does not give a sense of how important between-study heterogeneity is compared to within-study sampling variation.

Looking at table II we can see which variables seem important for heterogeneity and the different estimated average effect sizes. The subsample of studies which control for

endogeneity has a lower estimated heterogeneity and a smaller effect size compared to the correlational sample. Endogeneity can arise from unobserved variables correlated with both crime and lead. These could bias upwards the estimate of the effect of lead on crime. We cannot rule out that these variables may cause individuals both to commit more crime and be more exposed to lead, rather than lead being the cause. Therefore, the difference between the "addressing endogeneity" sample and the full sample could be related to these factors.

Studies that look at individual propensity to commit crime have lower estimated heterogeneity and estimated effect size compared to studies that look at crime committed within a geographic area. Studies which use homicide as the dependent variable appear to have less heterogeneity and find a stronger effect size. This reduction in heterogeneity may be due to lower measurement error in homicide data compared to other types of crime, combined with more similar classification of this crime across countries, and therefore less noise in the data. Whether a study reports odds-ratios is important for heterogeneity. Finally, when race, gender, education and income covariates are included in an estimation, these tend to lower the effect size and these subsamples also show less between-study heterogeneity than those which do not include these covariates. The estimated differences in effect size and heterogeneity between subsamples indicates observable variation is important and must be taken into account when we estimate an "average effect". We incorporate the observable variation indicated in table II into our meta-regression analysis in section 4.4.

A further, and common source of heterogeneity in effect sizes in meta regression analysis comes from publication bias. We investigate this in the next section.

4.3. Publication bias

Publication bias is a well-known problem across disciplines (see DeLong and Lang, 1992; Ioannidis, 2005; Ioannidis, Stanley and Doucouliagos, 2014; Ioannidis, 2016; Szucs and Ioannidis, 2017; and Ferraro and Shukla, 2020). Papers which contain statistically significant effect sizes are more likely to be published than those which show no effects, or those which contain counter-intuitive results (also known as the

bottom-drawer problem). It is standard practice to test for the presence of publication bias in meta-analysis.

The first and most common step is to simply chart the data and visually inspect for bias, using a funnel plot. Figure VI plots PCCs against their precision. A funnel with no bias should be symmetrical around one or more central tendencies. The estimates will tend to spread out as the precision decreases, but they should do so symmetrically if this is only due to sampling noise. Figure VI shows a pronounced asymmetry in the estimates, suggesting there may be a positive bias. It is also possible this is due to heterogeneity within the sample. We explore this possibility in section 4.4.

More formal testing of publication bias is also possible. A linear relationship between the estimate and its precision, as there seems to be in figure VI, would indicate the presence of publication bias (see Stanley and Doucouliagos, 2014). This naturally leads to the estimating equation (5).

$$(5) \qquad \hat{\theta}_{ij} = \theta + \beta_F \hat{\sigma}_{ij} + u_j + \epsilon_{ij}; \text{ where } \epsilon_{ij} \sim N\left(0, \sigma_j^2\right) \text{ and } u_j \sim N(0, \tau^2)$$

This is the combined Funnel Asymmetry Test (FAT) and Precision Effect Test (PET). Here the FAT is β_F, and is an estimate of the size and sign of publication bias. It is a function of the inverse Mills' ratio (Stanley and Doucouliagos, 2014). If positive then estimates that are positive are more likely to be published than negative ones. This test also gives an estimate of θ that takes into account this bias, called the PET. (5) nests the common effects model where τ^2 is zero.

The test in (5) would be subject to heteroskedasticity, as can be observed from figure VI. We have estimates of the heteroskedasticity in $\hat{\sigma}_{ij}$. These can therefore be used to weight the regression and we estimate the test with weighted least squares following Stanley (2008).

$$(6) \qquad \hat{t}_{ij} = \theta \frac{1}{\hat{\sigma}_{ij}} + \beta_F + v_j + e_{ij}$$

Here the dependent variable $\hat{t}_{ij}$ is now the t-ratio, rather than the estimate alone. The intercept of the regression is the FAT and the coefficient on $\frac{1}{\hat{\sigma}_{ij}}$ is the PET.

We estimate three variations on the FAT-PET. First with OLS and clustered standard errors by study, but no study fixed effects; second, a variation of this where we regress on the variance rather than the standard error[8]; and a full hierarchical FAT-PET with study fixed effects. We estimate this with restricted maximum likelihood (REML), as Monte Carlo simulations suggest REML performs well for unbalanced panels (Baltagi, Song and Jung, 2000). As a robustness check, we also estimate the FAT-PET and FAT-PEESE with only the representative estimates, rather than the full sample, and the results are similar (see appendix for details). The results in table III show positive bias is present across all tests, albeit with wide confidence intervals. They also show the estimate of average effect after adjusting for bias is close to zero for all tests.

The final test for publication bias used here is the Andrews and Kasy (2019) method. Monte Carlo simulations show it is among the best performing bias estimators and it performs particularly well when between-study heterogeneity is high (Hong & Reed, 2020), as we have in our sample. Andrews and Kasy observe that given different probabilities of publication, due to commonly used significance bounds, we will observe a truncated sample of effect sizes. If we set one probability of observing a value as a reference, they show we can identify the other probabilities relative to this. These probabilities can now be estimated up to scale with maximum likelihood. We then use these estimated, relative probabilities to reweight the observed sample. This allows us to reconstruct the true, untruncated distribution and estimate the mean of the effect size distribution.

[8] The Funnel Asymmetry test and Precision Effect Estimate with Standard Error (FAT-PEESE). Stanley and Doucouligas (2014) find the FAT-PEESE can sometimes perform better in simulations. They find the FAT-PEESE seems to especially perform better when the true effect is not equal to zero.

Using this method, we estimate three publication probabilities relative to that of a positive t-ratio greater than 1.96. That is, relative to a positive effect size, significant at the 95% level. The three probabilities are: negative and significant at a 95% level; negative and not significant; and positive and not significant. We also estimate the bias-adjusted mean effect size, assuming that the publication probability bias is not symmetric around zero.

The results of the test are in table IV. We can see that negative and significant estimates are 200 times less likely to be published than positive, significant ones. In fact, negative estimates in general are less likely to be published than positive estimates, given what we would expect from an untruncated distribution of t-ratios. Although the standard errors are large and there is some overlap of 95% confidence intervals for the three estimated publication probabilities, the 95% intervals for negative estimates do not cover 1, suggesting negative estimates are far less likely to be published than positive and significant estimates. The estimate of the mean effect size, after adjusting for publication bias, is -0.642, but the 95% confidence interval again is relatively large and covers zero.

All tests suggest publication bias is present in the sample. The tests also suggest the true mean effect size of lead on crime may be close to zero, but this could be due to the relatively small sample, or to characteristics of the studies. These characteristics can be investigated more thoroughly with a meta-regression analysis.

4.4. Meta-Regression Analysis

Meta-regression analysis (MRA) follows from (4) where we include common observable variation in our estimation. Given all tests suggest the presence of publication bias we include the FAT in all regressions. We also weight all regression covariates by the standard errors as in (6). Therefore, the specification is the same as in (6) except we now also regress on further weighted observable covariates, and the coefficient on the precision is only an estimate of the average effect size when all other covariates are set to zero.

The covariates included are based on common characteristics of the studies that are suggested by the literature. Their descriptive statistics are included in table V. The majority are dummy variables indicating whether that characteristic is present for that estimate. All variables are coded at estimate level, not at study level. That is, different estimates from the same study may have different characteristics, and therefore have different values for the covariates. There is a dummy variable that equals one when an estimate comes from a high-quality study design that attempts to deal with endogeneity concerns. There is a dummy variable which is one when an estimate is of crime in an area, and zero when it is at the individual level. There are four dummy variables which indicate whether specific controls were included in the estimation. Lead exposure is correlated with poverty (Baghurst e al. 1999) and race (Sampson and Winter, 2016), may have different effects on men and women (Denno, 1990), and may have a relationship with educational outcomes (Fergusson, Boden and Horwood, 2008). Therefore, when an estimation includes these variables we might expect it to influence the estimate. The interpretation of the effect of these variables depends on where they are in the causal chain[9]. If these variables are confounders, causing changes in lead and changes in crime, then omitting them will tend to overstate the effect of lead on crime (given they change both in same direction). If they are mediators, changed by lead and then changing crime, then conditioning on them can lead to understating the effect of lead on crime. This is especially important when study designs do not use some method to deal with endogeneity issues. Of course, there are other variables that may be important controls, but these were not found to be common enough across studies to include.

Next there are three dummy variable that describe what type of crime was used as the dependent variable (homicide, violent, and non-violent), with a reference group of total crime. This allows us to test whether the different mechanisms proposed in section 2 matter. The violent crime category nests homicide within it. They are separate categories because homicide data is thought to be the best quality crime data, and thus less likely suffer from bias (Fox and Zatz, 2000). We next have two dummy variables representing possible estimation effects. One for if simple OLS was used, another for if maximum likelihood was used. The reference group is any other estimation such as GMM or mean

[9] Grateful to Paul Ferraro for his comments on this.

differences. We have two dummy variables for further estimation effects. One for if panel data were used, and another for if the results are reported as odds ratios. A further three dummy variables are geographic dummies that equal one when an estimate come from a continent, with a reference group of Africa. These are added as it allows us to see how much estimates differ depending on where the data come from. The final dummy variable equals one when a direct measure of lead, from either blood, bone, or dentine samples, is used in the estimation and zero when a proxy measure or estimate, such as leaded gasoline use in an area, is used. This allows us to test whether there is a systematic difference in effect sizes found when lead levels are taken directly from subjects, which we might expect to give a more accurate measure of the true effect, rather than proxied. The final two covariates are the sample size and the number of covariates included in the estimation. These two variables have been standardised to aid the restricted maximum likelihood convergence.

We estimate many specifications due to model uncertainty. Our sample is relatively small and coefficient estimation varies significantly in alternative specifications. The number of different covariate combinations is 2^K where K is the total number of covariates. It is common in the meta-analysis literature to employ some method of model averaging or shrinkage to deal with model uncertainty[10]. However, with this many covariates and modern computational power it is possible to estimate all 2^K specifications[11]. In addition, table II showed that some subsamples have substantially less heterogeneity than the full sample. It may be that these sub-samples suit aggregation better than the full sample. For example, we might expect studies with individuals as the unit of analysis to share much more common information than those that have a geographic area as the unit of interest. We therefore also estimate all covariate specifications for these subsamples. It is not possible to estimate every combination as some dummy variables no longer have any variation in the subsamples, leading to collinearity. This can also lead to other variables being excluded as they become the new base case (for example if there are no African studies in a subsample, then another continent becomes the base case). A full list of the covariates included for each subsample is in table VI. We estimate every possible

[10] For an example, see Gechert, Havranek, Irsova, and Kolcunova (2020)

[11] As a robustness check we perform Bayesian Model Averaging in the appendix. The posterior mean PCC using the full sample and evaluated at the sample averages is 0.06. Lower than the method we use here.

combination of covariates for the full sample and the subsample. We include the FAT, the estimate of publication bias. We estimate with REML and include study fixed effects. In total, we estimate over 1 million meta-regression specifications.

The distribution of coefficient sizes for the full sample estimation is in plotted in figure VII. The means, medians and standard deviations are given in table VII. We can see that the FAT mean and median is much smaller than the FAT coefficient in our tests in table III. This suggests some of the bias may be explained by observable heterogeneity between studies. However, there remains some residuals bias nonetheless, and this has a very large effect on the PCC estimate of lead on crime. The mean and median of the coefficient on the addressing endogeneity dummy is negative, suggesting studies that control for endogeneity tend to have smaller effect sizes. Studies which have an area as a unit of interest tend to find larger effects than those which use individuals. The coefficient on the area dummy has a mean of 0.2 and a median of 0.16. Almost all the density of this coefficient is positive in figure VII. The coefficient on whether a study directly measures lead levels has a negative mean and median, suggesting direct measures of lead levels lower the estimated effect of lead on crime compared to studies that use a proxy measure such as lead air pollution. When we turn to different kinds of crime, we can see that the distributions of both violent and non-violent crime peak around zero and have small means, and medians. This suggests having violent or non-violent crime as the dependent variable does not lead to systematically different estimates compared to the base case, total crime. It is different when the dependent variable is homicide. Here the mean and median size is -0.05 and every estimate is negative, suggesting that a dependent variable of homicide tends to lower the estimated effect size.

