The Long-Term Impact of Antidepressant Pharmacotherapy in India*

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Abstract

Treating depression through pharmacotherapy is a widespread practice that may be cost-effective in LMICs, where the scarcity of mental health specialists limits access to care. However, the long-term effects of a brief course of antidepressants are unknown. This paper evaluates the seven-year impact of a 8-month course of pharmacotherapy that was offered to low-income, depressed adults in Karnataka, India, in 2017. The intervention accelerated remission by up to 27 months and did not increase recurrence. However, we find no mental health or economic benefits of the intervention after seven years, perhaps due to the natural recovery of many people in the control group. Offering pharmacotherapy increased people's awareness of effective depression treatment, the perceived costs of depression, and their likelihood of seeking treatment when depressed.

Keywords: Depression, Health, Poverty

JEL Codes: I15, I18

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1 Introduction

With a lifetime prevalence of 15-20 percent, depression is the leading cause of disability worldwide. Symptoms of depression include anhedonia (the inability to feel pleasure), impaired attention, and fatigue, all of which may affect productivity, investment, and decision making. Depression is more common among low-income populations and can contribute to poverty both immediately (e.g., through higher productivity) and over time (e.g., through higher investment or permanently better mental health) (Ridley et al., 2020; Kessler and Bromet, 2013).

Despite the high prevalence of depression in low- and middle-income countries (LMICs) and the availability of effective treatment, most depressed people do not receive care (Mekonen et al., 2021). Three barriers contribute to this gap. The scarcity of mental health care providers in LMICs limits the supply of depression treatment (Saxena et al., 2007). Moreover, pervasive stigma and low awareness of depression symptoms and treatment options limit patient demand for care (Heim et al., 2020).

Antidepressants such as selective serotonin re-uptake inhibitors (SSRI) provide effective short-term depression treatment (Gartlehner et al., 2017; Cipriani et al., 2018).[1] They are also a potentially important tool in LMICs because this approach requires less time from highly trained personnel, which is scarce. Off-patent SSRI are inexpensive.[2] Lastly, pharmacotherapy involves minimal contact with providers, thus protecting patient privacy, which may be especially important due to pervasive mental health stigma.

However, the long-term impact of a single course of pharmacotherapy on mental health and related outcomes remains unclear both in clinical and community settings, since most studies follow subjects for only 6 to 12 months after treatment (Cipriani et al., 2018; Kato et al., 2021). Thus, understanding the long-term impacts is important clinically, for policy purposes, and to understand the links between depression and poverty.

[1] Gartlehner et al. (2017) show that SSRI reduce depression severity by 0.35 standard deviations (SD), which is comparable to the 0.22 SD impact of cognitive behavioral therapy.

[2] For example, the minimum cost of a 30-day course of Sertraline, Citalopram, and Escitalopram, three commonly prescribed SSRI, is USD 0.60-3.5 in India (Patel, Patel and Patel, 2015).

The long-term effect is *ex ante* uncertain because a single course of pharmacotherapy can have multiple biological and behavioral impacts. The initial reduction in depression rates due to antidepressant use can diminish over time as spontaneous recovery occurs in the control group. Moreover, short courses of pharmacotherapy, such as the one in this study, have been thought to increase depression recurrence after treatment concludes (Baldessarini, 2013). At the same time, SSRI may induce neurochemical and physiological changes that could reduce the onset and severity of future depressive episodes (Connor et al., 2000; Murlanova et al., 2021).[3] Beyond biological effects, a prior experience with depression treatment may influence the behavioral response to future depressive episodes by enhancing patients' recognition of symptoms and knowledge of treatment options. This experience may also reduce depression stigma by teaching patients and their families that depression is a common disorder treatable with medication (Henderson et al., 2014). Thus, even without long-term biological changes, behavioral mechanisms may change the duration and severity of future depressive episodes through care seeking.

This paper investigates the long-term effects of pharmacotherapy on depression and possible behavioral responses. In 2017, we offered an eight-month course of pharmacotherapy through SSRI (Psychiatric Care, or PC), cross-randomized with a light-touch income-generating intervention (Livelihoods Assistance, or LA), to 1,000 adults experiencing mild to moderately-severe depression in Karnataka, India (Angelucci and Bennett, 2024). This treatment significantly reduced depression severity in the short term, with effects that were comparable to those reported in meta-analyses of pharmacotherapy. Impacts were more pronounced and longer-lasting when PC was paired with LA. We resurveyed the original participants eight years after initial enrollment and seven years after the PC intervention concluded. Since the LA intervention had no independent effects, the focus of this study is on the two pharmacotherapy arms, PC/LA and PC.

We have several key findings. First, we do not find evidence that a single course of

[3]For instance, antidepressants have been shown to reduce neuroticism (Tang et al., 2009; Soskin et al., 2012), enhance neuroplasticity (Page et al., 2024), and may alter sensitivity to negative outcomes through changes in serotonin re-uptake (Colwell et al., 2024).

antidepressants improved depression symptoms or prevalence seven years later. While taking antidepressants accelerated depression remission, this difference disappeared by month 27, at which point depression rates in the control and treatment groups were comparable. By the eighth year, depression prevalence was around 20% for all arms. Recurrence rates of depression were not higher in the treatment arms despite adopting a shorter course of pharmacotherapy than the ones often used in high-income countries.

Secondly, there was a positive effect of receiving pharmacotherapy on awareness of treatment efficacy and how to seek it: participants offered antidepressants by a psychiatrist were 0.16 SD more likely to understand that psychiatrists can prescribe these medications and that the medications are effective. In contrast, awareness of knowledge and practices not directly experienced in the intervention (such as the efficacy of psychotherapy) did not change. Similarly, there was no long-term improvement in knowledge of key depression symptoms, likely because there was a high knowledge base in the control group.

Thirdly, we observed improvements in beliefs about depression, driven by more optimistic views about the labor and marriage market costs of depression, and about the costs of having depressed household members. These improvements occur especially in the PC arm, with effect sizes of up to 0.2 SD. Changes in awareness and beliefs could affect care seeking during subsequent spells of depression. Consistent with this conjecture, we find that treatment participants were more likely to seek treatment when experiencing depression. For example, the likelihood of seeking treatment at least half the time when depressed increases from 13% to 22% in the pooled arms, and to 32% in the PC arm.

Finally, we find no economically or statistically significant long-term improvements in work hours, earnings, household consumption, durable goods ownership, hygiene, children's human capital investment, and incidence of socioeconomic shocks, as well as cognitive performance, risk intolerance, subjective well-being, and household decision-making. As we explain below, these findings do not rule out an effect of depression on long-term poverty.

We consider the scope for scaling up this intervention and the lessons learned from our study. Since the provision of pharmacotherapy is beneficial in the medium term, it should

be scaled up if it is cost effective. Scaling up in our study setting is to some extent feasible in the short term since some affordable mental health care infrastructure already exists and the population has a good understanding of core depression symptoms. Yet, a challenge to scale is the low demand for mental health care, partly likely due to high depression stigma and limited knowledge of effective treatment and where to seek it. Our findings show that inducing people to seek and receive treatment will gradually improve their perception of depression stigma and their knowledge of what treatment works and how to seek it. However, while prior experience with the health system increased care-seeking, it was insufficient to close the gap: for example, only 22% of the treatment group sought free care at least half the time after experiencing a spell of depression. Thus, addressing these challenges requires more than just expanding access to medication. Regular depression screening and guided support through treatment appear crucial, as most individuals do not seek care independently. Concurrently, broad-scale efforts may be needed to tackle mental health stigma at a societal level.

2 Setting and Intervention

In 2016, we conducted a study in 506 localities in Karnataka. The psychiatric care (PC) intervention provided a diagnosis and eight months of free psychiatric care at a local hospital. The most prescribed anti-depressants were SSRI. These drugs are available inexpensively in India, are widely used, have relatively few well-tolerated side effects (Ferguson, 2001; Cascade, Kalali and Kennedy, 2009), and are as effective as psychotherapy in treating depression (Gartlehner et al., 2017). Uptake was 46 and 43 percent in the PC and PC/LA arms, 91 percent of compliers were diagnosed with depression, and the median course of SSRI lasted 4 months (Angelucci and Bennett, 2024).[4]

We used a stratified and cluster-randomized design to cross-randomize PC and LA by locality, using district and terciles of a predetermined locality socioeconomic index as strata.

[4]The livelihoods assistance (LA) intervention provided two group meetings and personalized assistance during the first two months of the study. Overall, this intervention had no independent effects. Thus, it is not the focus of our study of long-run impacts.

The modal and median number of participants per locality is 2. This design minimizes spillovers and cross-arm contamination. Treating few people per locality limited information leakages, protecting patient confidentiality. This is important in contexts with high levels of mental health stigma.

We screened people for depression symptoms with the PHQ-9 scale (Kroenke, Spitzer and Williams, 2001*a*). It ranges from 0 to 27. Higher values indicate more severe symptoms. The PHQ-9 is widely validated to screen for depression and measure the response to treatment in India and throughout the world (Patel et al., 2008; Manea, Gilbody and McMillan, 2012; Indu et al., 2018). To obtain a sample of at least moderately depressed people, we recruited subjects with PHQ-9 scores of 9-20.

3 Data, Outcomes, and Hypotheses

In 2024, we surveyed 916 of the 1000 participants in the 2016 study. We reached 909 respondents in person and contacted seven over the phone, for a 8.4% attrition rate.[5] Attrition is generally balanced by arms. It is 11% in the control group and 10% in the pooled PC arms (9 and 11% in the PC/LA and PC arms).[6]

Our primary goal is to investigate the long-term effects of treatment with a single course of antidepressants. We measure depression severity using the standardized PHQ-9 score. We also consider an indicator for PHQ-9 scores of at least 10, which indicates symptoms of at least moderate depression. In addition, we asked participants to recall spells of depression since 2018. In this way, we have up to 10 individual observations of concurrent or recalled depression over 90 months since the beginning of the intervention. While recall bias in health research is well-documented (Althubaiti, 2016), differences in recalled rates of depression by arm can still be informative if the bias does not vary by arm. Since we observe both

[5]68 of the missing 84 participants had deceased, 8 could not be located, two had moved and we were unable to reach them, 2 provided incomplete information, and only 4 participants refused to respond.

[6]The attrition rate in the LA arm is lower, at 5%. This may be due to the lower participants' mortality rate of 4 percent (vs statistically indistinguishable 7, 6, and 8% rates in the control, PC/LA, and PC arms). Weighing the data to account for this small imbalance does not change our findings, since the PC arms and the control arm are balanced.

concurrent and recalled depression for 2018 and 2019, we can estimate the magnitude of recall bias and how it varies over time. We perform this exercise in Appendix A.2 and conclude that recall bias is likely small from 2020 on (42 to 78 months since baseline). Thus, we can track the annual prevalence of depression until the eighth year since the start of the intervention using a combination of concurrent and recalled data.

The baseline PHQ-9 is 14.3 in the control group and 13.8 in the pooled PC arms (13.5 and 14 in the PC/LA and PC arms). The difference between the PC/LA and the control arms is small but statistically significant ($p < 0.05$). The baseline share of people with $PHQ - 9 \geq 10$ is 0.89 in the control group and 0.87 in the pooled PC arms (0.86 and 0.87 in the PC/LA and PC arms). The difference between the pooled and individual treatment arms and the control arm is never statistically significant ($p > 0.1$).

Clinical studies typically examine the short-term effects of a single course of antidepressants, or the withdrawal symptoms of multi-year, long-term use (Gartlehner et al., 2017; Fava et al., 2015). Thus, to formulate a hypothesis about the long-term impacts of offering a single course of antidepressants, we consider the existing evidence of the long-term effects of psychotherapy, the effects of pharmacotherapy on recurrence, and our understanding of local behavioral barriers to care seeking. Those results suggest that impacts on depression eventually dissipate as many control participants also experience remission (Baranov et al., 2020; Bhat et al., 2022). In addition, pharmacotherapy could have long-term effects by altering the frequency or severity of future depression spells or by changing the propensity to seek care for future depression spells. Lastly, many people may relapse after short courses of antidepressants, undoing some of the initial benefits of medication (Kato et al., 2021). Thus, we also consider whether recurrence and remission vary by arm. We use the PHQ-9 data (measured in 2016, 2017, 2018, 2019, and 2024) and the recalled depression episodes (for the years 2020 to 2023) to measure depression remission, $P(PHQ - 9 < 5, t | PHQ - 9 \geq 10, t-1) = 1$, and recurrence, $P(PHQ - 9 \geq 10, t | PHQ - 9 < 5, t-1) = 1$.