Looking at the four important controls, education and race tend to lower the PCC, while controlling for gender or income tend to raise it, but only the mean and median of the race control are further than 0.01 from zero. In estimation effects, OLS has a large negative mean and median, tending to lower the PCC, while maximum likelihood has a positive mean and median but not as large in absolute values. Using panel data and reporting odds ratios both tend to have a large negative effect on the size of the lead/crime estimate. For continent dummies, an estimate using North American data has a strong negative effect on the PCC, with a mean of -0.28 and median of -0.29. Europe and Australia on the other hand tend to raise the PCC relative to the base case, Africa.

However, by far the majority of estimates, 70%, use North America data, compared with 16% using European data, and around 7% each for Australasia and African data. More covariates tend to lower the PCC, with a mean of -0.1 and median of -0.11. Finally, the sample size does not seem to affect the estimate of lead on crime, beyond the publication bias effects in the FAT. This is reassuring, as it means small sample studies are not estimating systematically different effect sizes, it is just that they tend to only be published when they are positive and significant.

Overall, for the full sample the results show that observable variation accounts for some of the large differences in effect sizes we observe in the sample. By the far the largest coefficient is our estimate of publication bias. Beyond that, a group of other variables have large effects: whether a study uses data from North America; estimation effects, such as using panel data or reporting odds ratios; whether a study examines individuals or areas, and how many covariates an estimate includes.

We next use the information from each specification to construct a distribution of estimates of the average effect of lead on crime. We are now estimating an average effect conditional on the observable heterogeneity in our specifications. In practice, meta-analysis tends to do this in two ways, either using the sample averages or taking some "ideal" specification. We do both. That is, for each specification we generate a predicted PCC estimate of the effect of lead on crime, using both the sample averages, or by using an ideal specification, and not including the FAT in the predicated value (i.e. removing the publication bias). The ideal specification we use is one that includes controls for race, education, income and gender, that uses individual data, directly measured lead levels, controls for endogeneity, uses panel data, is estimated with GMM (i.e base case compared to OLS and ML), uses total crime as the dependent variable, uses North American data (as most of our sample is from there), and uses the sample averages for sample size and number of covariates. This ideal specification is chosen to represent a robust and high-quality estimation, and as such we would expect it be generally lower than the sample averages estimates.

We plot the kernel density distributions for the full sample for using both sample averages and the ideal specification in figure VIII. In each there is a distribution of 524,288 predicted values. The mean and median PCC for the sample averages

distribution are 0.11 and 0.12 respectively, which is "moderately positive" according to the Doucouliagos (2011) taxonomy (in section 4.5 we look at what these imply for elasticities). The distribution appears to be bimodal with one peak close to zero and the other around 0.2. The distribution of the ideal specification is not bimodal and the density peaks close to zero and is roughly symmetrical. The mean and median are -0.05 and -0.02 respectively. As expected the ideal specification is lower than the sample averages, but this is in part due to the North America variable. An alternative specification where with the "Europe" dummy set to one instead gives a mean and median of 0.11 and 0.04. The median though is still much lower than in the sample averages distribution.

We next restrict the sample to only high-quality studies that estimate a causal effect rather than an association: our "addressing endogeneity" sub-sample. This consists of seven studies and 211 estimates. It is common in meta-analysis to exclude correlational studies altogether (e.g., Kraft, Blazer and Hogan, 2018). Although we have not excluded those studies in this meta-analysis, we now examine what a meta-analysis estimate with only causal studies would be. We saw in table II that the addressing endogeneity subsample has lower between-study heterogeneity than the full sample, so aggregation may yield comparatively more information.

We plot the sub-sample average specification and ideal specification in figure IX (excluding those variables that cannot be included in the estimation, see table VI). The distribution of the sample average predicated values is tight around zero with a mean and median of 0.01, and a sample standard deviation of 0.01. The ideal specification may be expected to perform less well as we have fewer variables in the addressing endogeneity sample. The mean is -97 due to a few negative estimates with large absolute values, and around 13% of the distribution is outside the feasible interval of a PCC [-1,1]. However, most of the density is once again close to zero and the median is 0.02. The results suggest there is a systematic difference between the high-quality causal estimating studies and the rest of the sample.

We next plot several other subsample distributions of interest in figure X. The means medians and standard deviations for the full sample and all subsamples are given in table VIII. We also show the percentage of estimates which fall outside the feasible [-1,1] interval, which suggests misspecification. The difference between the area and individual

sample is striking. The area sample means and medians are much larger than the individual sample for both the sample average specification and the ideal specification. The individual sample mean and median PCCs are close to zero, although larger in all cases, and the distributions are tight around the means compared to the area sample. This suggests that covariates matter less for the individual sample effect sizes compared to the area sample. Similar to the area-individual comparison, the correlational sample has much higher means and medians than the addressing endogeneity sample.

Comparing homicide, violent, and non-violent crime samples we can see again in table VIII that a large part of the distributions are outside the feasible interval of a PCC, suggesting misspecification and that the results may not be reliable. In the sample average specifications, the non-violent and homicide samples are the only ones with some percent of the distribution outside [-1,1], suggesting it may be an issue with those samples. The standard deviations for all the crime samples are large even when we only look at the sample average specifications in table VIII. Overall, the results suggest these samples may be less informative than the others, but what we can say is that they show positive, moderate to large, mean and medians PCCs. The non-violent and homicide samples generally show larger PCCs than the violent crime sample but, given the large variance of the distributions and the number of unfeasible PCC values returned, this may be due to model or sample issues. We can say that violent crime does not have a stronger relationship with lead compared to non-violent crime. We cautiously suggest that if lead does have an effect on crime it is across all categories of crime.

4.5. Estimating an Elasticity

We use PCCs to estimate the effect of lead on crime in this meta-analysis because studies use varying measures of lead and crime, with varying units of interest, and so cannot be directly compared. We find that the median PCC, evaluated at the sample averages, for the full sample is 0.11. This is a “small effect”, according to the Doucouliagos (2011) taxonomy. The equivalent PCC when only the high-quality, addressing endogeneity sample is considered is 0.01, below the threshold for a small effect according to Doucouliagos, and close to zero. However, a small effect size does not necessarily imply an economically insignificant one. When there are very large changes in a variable even small effects can sum to a huge change in welfare. Lead levels have dropped significantly

since the 80s in many countries as shown in figure II. We therefore examine plausible estimates of a lead-crime elasticity, using our PCC estimates. The PCC and the elasticity are related, but not in a straightforward manner. This forces us to make some strong assumptions in the interests of welfare analysis.

Given a PCC and the change in a given measure of crime for a given measure of lead, $\frac{\delta Crime}{\delta Lead}$, then the relationship between the two is given in (7).

$$(7) \qquad PCC = \frac{\delta Crime}{\delta Lead}\frac{sd(Lead)}{sd(Crime)}\frac{sd(\widetilde{Lead} - \tilde{\mathbf{z}}'\gamma_1)}{sd(\widetilde{Crime} - \tilde{\mathbf{z}}'\gamma_2)}$$

Where $sd(.)$ means the standard deviation. $\widetilde{Lead} - \tilde{\mathbf{z}}\gamma_1$ are the residuals from a regression of *Lead* on $\boldsymbol{z}$, a vector of variables related to lead and crime, where both lead and $\boldsymbol{z}$ have been standardised. Similarly, $\widetilde{Crime} - \tilde{\mathbf{z}}\gamma_2$ are the residuals from a regression of *Crime* on $\boldsymbol{z}$**,** where both have been standardised. If we wish to attach a causal interpretation to the elasticity, we can think of $\boldsymbol{z}$, following Peters , Bühlmann, and Meinshausen (2016), as the minimum set of variables under which the distribution of *Crime* is invariant when conditioned on both $\boldsymbol{z}$ and *Lead*.

It can be seen that a PCC will always share the same sign as $\frac{\delta Crime}{\delta Lead}$ but will be inflated or deflated according to the relative size of the standard deviations in (7). $\frac{\delta Crime}{\delta Lead}\frac{sd(Lead)}{sd(Crime)}$ is equivalent to a standardised coefficient. The intuition for the last ratio is as follows: the greater the variation in *Lead* that is not explained by $\boldsymbol{z}$, the larger the PCC, because the overlapping variation between the independent effect of *Lead* and *Crime* is relatively greater. The PCC is also greater the larger the amount of variation in *Crime* explained by $\boldsymbol{z}$**.** This is because the share of unexplained variation in *Crime* becomes smaller, so the share of variation jointly explained by *Lead* and $\boldsymbol{z}$ increases. As more of the variation in *Crime* is explained by both *Lead* and $\boldsymbol{z}$**,** their PCCs will tend to 1 or -1.

To evaluate an elasticity at the sample means we multiply both sides by $\frac{\overline{Lead}}{\overline{Crime}}$, where the bar indicates the mean. We can then rearrange (7) to put it in terms of the elasticity η.

$$(8) \qquad \eta = \frac{\overline{Lead}}{\overline{Crime}}\frac{sd(Crime)}{sd(Lead)}\frac{sd(\widetilde{Crime} - \tilde{\mathbf{z}}'\gamma_2)}{sd(\widetilde{Lead} - \tilde{\mathbf{z}}'\gamma_1)}PCC$$

We can see that the size of the PCC relative to the elasticity depends on three ratios. The first two, the relative means and standard deviations, depend on the measures of crime and lead. We use homicide and blood lead data from the US as an illustrative example to examine plausible elasticities, given the fall in both violent and non-violent crime was particularly pronounced there. The means, standard deviations, and sources are given in table IX. Given these, the relative size of the PCC to the elasticity depends upon the third ratio of residual standard deviations. This ratio could theoretically take any value between zero and infinity, and therefore so could the elasticity (assuming the PCC is positive). We therefore look at what are plausible values for this ratio and what is the range of the elasticity given these values.

The maximum value the numerator $sd\left(\widetilde{Crime} - \tilde{\boldsymbol{z}}'\gamma_2\right)$ can take is one, representing no common variation between $\boldsymbol{z}$ and *Crime*. We hold it at one, to inflate the PCC as much as possible. The final element of the equation is $sd(\widetilde{Lead} - \tilde{\boldsymbol{z}}'\gamma_1)$. This is the residual variation in *Lead* not explained by $\boldsymbol{z}$**.** The lower this is, the more the PCC will be inflated, and therefore the greater the elasticity. The elasticity is convex in $sd(\widetilde{Lead} - \tilde{\boldsymbol{z}}'\gamma_1)$, decreasing at a decreasing rate.

Figure XI plots the relationship between the elasticity and $sd(\widetilde{Lead} - \tilde{\boldsymbol{z}}'\gamma_1)$, given the estimated mean PCCs, the values in table IX, and holding $sd\left(\widetilde{Crime} - \tilde{\boldsymbol{z}}'\gamma_2\right)$ constant at the maximum value of one. The elasticities drop sharply with an increase in the denominator $sd\left(\widetilde{Crime} - \tilde{\boldsymbol{z}}'\gamma_2\right)$, with the elasticity for the addressing endogeneity sample approaching close to zero almost immediately. The elasticity for the full sample slopes down more gently but even so does not suggest a large elasticity except at extremely small values of $sd\left(\widetilde{Crime} - \tilde{\boldsymbol{z}}'\gamma_2\right)$.

We can now propose a range of plausible values for the elasticity. Given the uncertainties around the ratio of unexplained variations in (8), this is somewhat arbitrary, but we hope, given the discussion above, not unreasonably so. There is no compelling reason to suppose $\boldsymbol{z}$ would explain more of the variation in *Lead* than in *Crime*. Nevertheless, if we take as a lower bound that $sd(\widetilde{Lead} - \tilde{\boldsymbol{z}}'\gamma_1)$ is ten times as large as $sd\left(\widetilde{Crime} - \tilde{\boldsymbol{z}}'\gamma_2\right)$, and as a conservative upper bound that they are equal, then we can give a range of values based on our estimated PCCs. For the full sample PCC, this gives an elasticity of 0.22-0.02.