We also consider all outcomes studied in Angelucci and Bennett (2024), which include individual- and household-level socioeconomic outcomes and potential pathways that may

link them to depression. Since, with few exceptions, there was no short- or medium-term improvement in these outcomes, we expect no long-term improvements either. These are: participants' work time and earnings, household consumption and assets, hygiene/sanitation, and investment. For work time and earnings, we measure self-reported work time (the time spent on productive activities) in the previous 7 days. Productive activities include primary and secondary paid and unpaid jobs, agricultural work, and childcare, cooking, cleaning, doing laundry, and fetching water. We measure weekly earnings from primary and secondary paid jobs. For household consumption and assets, we measure per-capita non-durable consumption as the sum of household food consumption in the past week (across 23 food groups that are common locally) and expenditures on 13 non-durable non-food commodities (converted into weekly values from 1 or 2 month recalls) divided by household size.[7] We measure durable goods ownership according to indicators for household ownership of nine goods.[8] We measure hygiene and sanitation by observing whether there is open defecation or visible garbage at the respondent's home, whether the cooking area is clean, and whether the respondent has visibly dirty hands and fingernails. We measure investment with: a children's human capital investment from school enrollment, school attendance, homework time, and child labor; and an index of shocks experienced in the household in the previous 6 months, measured scoring the severity of shocks (Holmes and Rahe, 1967).

Possible pathways through which depression treatment may affect socioeconomic outcomes are: risk intolerance, cognitive performance, subjective wellbeing, and participation in household decisions. We elicit risk intolerance through items from the Blais and Weber (2006) DOSPERT scale, a generalized risk self-assessment (Dohmen et al. (2011)), and the Eckel and Grossman (2008) incentivized lottery game. We assess cognitive performance through three incentivized tests: Raven's Progressive Matrices, which estimates fluid intelligence, and forward and backward digit spans, which measure verbal short-term and working

[7]We include foods that were purchased, produced at home, or received from others. To compute the value of non-purchased food, we multiply the quantity consumed by median unit values. For wages and consumption, we winsorize the top 2.5%, following Tukey et al. (1977).

[8]These goods are a chair, a bed, a table, an electric fan, a television, a refrigerator, a bicycle, a motorcycle or scooter, and a car.

memory. We use the five-item Satisfaction with Life Scale to measure subjective wellbeing (Kobau et al. (2010)). As a measure of participation in household decisions, respondents indicate whether they make household financial and employment decisions alone, with other household members, or not at all.

Our data have few or no missing values. Thus, we include all available observations for a given outcome.

4 Identification and Estimation

We estimate "intent-to-treat" effects by including all respondents within an intervention arm regardless of compliance. In our previous analysis, a Young (2019) omnibus test failed to reject the hypothesis of no joint effect of LA across the study outcomes. Based on this finding, we pool the PC and PC/LA arms to increase precision and estimate the parameters of the following equation for respondent i in locality j:

$$Y_{ij} = \rho_0 + \rho_1[AnyPC_j] + \rho_2[LA_j] + \rho_3 X_{ij} + \mu_{ij} \tag{1}$$

The variable $AnyPC$ is an indicator for the pooled PC and PC/LA arms, while LA is an indicator for the LA-only arm. X_{ij} is a vector of predetermined covariates. Our primary empirical approach uses the Belloni, Chernozhukov and Hansen (2014) post double-selection LASSO method to select covariate controls.[9] We also show the ANCOVA estimates for depression severity since we observe this outcome at baseline.

The parameter ρ_1 identifies the Average Intent to Treat (AIT) effects of each intervention arm under the assumptions that potential outcomes of each treated person are unaffected

[9]The method selects from these baseline covariates: round and strata indicators, participant's gender, marital status, education, scheduled caste/tribe, literacy, household size, PHQ-9 index components, GAD-7 (anxiety) index components, activities of daily living index components, time use (all work, paid work, unpaid work, sleep, leisure, and job search hours), per capita household non-durable consumption and expenditures (food, non-food, clothes for children, medical), sanitation/hygiene index components, human capital index components, per capita net savings components, durable goods index components, risk intolerance index components, negative shock index components, cognition index components, subjective wellbeing index components, participation in household decision index components. To avoid dropping observations, we include indicators for missing values of all covariates and then set missing values to zero.

by the treatment status of other people and that treatment assignment is independent of potential outcomes. Assigning treatment by locality minimizes instances of violations of the first assumption through spillovers such as social interactions, while treating a median of 2 people per locality minimizes locality-specific equilibrium effects. Random assignment ensures that the second assumption holds. We estimate this equation by OLS and cluster standard errors by locality throughout the analysis.

In addition, we estimate the AIT effects of each pharmacotherapy arm:

$$Y_{ij} = \beta_0 + \beta_1[PC/LA_j] + \beta_2[PC_j] + \beta_3[LA_j] + \beta_4 X_{ij} + \epsilon_{ij} \tag{2}$$

The variables PC, and PC/LA are indicators for the arms that receive PC only, LA only, or both PC and LA. The other variables are defined similarly to Equation 1. The parameters β_1 and β_3 identify the AIT effects of PC and PC/LA under the previous assumptions.

Creating indices as described in Section 3 corrects for multiple hypothesis testing within families of outcomes. This approach is also consistent with how we dealt with multiple hypothesis testing in our previous analysis.

5 Effects on Depression

5.1 Symptoms and Prevalence

Table 1: Impact on Depression Symptoms (PHQ-9)

	PHQ-9 (STD)	
	(1)	(2)
Panel A: Pooled PC		
Any PC	0.042	0.046
	(0.079)	(0.079)
Panel B: PC/LA and PC		
PC/LA	0.014	0.012
	(0.091)	(0.091)
PC	0.067	0.072
	(0.105)	(0.106)
PC/LA = PC	0.652	0.613
N	909	909
Specification	ANCOVA	LASSO

Notes: The PHQ-9 is standardized. The standardized control group mean is 0. The non-standardized control group mean is 5.75. Standard errors clustered by locality in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$. The ANCOVA estimator conditions on the baseline value of the dependent variable and strata fixed effects. The LASSO estimator of Belloni, Chernozhukov and Hansen (2014) chooses covariates from a list described in footnote 9.

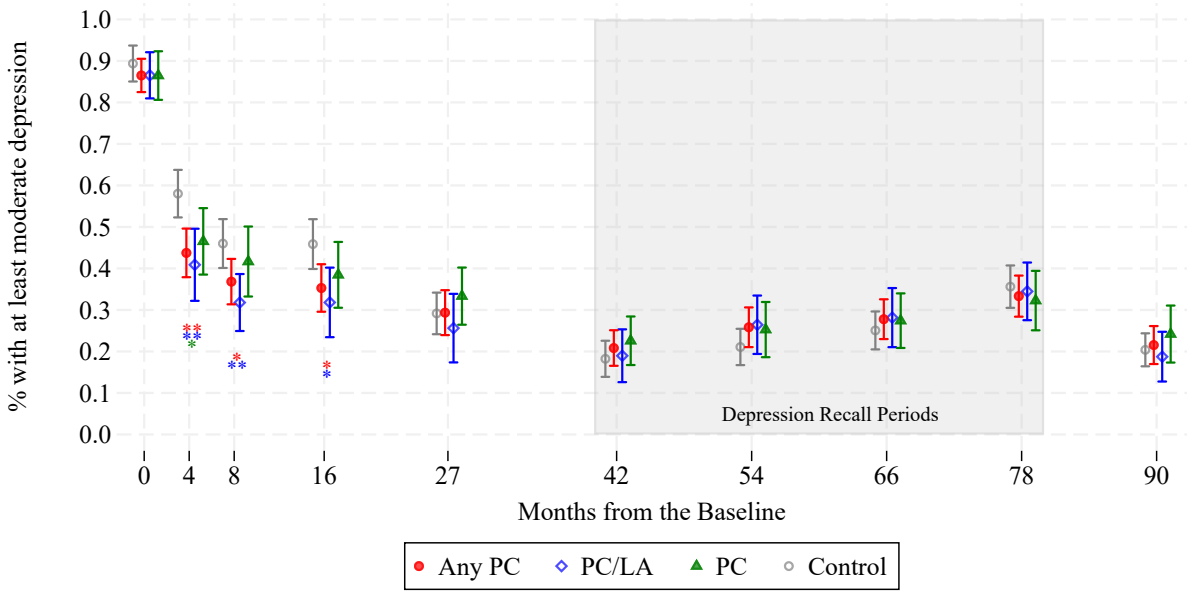
Table 1 shows the AIT effects on the PHQ-9 scale, which measures the severity of depression symptoms. Higher values indicate more severe symptoms. Results suggest that the pharmacotherapy (either alone or combined with LA) does not reduce long term depression severity. Estimates are small and statistically insignificant. In addition to analyzing the PHQ-9 means by arm, Figure A.1 shows the density of PHQ-9 scores by intervention arm. The densities of the arms are similar, and a Kolmogorov-Smirnov test fails to reject the equality of the distributions ($p > 0.1$). To examine possible impacts on specific PHQ-9 items, Figure A.2

shows estimates by item and also fails to find significant differences.

Despite the apparent lack of an impact, the confidence intervals do not allow us to reject moderately-sized effects, e.g., an impact of PC/LA of -0.16 SD, which are of similar size as the short-term effects of pharmacotherapy (Gartlehner et al., 2017). The high rate of spontaneous recovery by the control group is an important factor that limits the statistical power of this analysis, as we discuss next.

At some point between the third and eighth year after the intervention, the positive effects of PC/LA on depression faded out. To investigate when that occurred and how long it lasted, we collected retrospective histories of depression.

Figure 1: Prevalence of Moderate Depression over Time



Notes: ***, **, and * represent significant differences from the control group at the 1, 5, and 10% level. Red stars indicate the significance of the Any PC arms, blue stars indicate the significance of the PC/LA arm, and green stars indicate the significance of the PC arm. These estimates are from versions of equation 1, estimated by round through LASSO, with covariates from footnote 9 and clustering standard errors by locality. Any PC pools the PC and PC/LA arms. Depression is measured concurrently from the PHQ-9 (PHQ-9≥10) up to 27 months since the baseline and then at 90 months. Depression prevalence in the shaded area (42 to 78 months after the baseline) is estimated from depression histories recalled at 90 months. For those time periods, PHQ-9 data are unavailable. The recalled depression years, 2020 to 2023, overlap with the COVID pandemic. 95% confidence intervals.

Figure 1 shows that eight years after the intervention, depression prevalence is approximately 20% in all arms. This is consistent with findings that depression resolves gradually

for most people when untreated.[10] However, antidepressants help depression resolve sooner. The control group "catches up" to the pooled PC arms within 27 months, and sooner if we consider the PC arm only. Thus, a single course of SSRI accelerates recovery by at most 27 months in this sample.

5.2 Remission and Recurrence

Pharmacotherapy with SSRI is a first-line treatment for adults suffering from major depressive disorder. Benefits occur within 4-6 weeks. If effective, the medication is continued for at least 6–12 months following apparent clinical remission of acute depression at the full dosage, followed by gradual tapering that can last for months (Horowitz and Taylor, 2019). This approach is believed to prevent depression relapse and recurrence.[11] Premature discontinuation of medication can lead to high recurrence rates within 10 years since depression onset, most of which occur within six months of apparent clinical remission. A meta-analysis of continuation trials, which randomizes continued medication or placebo after remission, shows that the recurrence/relapse likelihood at 40 weeks is doubled (from 20 to 40%) after antidepressant discontinuation (Kato et al., 2021). Perhaps for this reason, the duration of a single course of antidepressants has increased over time. For example, in the United Kingdom, the interquartile range of a course of antidepressants has increased from 51-256 days in 1995 to 70-440 in 2010 (McCrea et al., 2016). This, together with an increase in the incidence of SSRI prescriptions, may lead to over-medication of depression (see, e.g., Currie and Zwiers (2023)).

Other meta-analyses challenge this view, showing that 4-6 month courses of antidepressants minimize the risk of relapse (Baldessarini et al., 2015). Thus, shorter courses of phar-

[10] E.g., Whiteford et al. (2013) estimate that 53% of untreated cases of major depression disorder remit within 12 months. Baranov et al. (2020) find a 30% depression prevalence 7 years after an untreated spell of perinatal depression. Bhat et al. (2022) find 20 and 45% depression prevalence 4 and 5 years after an untreated spell of depression.

[11] A relapse is the return of depressive symptoms before full recovery (mostly within 6 months from improvement). A recurrence is a new depressive episode after full recovery. The two concepts are often considered jointly in the literature, as there is ambiguity in the definitions and no biomarker to demarcate a full recovery from a depressive episode. Burcusa and Iacono (2007) describes the fluidity in the definition of relapse and recurrence.

macotherapy may be advisable. Lastly, in LMICs and other settings with scarce resources, it may not be feasible to offer more than a short course of SSRI. Thus, a more pertinent question is whether the rates of recurrence for a short course are sufficiently low. Answering this question involves comparing recurrence between the treatment and control arms, as we do here.

Given this knowledge, and absent long-term follow-ups in almost all clinical studies, the current data are a unique opportunity to look at the effect of a short course of SSRI on depression recurrence in the treatment and control groups. If pharmacotherapy accelerates recovery but then causes frequent relapses, its initial gains may be more than offset by later losses in mental wellbeing. [12]

[12]This part of the analysis was not specified in our trial pre-registration.