For the addressing endogeneity sample PCC, the range is 0.03-0.00, to two decimal places. The median blood lead level in children fell 88% from 1976-2009. The full sample elasticity estimates therefore would suggest the fall in lead has decreased homicide in the US by between 19.4% and 1.8%. The equivalent decrease for the addressing endogeneity sample is between 2.6% and 0%. The US homicide rate fell 54% from its peak in 1989 to 2014. This would mean that lead accounts for between 36% and 3% of the decrease in homicide using the full sample elasticity, and 5%-0% using the addressing endogeneity elasticity. Our generous assumptions of the lower bound on the ratio of residual variation in (8) imply that lead may be an important factor in the fall in homicide, but it does not account for the majority of the fall. Our upper bound on that same ratio implies lead accounts for very little of the fall in crime.

5. Discussion and Conclusion

Changes to the amount of lead in the environment have been put forward as one of the main causes of the decrease in crime, especially homicide, in many western countries. We performed the first meta-analysis of the effect of lead on crime. We find there is publication bias in the lead-crime literature, and that meta-analysis estimates that do not control for this will overstate the effect of lead on crime. We find that the average meta-analysis estimate for high-quality studies that take into account endogeneity is much smaller than for the full sample, or for the correlational sample. Similarly, the average effect size estimate for studies that have individuals as the unit of interest is much smaller than for the sample of studies that have a geographic area as the unit of interest. The full sample and area sample distributions have most of their density in the positive side of zero. Their average effects suggest a small to moderate effect, while both the addressing endogeneity and individual effect distributions are tight around zero and suggest there may be no effect. When we examined the differences between lead's effect on homicide, violent and non-violent crime, we could not confidently state there was any difference between them. Finally, we performed back-of-envelope calculations to convert our partial correlation coefficient estimates into elasticities. This gave a range of 0.22-0.02 for the full sample and 0.03-0.00 for the high-quality, addressing endogeneity sample.

This suggests the fall in blood lead levels may have led to a fall in homicide in the US of between 36-3% with the full sample elasticity, and between 5%-0% for the addressing endogeneity sample elasticity.

Overall, the results suggest that declines in lead pollution are not the cause of the majority of the fall in crime observed in many western countries. Our results however leave open the possibility that it may have a socially significant effect. The upper end of our range of elasticities would imply the lower lead pollution today saves around 6,000 lives a year in the US. The lower end, however, would mean lead has no effect and we must look to other causes entirely. We are unable to provide estimates on the size of other causes here but hope our results can provide a rough benchmark for relative importance in future meta-analysis. It is possible that the large differences in our samples can be reconciled. For example, the large difference between the individual and area samples may be because crime has fallen at the extensive margin rather than the intensive margin. Tcherni-Buzzeo (2019) observe that around 5% of the population are responsible for 50% of crime, and that the fall in crime in the US is likely due to falls in this high-crime population, rather than less crimes per individual in that population. If less lead pollution only meant less probability of committing crime for this small slice in the population, it might nevertheless lead to a large fall in crime at the area level.

There are a number of limitations to our analysis. Most importantly, the sample size is not large. We have 24 studies and 529 estimates, this is not unusual for a meta-analysis but, particularly for our subsample estimates, this could play a part in the differences. It may explain why so much of the distribution for the different types of crime in table VIII were outside the feasible PCC interval of [-1,1]. We attempt to mitigate this by using various tests for publication bias, and estimating many different specifications, but we cannot rule out that the results are due to small sample effects. Secondly, the between-study heterogeneity is large in our sample. This calls into question how comparable the studies are. This is to be expected as studies use different concepts and measures of crime and lead, different units of interest, and different estimations, from simple correlations to LATEs. We try to mitigate this by converting to PCCs, using different sub-samples that have lower between-study heterogeneity, and using meta-regression with covariates. However, even with these mitigations, it may be that the literature is not comparable and therefore meta-analysis estimates will be noise. In this case it casts doubt on the external

validity of the studies examining the lead-crime hypothesis. The solution would be far more studies that estimate elasticities using comparable measures of lead and crime.

For policymakers, our results are a warning against assuming the large crime levels in past decades cannot return now that lead pollution is much lower. The results are not a signal that lead abatement is fruitless. As outlined in section 2, the evidence of harmful biological and health changes due to lead is overwhelming. There is no known safe level of lead. Even if outcomes higher up the causal chain, such as crime, are not as affected by lead, the evidence still shows lead abatement will increase health outcomes, especially for the very young.

For future research, we have two main suggestions. The first is that there are enough low sample size, correlational studies in the lead-crime literature. What is needed now is high power, high-quality causal estimates of the effect of lead on crime. The value added of such studies would be increased by testing the effect on different types of crime, and the possible interaction of lead with other potential causes. The second is that more high-quality causal estimates of the elasticity of other causes of crime are needed. Our results suggest lead is not responsible for the majority of the fall in crime since the 80s and therefore leaves open room for other explanations. These explanations must account for the fact homicide has fallen across many (but not all!) western countries at roughly the same time. They must also account for the fact that total crime has risen in Europe and fallen in the US, while the homicide rate has fallen in both. Further comparison of the relative shares of responsibility for the fall in crime, as well as the interaction between causes, may also be fruitful and we suggest further meta-analyses, using the up-to-date methods, would be helpful in this area.

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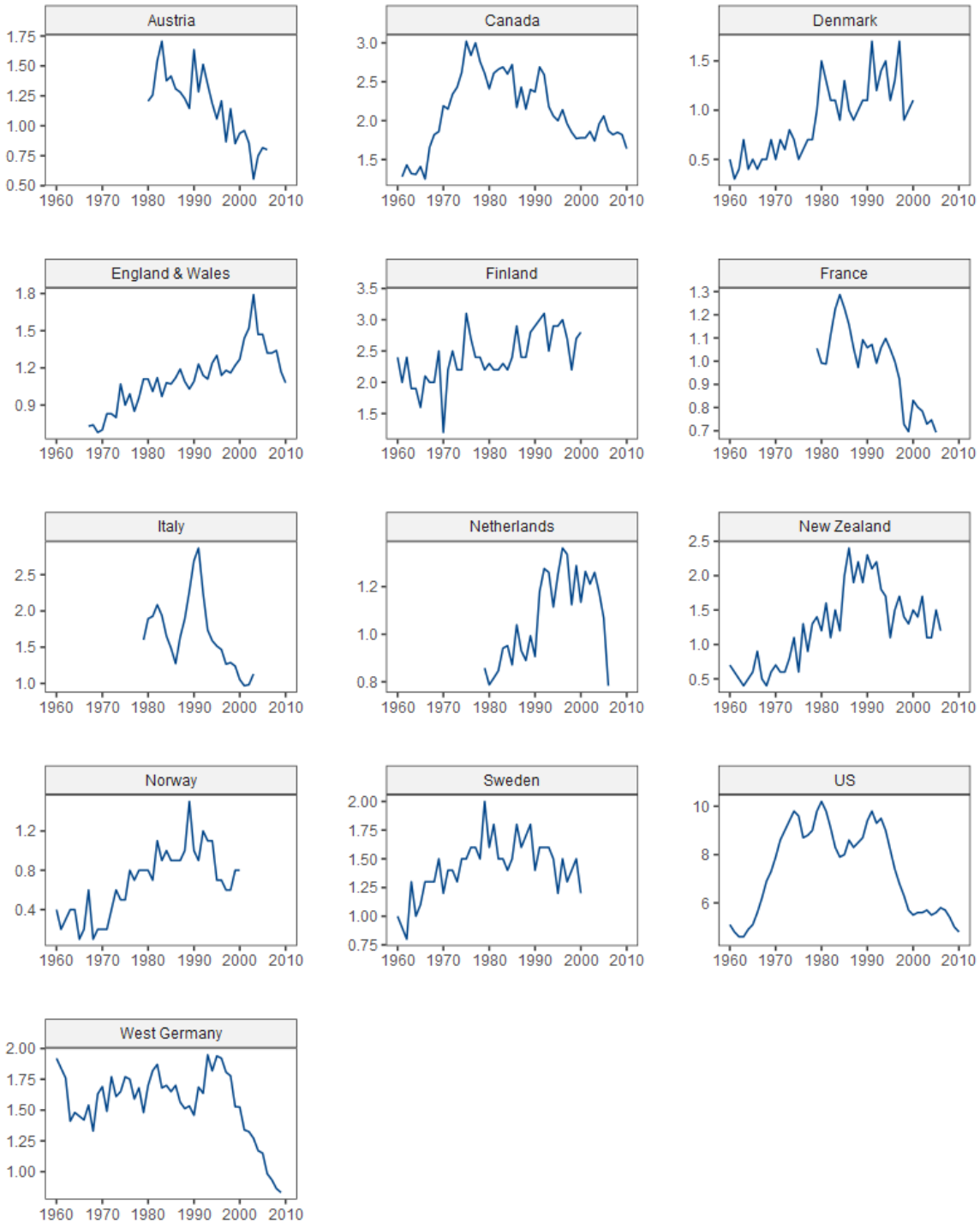


Figure I

Homicide rate per 100,000 by country

Sources: New Zealand Police (2018); Buonanno *et al*. (2011), UK Home Office (2019); Uniform Crime Reports for the United States (2019); Falck, Von Hofer & Storgaard (2003); Statistics Canada (2019); Birkel and Dern (2012).

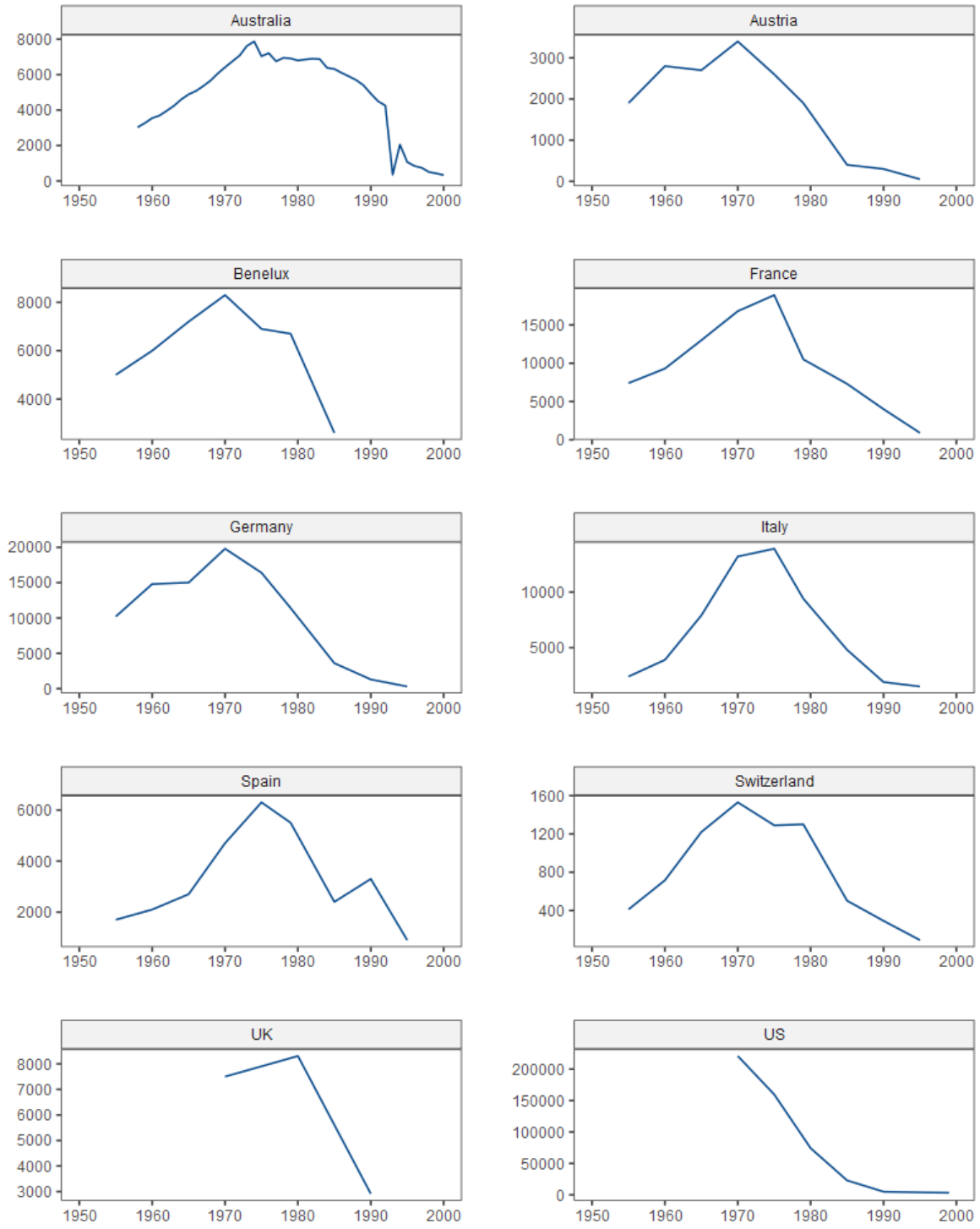


Figure II

Lead Emissions by Country (1000 kg Y^{-1})

Source: Dore *et al*. (2006), Schwikowski *et al*. (2004), Kristensen (2015), Statistical Abstract of the United States (2009).