Table 2: Prevalence of Recurrence and Remission

	Using PHQ-9 data only		Using PHQ-9 data & recalled depression history	
	Remission	Recurrence	Remission	Recurrence
	(1)	(2)	(3)	(4)
Panel A: Pooled PC				
Any PC	-0.001	0.007	-0.003	0.001
	(0.009)	(0.005)	(0.006)	(0.005)
Panel B: PC/LA and PC				
PC/LA	0.005	0.009	-0.005	0.004
	(0.012)	(0.007)	(0.008)	(0.007)
PC	-0.004	0.005	-0.003	-0.003
	(0.010)	(0.006)	(0.007)	(0.007)
Control Mean	0.074	0.020	0.075	0.052
PC/LA=PC	0.482	0.601	0.834	0.365
N	4385	4385	8005	8005
Specification	LASSO	LASSO	LASSO	LASSO

Notes: We estimate the parameters of the following equation: $Y_{ijr} = \phi_0 + \phi_1 AnyPC_j + \phi_2 LA_j + \phi_3 X_{ij} + \omega_{ijr}$, where the outcome $Y_{ijr} = 1$ for a person i in locality j at round $r > 1$ is, alternatively, depression remission and recurrence. We define a remission as $P(PHQ-9 < 5, t | PHQ-9 \geq 10, t-1) = 1$, and a recurrence as $P(PHQ-9 \geq 10, t | PHQ-9 < 5, t-1) = 1$. We select the X_{ij} coefficients through LASSO using the covariates described in footnote 9. Different versions of the above equations estimate separate coefficients for the PC/LA and PC arms. In columns 1-2, the outcomes are created using PHQ-9 data only (up to 6 observations per respondent), while in columns 3-4 we also consider the recalled depression history from 2020 to 2023 (up to 10 observations per respondent). Standard errors clustered by locality in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table 2 shows that there are no statistically or clinically significant differences in remission and recurrence rates: the estimated effect sizes are less than one percentage point ($p < 0.10$). These estimates and their confidence intervals show recurrence rates in the PC arms that are 1-2 orders of magnitude lower than the 40% recurrence rates in Kato et al.

(2021). The coefficient estimates for recurrence and remission remain small, statistically insignificant, and precisely estimated also when we include the recalled depression histories. Thus, there is no evidence that an eight-month course of antidepressants increases depression recurrence more than in the control group. Instead, it seems that antidepressants accelerate recovery without increasing recurrence. These findings may be important to scale the provision of antidepressants in this setting and suggest that shorter courses of antidepressants than commonly prescribed in high-income countries may be effective at accelerating remission without exacerbating recurrence.

6 Awareness, Beliefs, and Care Seeking

The intervention may have changed people's awareness of depression symptoms, what treatment is effective and how to seek it, as well as changed their beliefs about depression.

6.1 Knowledge and Awareness

In Table 3, Columns 1-3 show the AIT effects on the three indices for depression knowledge and awareness. The index components range between 1 and 5, with higher values meaning better awareness. The control group has a 3.8 average score for knowledge related to the intervention (knowing that psychiatrists can prescribe antidepressants and that antidepressants are an effective treatment for depression), and a 3.1 average knowledge unrelated to the intervention (the efficacy or psychotherapy, the availability of antidepressants at local pharmacies, and local public hospitals' ability to prescribe antidepressants). It also displays a high awareness of depression and its core symptoms, with a mean of 4.1. We label the standardized indices "direct knowledge," "indirect knowledge," and "depression awareness."

The treatment increases direct knowledge by 0.17 SD ($p < 0.05$), with an effect that does not vary for the PC and PC/LA arms ($p > 0.10$). Conversely, there is no significant impact on indirect knowledge. This is not surprising, since the 2016 study did not provide this information. Lastly, there is no economically or statistically significant effect on awareness

Table 3: Impacts on Knowledge and Depression Awareness, and Beliefs about Depression (Positive sign = more awareness; less negative beliefs)

	Knowledge & Awareness (STD)			Beliefs (STD)			
	Direct Knowledge	Indirect Knowledge	Depression Awareness	Overall Beliefs	Depression Attributes	Societal Costs	Costs of Dep. Relative
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Pooled PC							
Any PC	0.158**	0.009	-0.048	0.097	0.042	0.091	0.125*
	(0.078)	(0.083)	(0.070)	(0.069)	(0.071)	(0.071)	(0.074)
Panel B: PC/LA and PC							
PC/LA	0.122	-0.024	-0.015	0.005	-0.007	0.007	0.065
	(0.097)	(0.107)	(0.082)	(0.082)	(0.090)	(0.085)	(0.086)
PC	0.193**	0.023	-0.098	0.188**	0.081	0.185**	0.204**
	(0.091)	(0.095)	(0.085)	(0.086)	(0.085)	(0.088)	(0.096)
PC/LA=PC	0.500	0.685	0.368	0.056	0.393	0.072	0.186
N	905	901	909	909	909	909	909
Specification	LASSO	LASSO	LASSO	LASSO	LASSO	LASSO	LASSO

Notes: The control group mean is 0. "Direct Knowledge:" standardized first principal component of medical knowledge related to the intervention (knowing that psychiatrists can prescribe antidepressants and that antidepressants are an effective treatment for depression). "Indirect Knowledge:" standardized first principal component of medical knowledge unrelated to the intervention (the efficacy or psychotherapy, the availability of antidepressants at local pharmacies, and local public hospitals' ability to prescribe antidepressants). "Dep awareness:" standardized first principal component of knowledge of depression and its core symptoms. "Overall beliefs:" standardized first principal component of the pooled components of the next three indices. "Depression attributes:" beliefs about depression attributes (e.g., whether people with depression are lazy or can take care of their children). "Societal costs:" beliefs about the societal costs of depression (e.g., whether people will want to hire, befriend, or marry people with depression). "Costs of Dep. Relative:" beliefs about the costs of having a depressed household member (e.g., whether it will be difficult for a daughter-in-law to take care of a depressed parent-in-law). Covariates are chosen through LASSO from the set in footnote 9. Standard errors clustered by locality in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

of the symptoms of depression, jointly or individually, possibly because awareness is already high in the control group and, thus, there is limited room for improvement.

6.2 Beliefs

Many people have negative beliefs about depression. For example, 76% of the control group believes that people with depression are lazy, and 89% that they cannot take care of themselves; 93% and 83% believe that it is difficult for a person with depression to find a spouse or a job; and 87% that a daughter-in-law will have a hard time taking care of a depressed in-law.