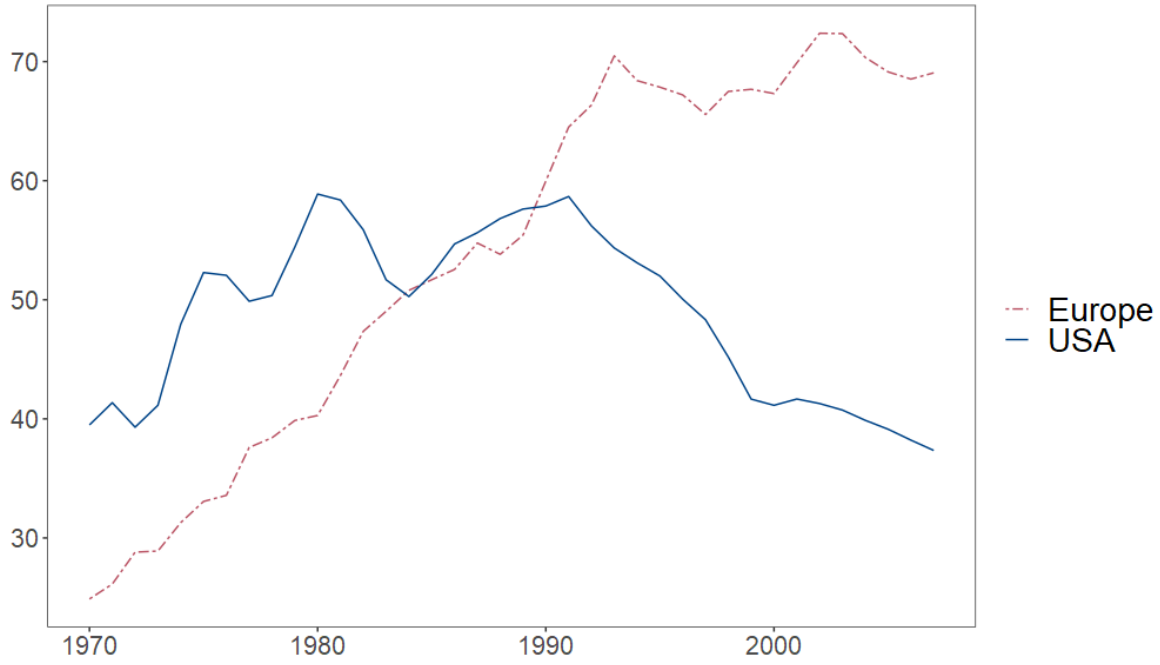


Figure III

Total recorded crime rate per 100,000 in USA and seven European Countries

Source: Buonanno *et al*. (2011). The countries are: Austria, France, Germany, Italy, The Netherlands, Spain, and the UK.

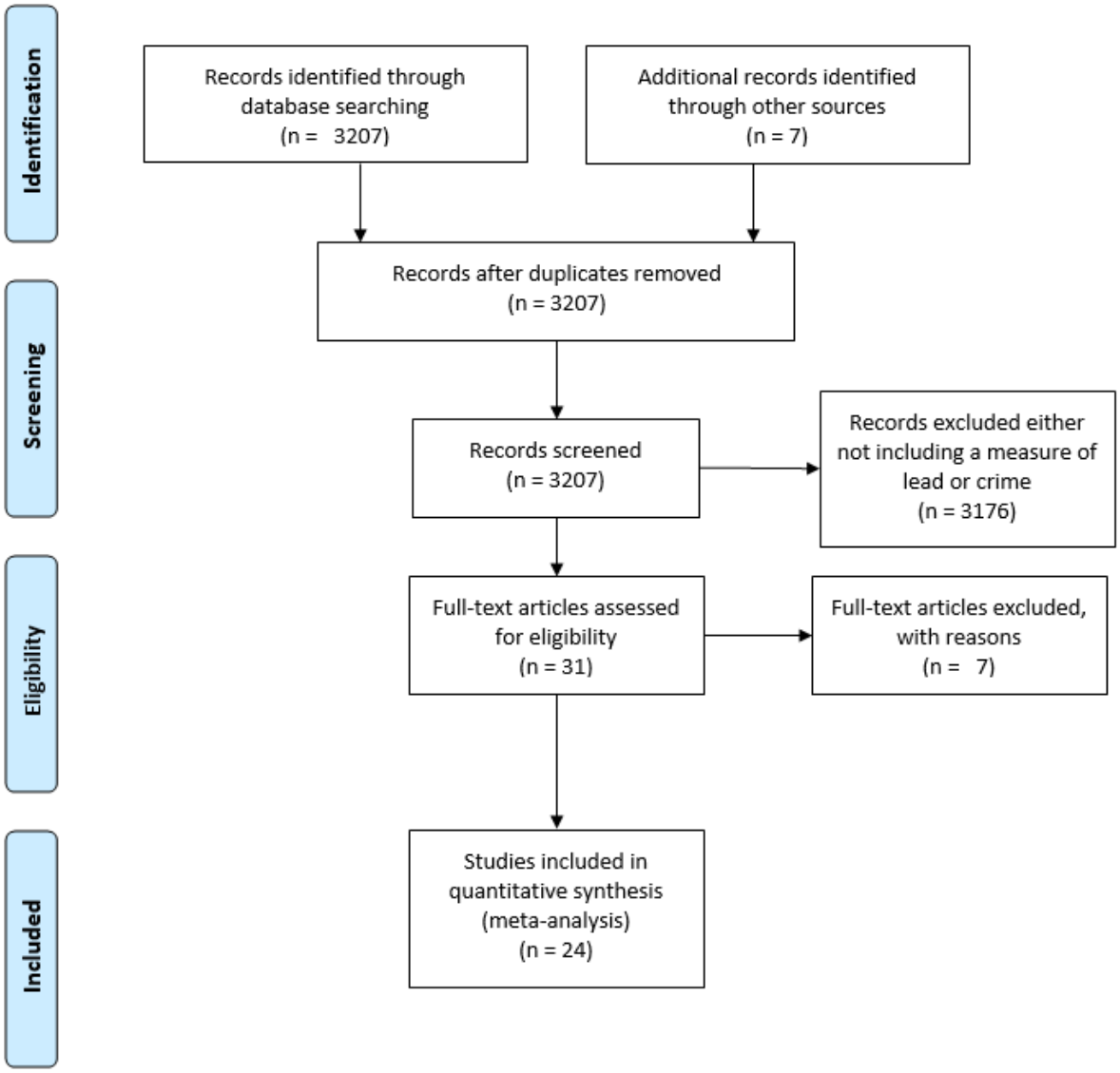


Figure IV

PRISMA Flow Diagram of selection process

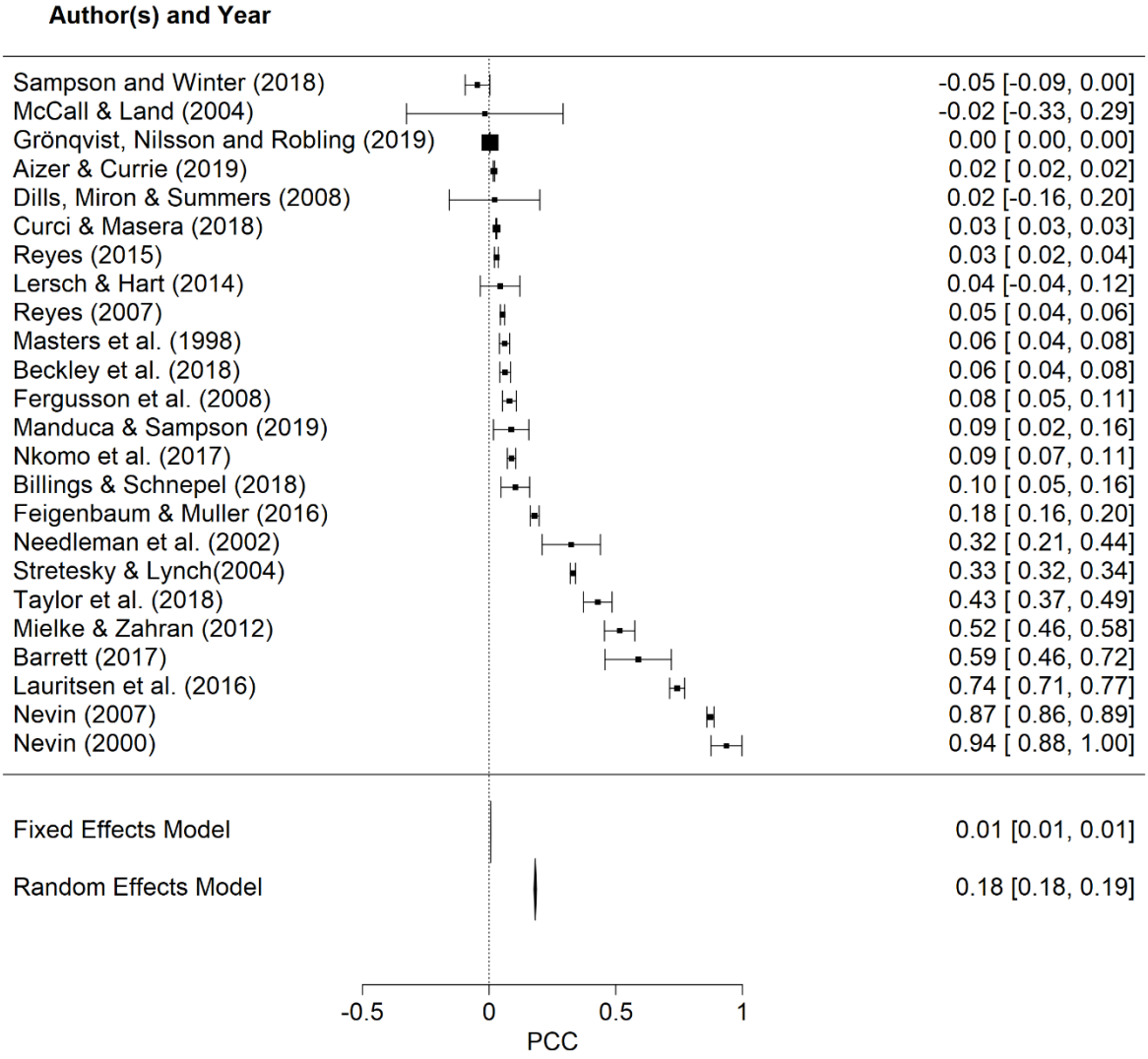


Figure V

Forest plot

Notes. Chart shows weighted average partial correlation coefficients (PCCs) of each study's effect size along with corresponding 95% confidence intervals. The weighted averages are calculated by first normalizing the PCCs so that confidence intervals can be constructed, then the fixed effects average is calculated, finally the estimates are converted back to PCCs (see appendix for details).