6.3 Care Seeking

Earlier, we conjectured that the pharmacotherapy intervention may have affected both people's knowledge and perceptions about depression and how they react to them. For example, a person who received a depression diagnosis and a prescription for antidepressants and whose mental health subsequently improved may have become more aware about the symptoms of depression, how to seek treatment, that treatment can be effective. In addition, experiential learning may have decreased the cost of care-seeking. This may induce people who experience future spells of depression to seek treatment, thus improving their future mental health through medical care.

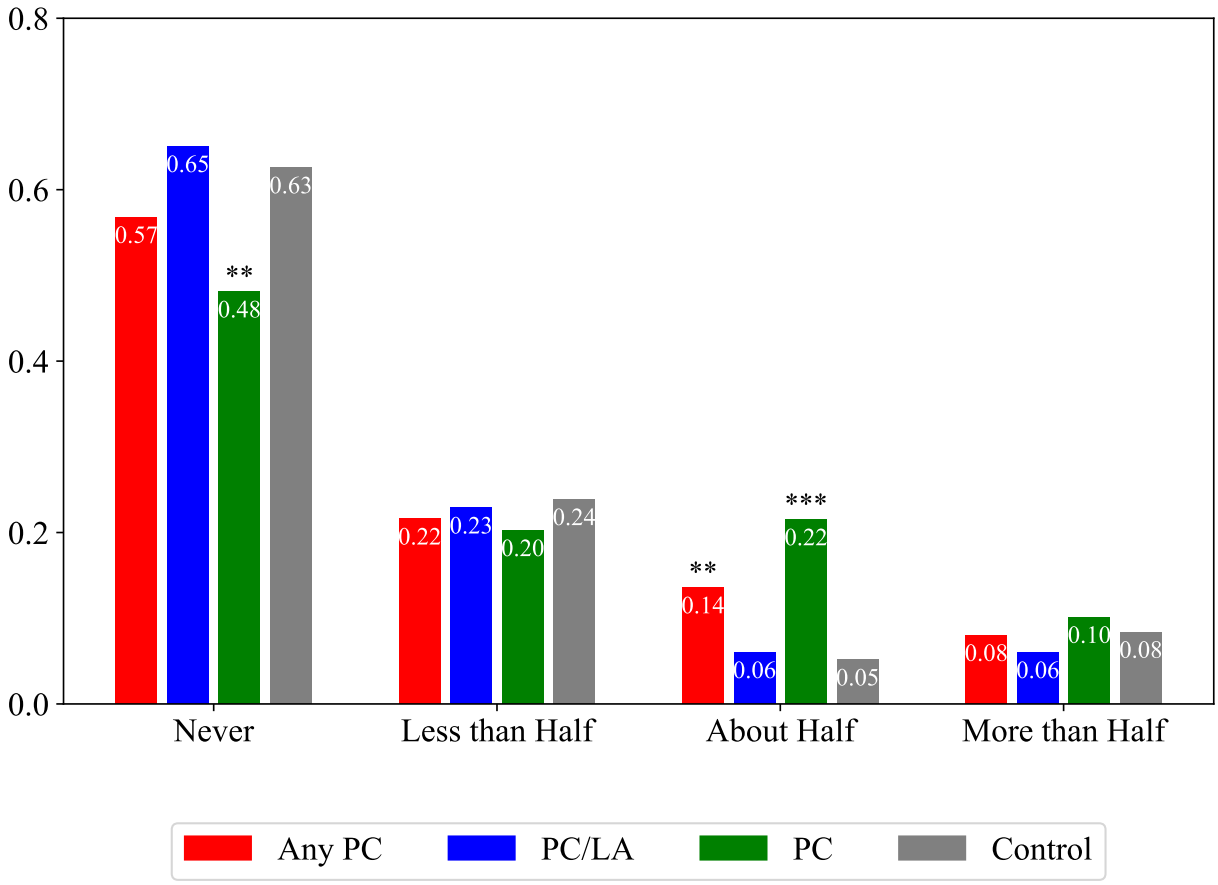
Figure 2 provides suggestive evidence for this hypothesis: the likelihood of seeking care half the time after experiencing spells of depression increases from 5% to 14% in the pooled arms ($p<0.05$) and 22% in the PC arm ($p<0.01$), with analogous decreases in the likelihood of never or infrequently seeking care. When we compare the frequency of these responses with the control group, we weakly reject the hypothesis of equality of frequency for the pooled PC arms ($p = 0.10$) and we reject it for the PC arm ($p < 0.01$), but not for the PC/LA arm ($p > 0.10$).

6.4 Do these effects vary by arm?

The long-term effects of offering pharmacotherapy on depression awareness and stigma at times appeared to vary between the PC and PC/LA arms. To test whether these differences are systematic, we estimate the equations for these outcomes as a system and test the hypothesis that the treatment effects are jointly equal for the two arms using a SUR estimator. We fail to reject this hypothesis with a p-value of 0.28 (and 0.57 if we also include the symptoms of depression at 90 months, the outcome from Table 1).[13]

[13]The outcomes of this system of equations are: direct knowledge, indirect knowledge, depression awareness, overall depression beliefs (columns 1-4 in Table 3), and PHQ-9 (column 2 from Table 1. If we replace overall depression beliefs with its 3 components (using the outcomes from columns 5-7 in Table 3), the results do not change. We cannot use the care-seeking outcomes described in the next section because we observe them only for people who experienced depression since 2018.

Figure 2: Impact on seeking depression treatment (conditional on being depressed)



Notes: We tested the hypothesis that care seeking differs from the control group's in each arm by estimating versions of equations 1 and 2, selecting covariates through LASSO from the list in footnote 9 and clustering standard errors by locality. The dependent variables are 4 indicators for how often people sought treatment when depressed between 2018 and 2023 (never, less than half the time, about half the time, and more than half the time). Any PC pools the PC and PC/LA arms. ***, **, and * represent statistically significant differences from the control group at the 1, 5, and 10% level. The p-values of the Pearson's χ^2 test of pairwise equality of distributions are: Any PC vs. Control: p=0.10; PC/LA vs. Control: p=0.78; PC vs. Control: 0.01.