Bottom of table shows fixed effects and random effects estimates for all studies combined (see appendix for details).

Numbers on right are the point estimates and the 95% confidence intervals.

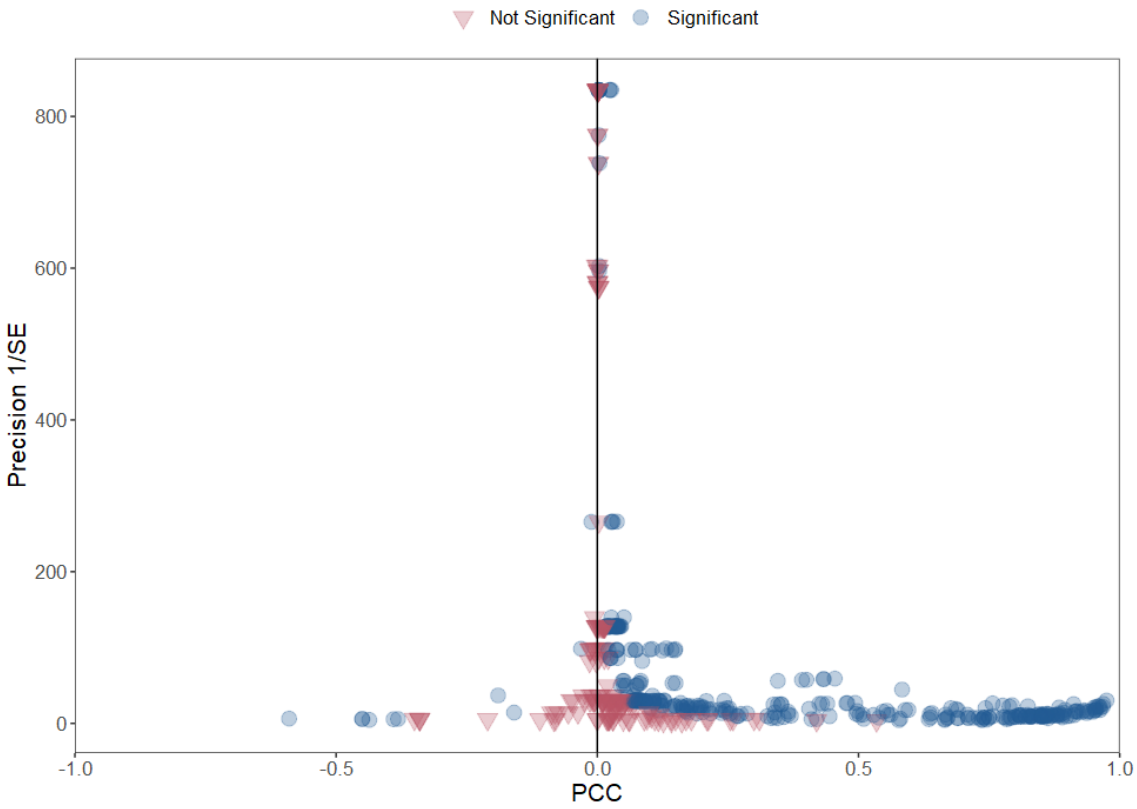


Figure VI

Funnel chart

Notes. PCC = Partial Correlation Coefficient

Precision is one divided by the standard error of the PCC.

"Significant" means statistically significant at the 95% confidence level using two-sided critical values of a normal distribution.

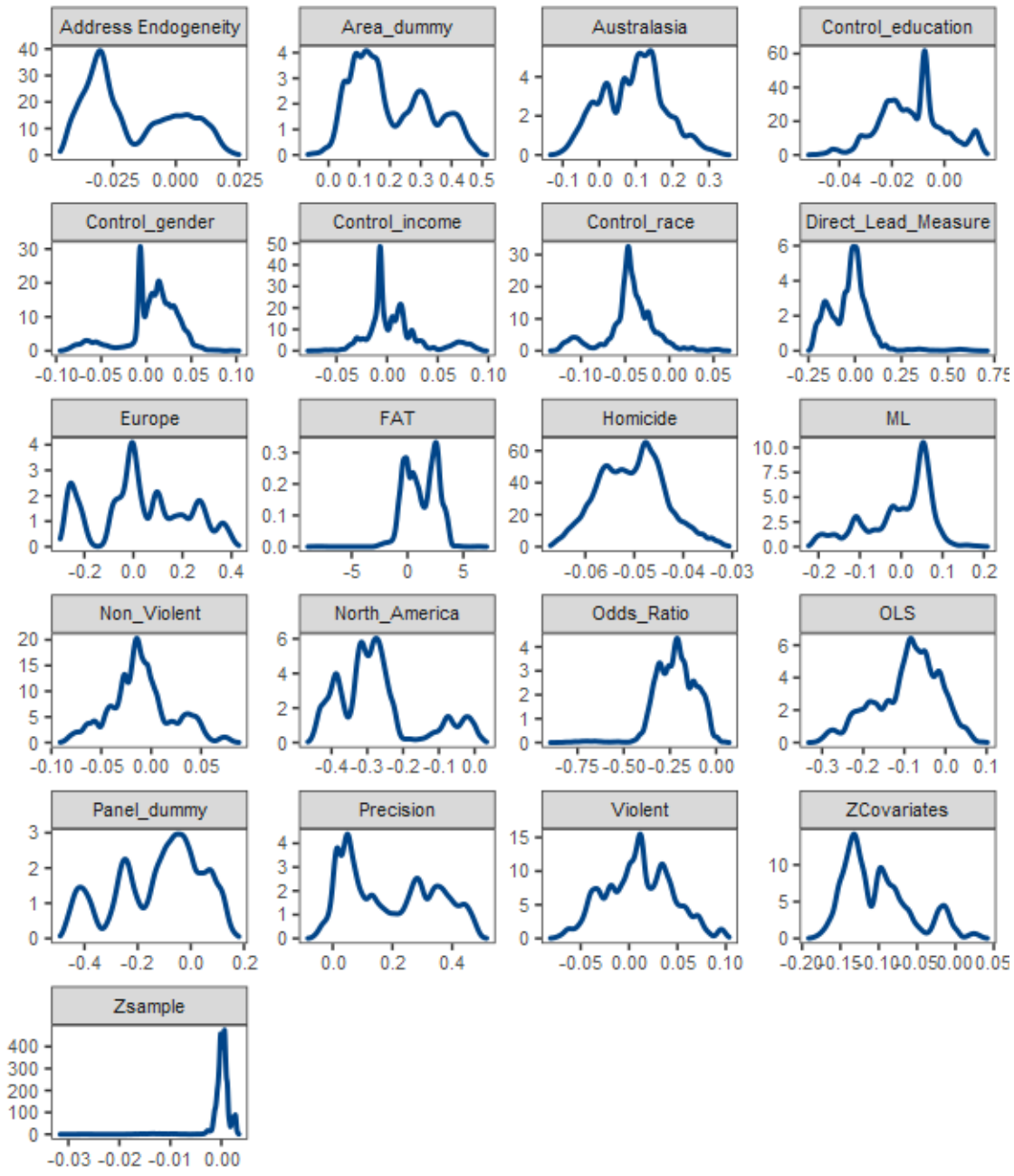


Figure VII

Densities of covariates in full sample meta-regressions

Notes. Chart shows kernel densities for the meta-regression estimated coefficients on each covariate. Note different axes scales.

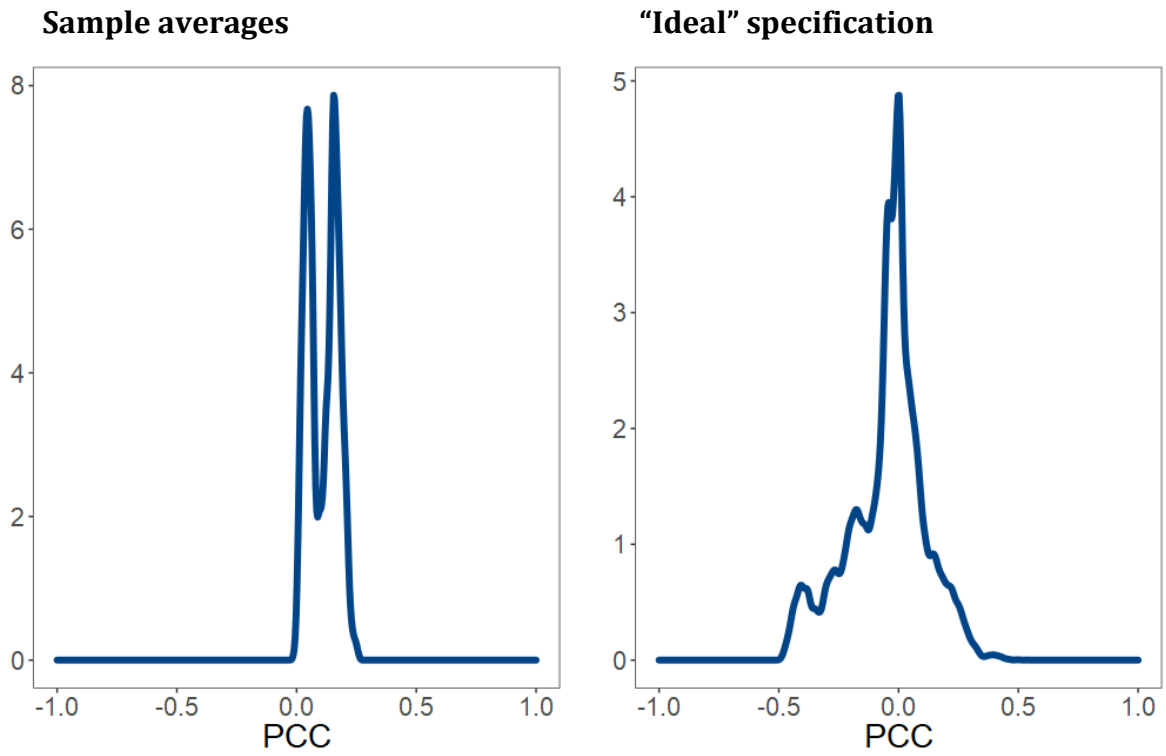


Figure VIII

Density of meta-analysis average effect size estimates from full sample

Notes. Chart shows kernel densities for the distribution of meta-regression estimated average effect sizes. Chart on left shows estimated average effect for each specification evaluated at the sample averages. Chart on right shows estimated average effect for each specification evaluated-at an "ideal" specification. X axis truncated at feasible interval of a PCC, [-1,1].

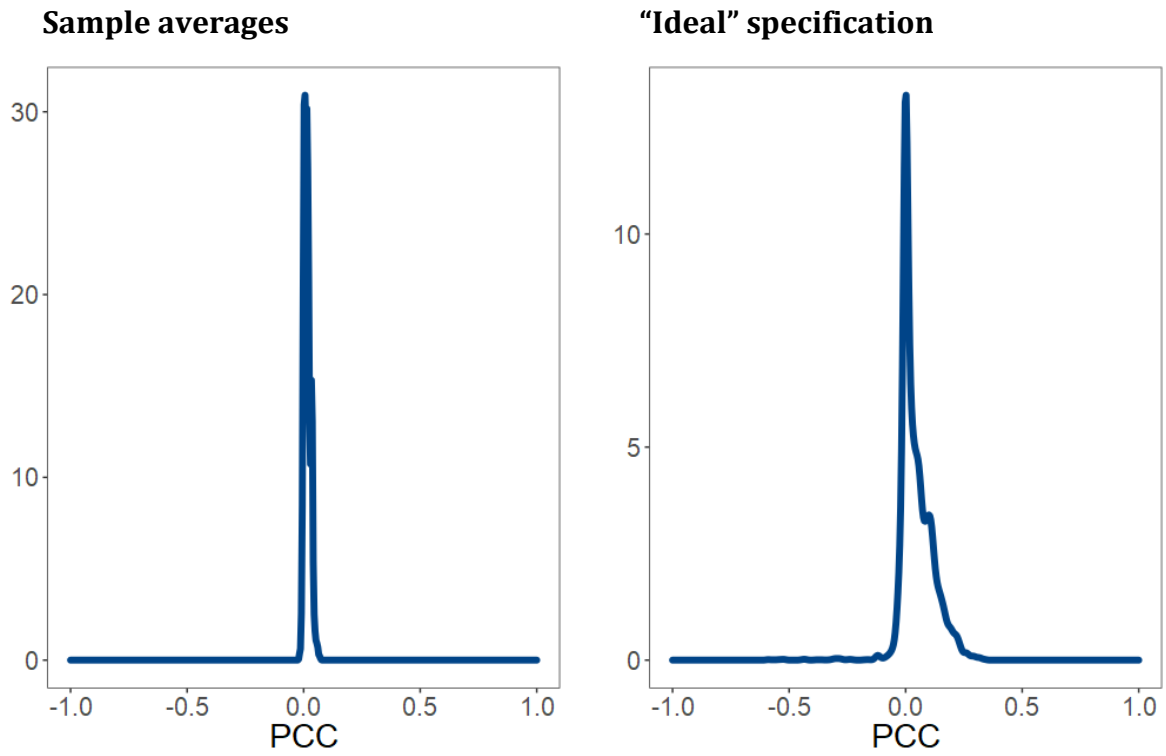
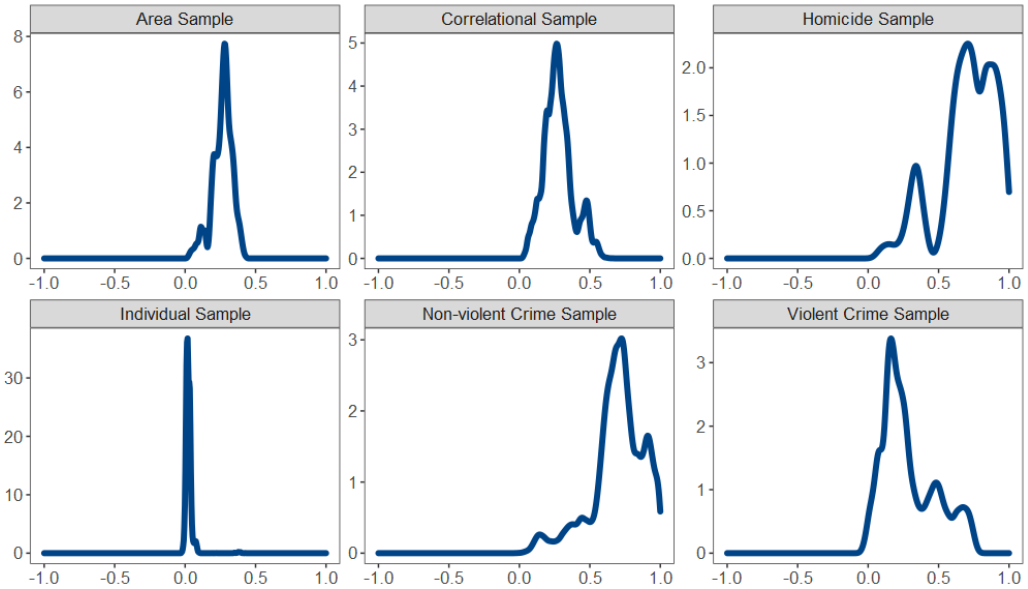


Figure IX

Density of meta-analysis average effect estimates for "Addressing Endogeneity" subsample

Notes. Chart shows kernel densities for the distribution of meta-regression estimated average effect sizes for the addressing endogeneity sub-sample.. Chart on left shows estimated average effect for each specification evaluated at the sample averages. Chart on right shows estimated average effect for each specification evaluated-at an "ideal" specification. X axis truncated at feasible interval of a PCC, [-1,1].

Sample averages



"Ideal" specification

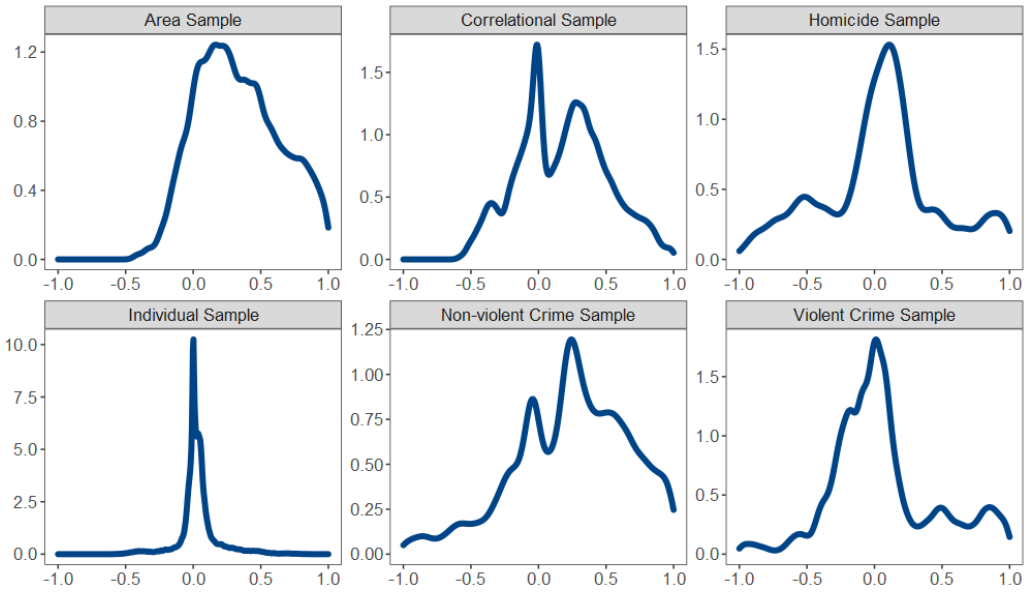


Figure X

Densities of Meta-analysis average effect estimates from subsamples

Notes. Chart shows kernel densities for the meta-regression estimated average effect sizes for a number of subsamples. Top chart shows estimated average effect for each specification evaluated at

the sample average for each subsample. Chart on right shows estimated average effect for each specification evaluated-at an "ideal" specification. X axes truncated at feasible interval of a PCC, [-1,1].

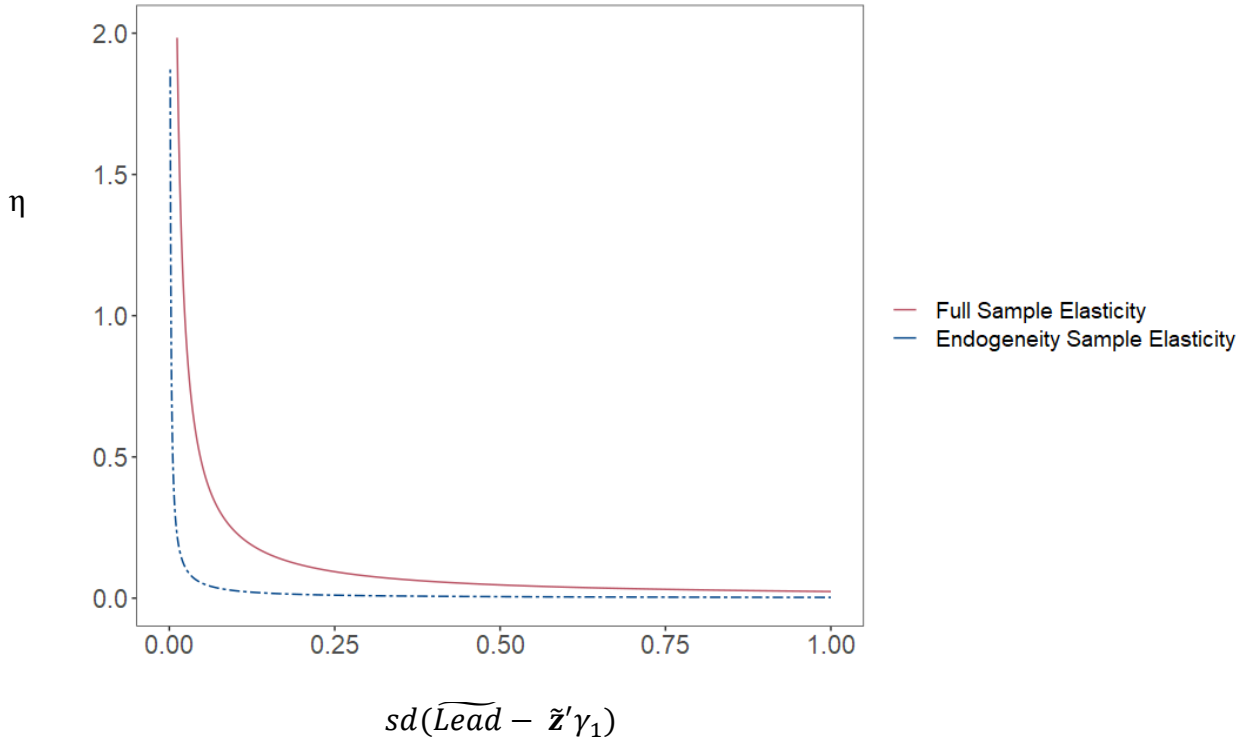


Figure XI

Estimated Elasticity of on lead on crime

Notes. Chart shows how η, the calculated elasticity of lead on crime, varies with changes in $sd(\widetilde{Lead} - \tilde{\mathbf{z}}'\gamma_1)$, the standard deviation of the residual in a regression of a set of standardised variables $\tilde{\mathbf{z}}$, and the standardised measure of lead $\widetilde{Lead}$.

Table I – Studies Used in Meta-analysis

Study & Year	Median	Mean	Weighted Average	Type of Crime	Individual or Area	Addresses Endogeneity
Aizer & Currie (2019)	0.027	0.019	0.019	Violent and non-violent	Individual	Yes
Barrett (2017)	0.556	0.556	0.589	Violent	Area	No
Beckley *et al.* (2018)	0.065	0.061	0.063	Violent and non-violent	Individual	No
Billings & Schnepel (2018)	0.122	0.113	0.103	Violent and non-violent	Individual	Yes
Curci & Masera (2018)	0.025	0.043	0.029	Violent	Area	Yes
Dills, Miron & Summers (2008)	0.022	0.021	0.021	Violent and non-violent	Area	No
Feigenbaum & Muller (2016)	0.189	0.192	0.180	Only Homicide	Area	Yes
Fergusson *et al.* (2008)	0.080	0.079	0.080	Violent and non-violent	Individual	No
Grönqvist, Nilsson and Robling (2019)	0.002	0.003	0.003	Violent and non-violent	Individual	Yes
Lauritsen *et al.* (2016)	0.740	0.495	0.742	Violent and non-violent	Area	No
Lersch & Hart (2014)	0.043	0.043	0.043	Violent and non-violent	Area	No
Manduca & Sampson (2019)	0.087	0.087	0.087	Violent and non-violent	Individual	No
Masters *et al.* (1998)	0.051	0.061	0.061	Violent and non-violent	Area	No
McCall & Land (2004)	-0.017	-0.017	-0.017	Only Homicide	Individual	No
Mielke & Zahran (2012)	0.526	0.497	0.515	Violent	Area	No
Needleman *et al.* (2002)	0.336	0.307	0.324	Non-violent	Individual	No
Nevin (2000)	0.914	0.912	0.937	Violent	Area	No
Nevin (2007)	0.808	0.710	0.874	Violent and non-violent	Area	No
Nkomo *et al.* (2017)	0.004	0.052	0.088	Violent	Individual	No
Reyes (2007)	0.059	0.053	0.053	Violent and non-violent	Area	Yes
Reyes (2015)	0.026	0.036	0.029	Violent and non-violent	Individual	Yes
Sampson and Winter (2018)	-0.065	-0.046	-0.046	Violent and non-violent	Individual	No
Stretesky & Lynch(2004)	0.396	0.352	0.331	Violent and non-violent	Area	No
Taylor *et al.* (2018)	0.371	0.377	0.429	Violent	Area	No

Notes. Table shows median and mean partial correlation coefficient (PCC) estimates from each study of the effect of lead on crime. It also shows an average where estimates are combined in a weighted average with the weights equal to one divided by the standard error. Table also shows what type of crime was used as dependent variable in each study, whether the study unit of interest was an individual or a geographic area, and whether any estimates in the study used a design that attempted to account for endogeneity. All coding is done at an estimate level, so a study may include both "addresses endogeneity" and "correlational" estimates, violent and non-violent estimates etc.