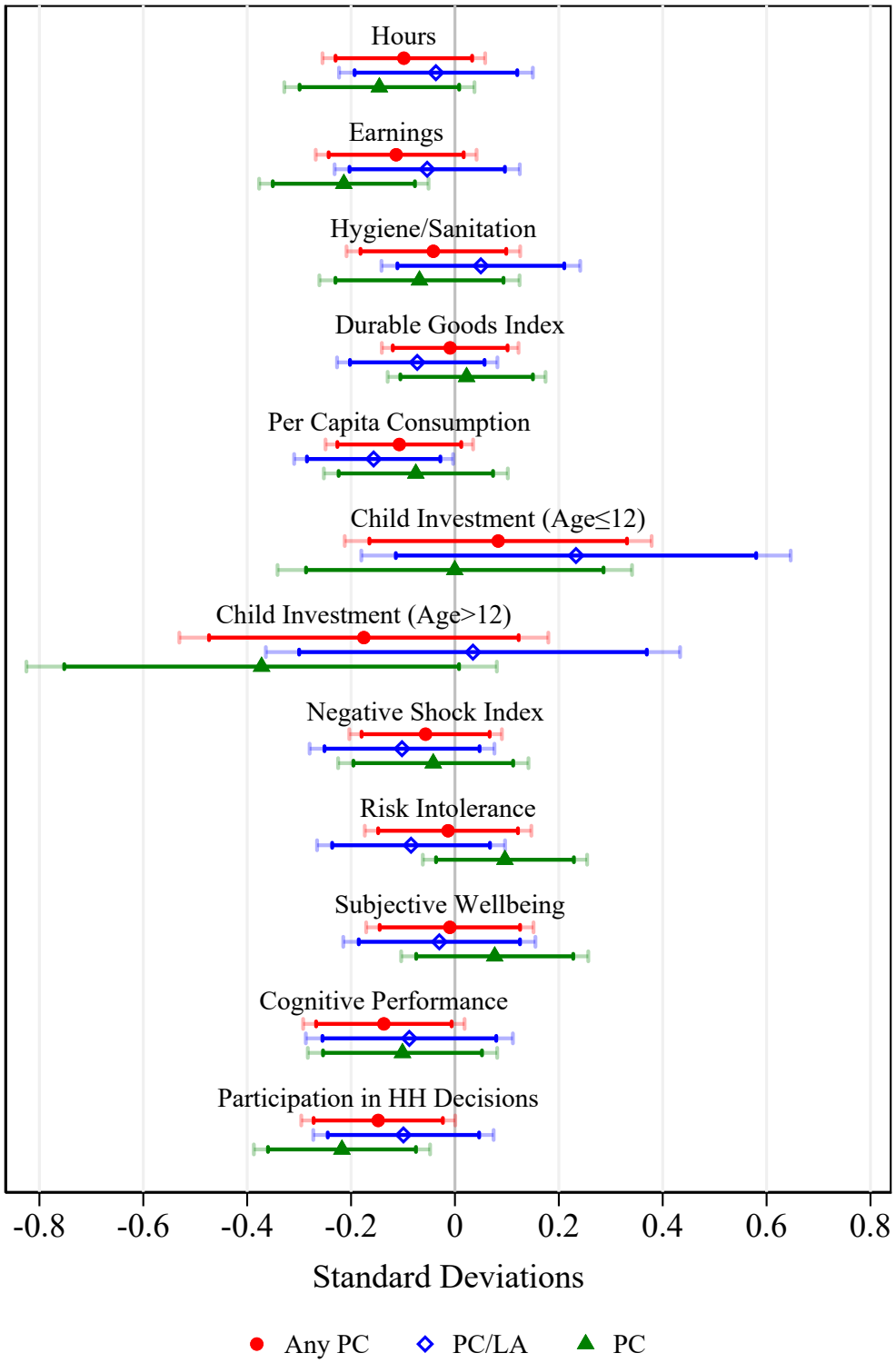
7 Socioeconomic Impacts

Figure 3 shows that the PC arms did not cause any statistically or economically significant improvements in all other in individual and household outcomes and did not improve any of the considered pathways. The only exceptions are earnings and participation in household decisions, which decrease by 0.2 SD in the PC arm ($p < 0.05$) but not in the pooled PC arms. These findings are broadly consistent with our hypotheses, since, with few exceptions, there were no positive short- and medium-term outcomes of pharmacotherapy on these outcomes. The lack of long-term increases in child human capital investment, decreases in incidence of risk, and increases in consumption suggest that any initial effects of pharmacotherapy on investment detected in Angelucci and Bennett (2024) were at most temporary and did not cause any long-term reductions in poverty.

8 Discussion

Antidepressants can provide treatment when personnel are scarce, as occurs in India and other LMICs. However, there is little evidence about their effectiveness in community settings, since most research focuses on short-term impacts in clinical settings in industrialized countries. Moreover, the long-term impacts of a short course of antidepressants are not well-understood, in developing countries or elsewhere. This medication may induce long-lasting physiological changes that affect depression recurrence in ambiguous directions even after the treatment itself has been discontinued. Additionally, there may be long-term impacts unmediated by biological changes: exposing people to treatment can make people more aware of the symptoms of depression, what treatment is effective, and how to seek it. If so, a single course of treatment can have lasting impacts by teaching people how to recognize symptoms and seek care. Lastly, increased familiarity with depression symptoms and treatment can also dispel harmful beliefs about people with depression, contribute to normalize the condition, and thus alleviate self-stigma and stigma in the household. This channel can

Figure 3: Impacts on Standardized Socioeconomic Outcomes and Pathways



Notes: Standardized impacts for socioeconomic outcomes and pathways. Light (dark) bars indicate 95 (90) percent confidence intervals. Standard errors clustered by locality. Estimates from equations 1 and 2 select controls using post-double-selection LASSO with covariates from footnote 9.

also have lasting beneficial impacts within the household. Thus, this research contributes to closing this knowledge gap by estimating the long-run impacts of pharmacotherapy in poor communities in India. Understanding these benefits helps inform policymakers about the desirability of scaling up the provision of pharmacotherapy.

We found that offering a short course of antidepressants accelerates recovery without causing recurrence rates that undo the initial benefits of medication. However, the control group catches up by month 27 and rates do not differ after. Thus, there are no improved symptoms of depression 7 years later, likely because depression has spontaneously resolved in 80% of the control group.

The offer of antidepressants affects treatment knowledge and beliefs about depression. While we observe no impact on awareness of key depression symptoms – likely due to a high baseline level of knowledge among participants – there is evidence of improved awareness of available treatment options. This is consistent with the hypothesis that experiential learning enhances individuals' understanding of mental health care possibilities and may potentially offset biologically-driven relapses. That is, the short course of pharmacotherapy may increase the future prevalence of depression through a biological pathway, but at the same time decrease this prevalence through a knowledge/behavioral pathway. These two effects may offset each other in our sample.