Table II – Meta-Averages and Heterogeneity Estimates by Subsample

Sample	RE Estimate	SE	$\hat{\tau}^2$	$\hat{I}^2$	$\hat{H}^2$	Studies	Estimates
Full Sample	0.182	0.002	0.002	99	111	24	529
Addressing Endogeneity	0.013	0.001	0.000	90	10	7	211
Correlational	0.519	0.015	0.067	99	160	20	318
Individual	0.008	0.001	0.000	95	20	11	125
Area	0.414	0.010	0.037	99	128	13	404
Homicide	0.298	0.025	0.048	95	19	8	93
Violent Crime	0.290	0.008	0.018	99	75	18	328
Non-Violent Crime	0.503	0.043	0.140	99	142	15	80
Total Crime	0.077	0.003	0.001	99	152	11	119
North America	0.243	0.007	0.012	98	61	19	373
Europe	0.069	0.003	0.001	100	201	2	85
Australasia	0.507	0.094	0.357	99	149	4	41
Direct Lead Measure = TRUE	0.092	0.026	0.031	95	19	9	54
Direct Lead Measure = FALSE	0.190	0.003	0.002	99	121	15	475
Representative Estimate = TRUE	0.195	0.021	0.007	98	54	24	24
Representative Estimate = FALSE	0.184	0.003	0.002	99	114	24	505
Control Gender = TRUE	0.007	0.001	0.000	95	20	8	103
Control Gender = FALSE	0.382	0.007	0.018	99	127	18	426
Control Race = TRUE	0.134	0.010	0.008	97	34	13	104
Control Race = FALSE	0.192	0.003	0.002	99	129	14	425
Control Income = TRUE	0.028	0.002	0.000	97	31	13	174
Control Income = FALSE	0.433	0.008	0.017	99	145	16	355
Control Education = TRUE	0.006	0.001	0.000	95	19	11	106
Control Education = FALSE	0.372	0.007	0.016	99	128	17	423

Notes. RE Estimate is a random effects, meta-analysis estimate computed using DerSimonian-Laird (1986) method. SE is the standard error. τ^2, $\hat{I}^2$, and $\hat{H}^2$ are estimates of between-study heterogeneity. See section 4.2 for more details.

Table III – Bias and average effect estimates

Variable	FAT-PET	FAT-PEESE	Multi-level FAT-PET
$\hat{\beta}_F$	5.087	32.479	3.562
	(1.291)	(8.474)	(0.880)
$\hat{\theta}$	-0.003	0.005	0.005
	(0.002)	(0.002)	(0.004)

Notes. Estimates presented with their standard errors in brackets. FAT-PET is Funnel Asymmetry test and Precision Effect Test. FAT-PEESE is Funnel Asymmetry Test and Precision Effect Estimate with Standard Error. The multi-level FAT-PET is a mixed effects-multi-level model with a different slope coefficient for each study. $\hat{\beta}_F$ is the estimate of bias in a meta-analysis sample. $\hat{\theta}$ is the estimate of the "true" average effect size accounting for the estimated bias $\hat{\beta}_F$.

Table IV – Estimated Meta-average and relative publication probabilities using Andrews-Kasy method

Meta-average	**Relative publication probabilities, where reference p($1.96 \leq Z$) is 1**		
$\boldsymbol{\theta}$	$\boldsymbol{Z < -1.96}$	$\boldsymbol{-1.96 \leq Z < 0}$	$\boldsymbol{0 \leq Z < 1.96}$
-0.642	0.005	0.321	1.505
(0.428)	(0.007)	(0.148)	(0.799)

Notes. Table shows results from Andrews-Kasy (2019) method with standard errors in brackets. θ is the estimate of the "true" average effect size accounting for the estimated publication bias. The right three columns give the estimated publication probability relative to a positive estimate that is significant at the 95% confidence level. Z is the Z score (estimate divided by standard error). The publication probabilities are estimated for Z scores between the shown cut-offs.

Table V – Descriptive statistics of MRA covariates

Name	Mean	Standard Deviation
Control_gender	0.19	0.40
Control_race	0.20	0.40
Control_income	0.33	0.47
Control_education	0.20	0.40
Homicide	0.18	0.38
Violent	0.62	0.49
Non_Violent	0.15	0.36
Both	0.22	0.42
Area	0.76	0.43
OLS	0.40	0.49
ML	0.14	0.35
Odds_Ratio	0.03	0.17
Panel	0.67	0.47
Addressing Endogeneity	0.40	0.49
North America	0.71	0.46
Europe	0.16	0.37
Australasia	0.08	0.27
Direct Lead Measure	0.10	0.30
Number of Covariates*	0.00	1.00
Sample Size*	0.00	1.00

Notes. See section 4.3 for description of variables.

* indicates variables have been standardised.

Table VI – Variables used in combinations for each sample estimation

Variables used	**Estimation**
Control gender, Control race, Control income, Control education, Homicide, Violent, Non-Violent, Area dummy, OLS, ML, Odds Ratio, Panel dummy, Addressing Endogeneity, North America, Europe, Australasia, Direct Lead Measure, Covariates, Sample Size	Full Sample
Control gender, Control race, Control income, Homicide, Violent, Non-Violent, Area dummy, OLS, ML, Panel dummy, North America, Direct Lead Measure, Covariates, Sample Size	Addressing Endogeneity Sample
Control gender, Control race, Control income, Control education, Homicide, Violent, Non-Violent, Area dummy, OLS, ML, Odds Ratio, Panel dummy, North America, Europe, Australasia, Direct Lead Measure, Covariates, Sample Size	Correlational Sample
Control race, Control income, Control education, Homicide, Violent, Non-Violent, OLS, ML, Panel dummy, Addressing Endogeneity, North America, Europe, Australasia, Direct Lead Measure, Covariates, Sample Size	Area Sample
Control gender, Control race, Control income, Control education, Homicide, Violent, Non-Violent, OLS, ML, Odds Ratio, Panel dummy, Addressing Endogeneity, North America, Europe, Australasia, Direct Lead Measure, Covariates, Sample Size	Individual Sample
Control race, Control income, Control education, Area dummy, OLS, ML, Panel dummy, Addressing Endogeneity, North America, Europe, Australasia, Covariates, Sample Size	Homicide Sample
Control gender, Control race, Control income, Control education, Area dummy, OLS, ML, Odds Ratio, Panel dummy, Addressing Endogeneity, North America, Europe, Australasia, Direct Lead Measure, Covariates, Sample Size	Violent Crime Sample

Notes. Table shows which covariates were included for each sub-sample estimation. Inclusion depended on whether there was variation in the covariate for that subsample.

Table VII – Estimates of the coefficients on covariates included in meta-regression analysis

Variable	Mean	Median	Standard Deviation
FAT	1.22	1.16	1.41
Precision	0.20	0.17	0.15
Control_gender	0.01	0.01	0.03
Control_race	-0.05	-0.05	0.03
Control_income	0.01	0.00	0.03
Control_education	-0.01	-0.01	0.01
Homicide	-0.05	-0.05	0.01
Violent	0.01	0.01	0.03
Non_Violent	-0.01	-0.01	0.03
Area_dummy	0.20	0.17	0.12
OLS	-0.09	-0.08	0.08
ML	0.00	0.02	0.08
Odds_Ratio	-0.22	-0.22	0.11
Panel_dummy	-0.12	-0.09	0.16
Addressing Endogeneity	-0.02	-0.02	0.02
North_America	-0.28	-0.29	0.11
Europe	0.04	0.02	0.18
Australasia	0.09	0.10	0.09
Direct_Lead_Measure	-0.03	-0.02	0.12
*Covariates	-0.10	-0.11	0.04
*Sample Size	0.00	0.00	0.00

Table VIII – Meta-analysis average estimates for the full sample and each subsample

Sample averages

Sample	Mean	Median	SD	N	$\% < -1 \; or \; > 1$
Full Sample	0.11	0.12	0.06	524288	0%
Addressing Endogeneity Sample	0.01	0.01	0.01	16384	0%
Correlational Sample	0.28	0.27	0.10	262144	0%
Area Sample	0.26	0.27	0.07	65536	0%
Individual Sample	0.02	0.02	0.03	262144	0%
Homicide Sample	0.72	0.74	0.21	8192	5%
Violent Crime Sample	0.29	0.23	0.19	65536	0%
Non-violent Crime Sample	0.76	0.74	0.24	65536	15%

"Ideal" specification

Sample	Mean	Median	SD	N	$\% < -1 \; or \; > 1$
Full Sample	-0.05	-0.02	0.16	524288	0%
Addressing Endogeneity Sample	-96.52	0.02	299.66	16384	13%
Correlational Sample	0.19	0.19	0.33	262144	1%
Area Sample	0.39	0.33	0.37	65536	6%
Individual Sample	0.04	0.02	0.15	262144	0%
Homicide Sample	7.70	2.15	11.41	8192	66%
Violent Crime Sample	0.13	0.01	0.72	65536	19%
Non-violent Crime Sample	0.63	0.43	2.37	65536	57%

Notes. Table shows results from combining multiple meta-regression estimates, each using different specifications. All regressions carried out by restricted maximum likelihood. This is done for the full sample and subsamples. N is the number of regressions carried out, each a different specification. The mean and median are the summary statistics of the average effect size from these regressions, given in Partial Correlation Coefficients (PCCs). PCCs are bounded between -1 and 1. The last column gives the percent of PETs which fall outside this range.

Table IX – Descriptive statistics of data used for elasticity estimation

Variable	Mean	Standard Deviation
Median blood lead level for children ages 1-5 in US	3.39	4.42
US Homicide rate	6.98	1.81

Sources. NHANES data for blood lead and FBI uniform crime reports for the homicide data.

Appendix

The Lead-Crime Hypothesis: A Meta-Analysis

A. Converting to common estimates

To conduct a meta-analysis all estimates must be converted to a common metric. We calculate the partial correlation coefficient (PCC) as shown in equation (I):

$$\text{(I)} \qquad PCC_{ij} = \frac{t_{ij}}{\sqrt{t_{ij+df_{ij}}^2}}$$

Where t_{ij} is the t-ratio for estimate *i* of study *j*, and df_{ij} is the degrees of freedom. The standard error of each PCC is calculated according to equation (II):

$$\text{(II)} \qquad SE_{ij} = \frac{PCC_{ij}}{t_{ij}}$$

Some papers reported odds ratios rather than correlation coefficients. Following Polanin and Snilstveit (2016), we converted these to PCCs.

$$\text{(III)} \qquad PCC_{ij} = \frac{ln(OR_{ij}) \times \left(\frac{\sqrt{3}}{\pi}\right)}{\sqrt{\left(\ln(OR_{ij}) \times \left(\frac{\sqrt{3}}{\pi}\right)\right)^2 + a_{ij}}}$$

Where OR_{ij} is the odds ratio *i* for study *j* and $a_{ij} = \frac{\left(n_{ij1}+n_{ij2}\right)^2}{n_{ij1}n_{ij2}}$. Here a_{ij} is a correction factor which depends on the sample size in the control and treatment groups (n_{ij1} and n_{ij2}). If the sample sizes are unknown, or there are no treatment and control groups, we follow Borenstein *et al*. (2009) and set them to be equal, which gives $a = 4$.

In a similar way we calculate standard error equivalents for odds ratio estimates. Following the Cochrane Handbook (Higgins and Green, 2011), first I convert the 95% confidence intervals to odds ratio standard errors (ORSE).

(IV) $$ORSE_{ij} = \frac{(\ln(\overline{CI}) - \ln(\underline{CI}))}{3.92}$$

Where $\overline{CI}$ is the upper confidence interval limit and $\underline{CI}$ is the lower confidence interval limit. I then convert this into partial correlation coefficient standard errors.

(V) $$SE_{ij} = \sqrt{\frac{(a^2 \times ORSE_{ij}^2 \times \left(\frac{3}{\pi^2}\right)}{\left(\left(\log(OR_{ij}) \times \left(\frac{\sqrt{3}}{\pi}\right)\right)^2 + a\right)^3}}$$

Only one study (Billings and Schnepel, 2018) has estimates which are similar to randomised control trial estimates, with a mean difference shown between control and treatment groups. These can also be converted to PCCs. For these we follow Borenstein *et al*. (2009) and first compute the within-groups standard deviation SD_{ij} for estimate *i* of study *j*, as shown in (VI).

(VI) $$SD_{ij} = \sqrt{\frac{(n_{ij1}-1) \times S_{ij1}^2 + (n_{ij2}-1) \times S_{ij2}^2}{n_{ij1} + n_{ij2} - 2}}$$

Here, n_{ij1} is the sample size for the control group for *i* of study *j*, S_{ij1} is the standard deviation for the control group, while n_{ij2} and S_{ij2} are the same from the treatment group.

We use this to calculate Cohen's D:

(VII) $$D_{ij} = \frac{\bar{X}_{ij1} - \bar{X}_{ij2}}{SD_{ij}}$$

Where $\bar{X}_{ij1}$ is the sample mean for the control group and $\bar{X}_{ij2}$ for the treatment group. Finally, we convert Cohen's D to a PCC by equation (VIII).

(VIII) $PCC_{ij} = \frac{D_{ij}}{\sqrt{D_{ij}^2 + a_{ij}}}$

Here a_{ij} is the same as that for equation (III) except we have the sample sizes for each group so we do not set it to equal 4. The variance for Cohen's D is calculated as in (IX).

(IX) $DVar_{ij} = \frac{n_{ij1}+n_{ij2}}{n_{ij1}\times n_{ij2}} + \frac{D_{ij}^2}{2(n_{ij1}+n_{ij2})}$

This is then used to calculate the standard error of the PCC.

(X) $SE_{ij} = \sqrt{\frac{a_{ij}^2 \times DVar_{ij}}{\left(D_{ij}^2 + a_{ij}\right)^3}}$

One further study only uses simple correlations (Lauritsen *et al*., 2016). The standard errors for these must be approximated. We use the approximation of one divided by n-3 for the correlation standard errors, as n is the same for all estimates, the standard errors are the same for all these estimates.

B. Common effects and random effects meta-analysis

This section explains how common and random efffects meta-analysis estimates are calculated.

Before calculating fixed or random effects meta-averages, first we convert all PCCs to normalised estimates with equation (XI), so that correct confidence intervals can be calculated.

(XI) $Z_{ij} = 0.5\ ln\left(\frac{1+PCC_{ij}}{1-PCC_{ij}}\right)$

Where Z_{ij} is the normalised effect size of a PCC. The process is that first PCCs are converted to normalised estimates, we estimate using either common effects or random effects, then the estimates are converted back to a PCC with equation (XII).

(XII) $PCC = \frac{e^{2z}-1}{e^{2z}+1}$

Where in this case the PCC is the meta-analysis estimate as a correlation coefficient, and Z is the estimate obtained from the normalised PCCs.

To calculate the common effects averages we weight each estimate by the inverse of the variance, and then divide the sum of these weighted estimates by the sum of the weights as shown in following two equations:

(XIII) $W_{ij} = \frac{1}{V_{ij}}$

(XIV) $FE = \frac{\sum_{i=1}^{N} W_{ij} Z_{ij}}{\sum_{i=1}^{N} W_{ij}}$

Where V_{ij} is the variance of estimate i of study j , FE is the fixed effects average, and Z_{ij} is normalised PCC. This average is converted back into a PCC by equation (XII). Along with the averages I calculate 95% confidence intervals, first by obtaining the standard errors of FE.

(XV) $SE_{FE} = \sqrt{\frac{1}{\sum_{i=1}^{k} w_{ij}}}$

Then obtaining lower and upper limits in the normal fashion. The fixed effect averages and standard error can be used to calculate Z-scores for hypothesis testing as normal.

Random effects meta-averages are estimated in the same way as fixed effects, except we replace V_{ij} in equation (XIII) with V_{ij}^{*}. Where $V_{ij}^{*} = V_{ij} + T^2$, and T^2 is an estimate of the between-study variation. There are different methods of estimating T^2, we use the DerSimonian-Laird (1986) method.

C. Review of literature used in meta-analysis

There are 24 total studies included in this meta-analysis. The studies use different methods to examine the lead-crime relationship. Longitudinal studies, which track the same people over time, are common. Fergusson, Boden and Horwood (2008) use a longitudinal sample and find a positive association between dentine lead levels at 6-9 years of age and later offending while including race and family socioeconomic status covariates. However, the effect was smaller once variation in education grades was added. They reasoned that the effect of lead was in reducing education outcomes, leading to more crime. Overall, they find that lead only explains 1% of the variation in crime. Nkomo *et al*. (2017) used a longitudinal sample in South Africa and found a positive association between blood lead levels at age 13 and violent crime in later life. Beckley *et al*. (2018) find only a small positive effect of childhood lead levels and both violent and non-violent crime in their longitudinal sample of New Zealand residents. They conclude other factors are much more important for determining crime rates. Finally, Sampson and Winter (2018) follow a longitudinal sample in Chicago and find school age lead levels are not associated with an increase in arrests in later life. Overall, longitudinal studies show a mixed picture, both on whether there is an effect and whether it is a strong one.

A different strand of research looks at the correlation of lead levels and crime across time and areas, rather than at an individual level. Three studies look at time series of lagged lead levels and crime for the US. Nevin (2000) finds a positive effect, but McCall and Land (2004) find no effect on the age cohorts most affected in youth by the increase in leaded gasoline. They reason that increased lead levels at one time should only affect the crime rates of that cohort, not earlier cohorts, and so only look at crime rates for those certain age ranges. Lauritsen, Rezey, and Heimer (2016) look at two different data series of

crime: the National Crime Victimization Survey (NCVS) and the Uniform Crime Reports (UCR). They find that lead is positively correlated with violent crime in the UCR but not the NCVS, which they consider a better measure of violent crime. However, they consider both data sources equally valid for property crime. Stretesky and Lynch (2004) find a strong effect when looking across US countries for both property and violent crime using the UCR. Mielke and Zahran (2012) find a strong effect across six US cities, Lersch and Hart (2014) find the same looking at Florida census tracts. Both Barrett (2017) and Manduca and Sampson (2019) find a strong positive relationship in census tracts in Chicago using different methods. Looking outside the US, Taylor *et al*. (2018) find positive results for violent crime in Australia, and across six suburbs in New South Wales. Nevin (2007) estimates the relationship for many OECD countries and finds pre-school blood levels are strongly associated with a whole range of violent and non-violent crime. On the whole, studies which look at geographic areas as the unit of interest tend to find the strongest positive associations between lead and crime.

The final strand of the literature are those studies that attempt to identify a casual effect while accounting for endogeneity from unobserved variables correlated with both crime and lead. These could bias the estimate of the effect of lead on crime. Lead exposure is correlated with poverty (Baghurst *e al*. 1999) and race (Sampson and Winter, 2016) and likely with other, unobservable, variables. We cannot rule out that these variables may cause individuals to commit more crime and be more exposed to lead, rather than lead being the cause. Even panel data designs with controls may not account for this endogeneity. The endogeneity threat has led to some, more recent, studies using quasi-experimental methods. Needleman (2002) carried out a "case control" study where young offenders were matched to a "control" group chosen for similar observable characteristics. The offender group was found to have higher bone lead levels. Although

this this is an improvement beyond looking at correlation alone, the likelihood of unobservable group differences means that the problem of endogeneity was not adequately resolved.

Reyes (2007) is the first study to use quasi-experimental methods to derive a causal estimate. She uses the different grades and concentration of lead in gasoline in US states as an instrumental variable for lead levels. She finds an effect of lead on violent crime but not property crime. In a later paper (2015) she uses a similar identification strategy with individual-level data. Here she finds a positive effect on both property and violent crime. Feigenbaum and Muller (2016) also use an instrumental variable strategy. They instrument for the presence of lead water pipes in US cities using the distance to the nearest lead refinery in 1899, a period in which thousands of US cities built their water supplies. They find a positive causal effect on homicides in 1921-1936. Aizer and Currie (2018) use nearby traffic volume interacted with year of birth as an instrument for lead and include sibling fixed effects. They find a positive relationship between lead and incarceration. Curci and Masera (2018) also find a positive association when they look across 300 US cities. Most of the estimates from this paper do not fall under the "addressing endogeneity" category, but in one chart of estimates they use soil quality as an instrument for lead. Grönqvist *et al*. (2019) use a sample of 800,000 Swedish children grouped by neighbourhoods and cohorts. They instrument for blood lead levels by the lead measured in moss in the areas. The estimates are mixed but tend to show a small positive effect on crime. Finally, Billings and Schnepel (2018) match a treatment group of children who had blood lead levels above a 10μg/dL threshold in two tests, with a control group of children who were above the threshold in the first test and just below in the second test, thus failing to qualify for treatment. This, close to randomised control trial, study finds a positive effect of lead on crime, with a stronger effect on property crime

than violent crime. Overall, the few studies that use quasi-experimental methods all find a positive effect on crime, but they tend to find a smaller effect than the studies that look at correlations across geographic areas.

D. Publication Bias Using Only Representative Estimates

In section 4.3 we test for publication bias using all estimates. Here we repeat the exercise using only the representative estimates. However, we cannot estimate the hierarchical model, or cluster errors as we only have one estimate per study.

Table D.1 – Bias and average effect estimates using representative estimates

Variable	FAT-PET	FAT-PEESE
$\hat{\beta}_F$	3.714	12.049
	(0.892)	(9.070)
$\hat{\theta}$	-0.001	0.006
	(0.005)	(0.006)

Notes. Estimates presented with their standard errors in brackets. FAT-PET is Funnel Asymmetry test and Precision Effect Test. FAT-PEESE is Funnel Asymmetry Test and Precision Effect Estimate with Standard Error. The multi-level FAT-PET is a mixed effects-multi-level model with a different slope coefficient for each study. $\hat{\beta}_F$ is the estimate of bias in a meta-analysis sample. θ is the estimate of the "true" average effect size accounting for the estimated bias $\hat{\beta}_F$.

E. Bayesian Model Averaging

As a robustness check we carry out Bayesian model averaging with all variables used in our meta-regression analysis. We estimate a normal-gamma conjugate model with a uniform model prior and unit information g-prior. These are the same as in Bajzik *et al*. (2019), see there for more information. The results are given below.

Table E.1 – Posterior results from Bayesian model averaging

Variable	Posterior Mean	Posterior Standard Deviation	Posterior Inclusion Probability
Precision	0.33	0.03	1.00
Homicide	-0.01	0.02	0.15
Violent	0.00	0.01	0.08
Non_Violent	0.00	0.01	0.11
Odds_Ratio	-0.12	0.09	0.75
ML	0.12	0.02	1.00
Panel dummy	-0.16	0.03	1.00
Addressing Endogeneity	0.00	0.00	0.05
Area dummy	0.18	0.02	1.00
Covariates	-0.07	0.01	1.00
Sample Size	0.00	0.00	0.10
North_America	-0.43	0.03	1.00
Europe	0.01	0.03	0.11
Australasia	-0.01	0.03	0.11
Control_gender	0.00	0.01	0.12
Control_race	-0.04	0.02	0.89
Control_income	0.00	0.01	0.09
Control_education	0.00	0.00	0.07
OLS	0.10	0.02	1.00
Direct Lead Measure	-0.39	0.04	1.00
FAT	3.88	NA	1.00

We evaluate the posterior means at the sample averages for each variable (excluding the FAT as normal). This gives a point estimate PCC of 0.06.

F. Appendix References

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