Thus, improved treatment knowledge and depression beliefs may increase care-seeking and potentially mediate depression outcomes. Indeed, we find an increase in mental health care-seeking behavior among individuals experiencing depressive episodes. However, despite this positive trend, many depressive episodes remain untreated. This gap underscores the need for a deeper examination of barriers to accessing care, and raises important questions about how to design interventions that effectively bridge this gap to ensure adequate treatment coverage.

Lastly, our findings suggest that a single course of pharmacotherapy does not yield long-term socioeconomic benefits. We should be cautious about concluding that depression does not matter for long-term poverty. Reductions in mental illness are linked to greater pro-

ductivity in the short run (e.g. Lund et al., 2024), which could lead to long-term poverty reduction. Secondly, the extent of spontaneous recovery in the control arm limits our ability to understand the socioeconomic impact of being depressed over the long term. Thirdly, interventions such as providing pharmacotherapy or psychotherapy inherently bundle diagnosis and treatment. Therefore, in settings with high depression stigma, such as our study area, a formal diagnosis may undo some of the beneficial socioeconomic impacts of better mental health by, e.g., reducing the opportunity for employment and socialization. Finally, this sample is 86% female. Given the low rates of female labor force participation, these effects may differ by gender.

This study is useful to understand barriers to the delivery of mental health care in LMICs. While addressing supply-side barriers to care is important, our findings highlight both the potential and the limitations of pharmacotherapy as a scalable intervention in these settings. We find that increasing the supply of pharmacotherapy is helpful but insufficient on its own to address the full scope of the mental health care gap. On the positive side, a short course of pharmacotherapy can accelerate recovery from depression without increasing the likelihood of recurrence. A shorter course of SSRI seems adequate in this context. This may be optimal when health care providers or medications are scarce. However, there remain significant challenges on the demand side. Despite high levels of awareness regarding depression symptoms, demand for treatment remains low. This may be partly driven by limited knowledge about available treatments and their efficacy, as well as pervasive mental health stigma, which increases the costs of seeking care. This study suggests that prior direct experience with the local health care system can increase future mental health care seeking, but not enough to close the care gap even when health care is free.

To address these challenges, interventions must go beyond increasing the availability of pharmacotherapy. Regular screening for depression and active guidance through the treatment process appear essential, as most individuals do not independently seek care. These findings underscore the importance of integrating demand-side interventions, such as improving treatment awareness and addressing beliefs about care, into policies designed to

close the mental health care gap in LMICs. We speculate that community health workers, who routinely provide basic health care and connect families with the health system, may play an active role in increasing awareness about mental health care and connecting people in need with the right providers. Apps and artificial intelligence may play a similar role in the future. Lastly, broad-scale action may be needed to systematically affect mental health stigma at a societal level.

A Appendix – Not for Publication

A.1 Data Creation: Depression Awareness and Beliefs

We collected the relevant outcomes from a series of statements about awareness of depression symptoms, effective treatment and where to seek it, and depression beliefs, to which people could agree or disagree on a scale from 1 (complete disagreement) to 5 (complete agreement). After collecting the data, we changed the signs of each outcome such that a bigger value means either a correct statement (for objective statements related to depression awareness) or a positive statement (for depression beliefs).[14] All the indices that we create, which we describe below, are the first principal component, standardized around the control group mean.

A.1.1 Depression Awareness

Depression awareness encompasses awareness of the symptoms of depression and awareness of effective depression treatment and where to seek it. To measure awareness of the symptoms of depression, we create an index from four outcomes: agreement with the statement "I know what depression is" and knowledge of core symptoms of depression: anhedonia, sadness, tiredness, and hopelessness. To measure anhedonia (a term most respondents are unfamiliar with), we asked how much they agreed with following two statements: "People with depression are often detached from what is going on around them," and "People with depression do not enjoy life's good things."

To measure awareness of effective depression treatment and where to seek it, we create two separate indices to distinguish knowledge that participants acquired during the intervention through experiential learning, and knowledge that the intervention did not directly affect. The first group encompasses whether antidepressants are effective as a treatment

[14] For example, the statement "Psychiatrists can offer treatment with antidepressants" is correct. Thus, we gave lower values to disagreement and higher values to agreement. Conversely, the belief that "people with depression are attention-seekers" is a negative belief about depression. Thus, we gave lower values to agreement and higher values to disagreement.

for depression and whether psychiatrists can offer treatment with antidepressants. This is what participants in the PC and PC/LA arms experienced. The second group encompasses whether psychotherapy is effective as a treatment for depression, whether doctors at the local hospital can offer treatment with antidepressants, and whether antidepressant medications are available from nearby pharmacies. All of the above is correct, but the intervention did not provide this type of experiential knowledge to its participants, since they did not interact with the local hospital and pharmacies and did not receive psychotherapy.

A.1.2 Depression Beliefs

We measure participants' beliefs of depression and the related stigma through a battery of thirty statements belonging to three different groups. First, we measure beliefs about depression's positive and negative attributes (e.g., intelligence, laziness) and costs (e.g., ability to look after themselves or others). Then, we measure beliefs about the costs of depression in the marriage and labor markets (e.g., costs and difficulties of finding a spouse, likelihood and extent of labor discrimination) and the risk of violence (e.g., restriction of movement, insults, physical violence). Finally, we measure beliefs of the perceived social, marriage, employment, and household cost of having a household member with depression (e.g., whether people want to socialize with a family with a depressed member, or how difficult it is for a daughter-in-law to look after parents-in-law with depression). Each individual variable is measured of a 1-5 Likert Scale.

We create four indices: first, we pool all beliefs; then, we disaggregate its three subgroups: beliefs about depression attributes, beliefs about societal costs of depression, and beliefs about having a depressed household member.

A.2 Assessing Recall Bias

Angelucci and Bennett (2024) measured the prevalence of depression in 2018 and 2019. Thus, for those two years we observe both the concurrent and recalled rates of depression. Comparing the concurrent and recalled rates can help us quantify the magnitude and direction

of recall bias.[15]

Table A.1 shows that the recalled depression prevalence in the C, PC, and LA arms is 40-45% of the concurrent prevalence in 2018 and 70-77% in 2019. That is, there is severe recall bias in 2018, but it improves by about 25-30 percentage point in 2019. under-report the prevalence of depression by about. Recall bias is lower in the PC/LA arm – about 76% in 2018, but it also improves by 25 percentage points by 2019. If recall bias continues to decrease by about 25 percentage points per year in the C, PC, and LA arms, it is plausible to assume that recall bias is negligible in all arms by 2020. Thus, we can use the recalled prevalence of depression in 2020 to 2023, approximately 3.5 to 7.5 years since the start of the intervention, to look at how a single course of antidepressant has affected the yearly prevalence of depression for the following 7 years. In any case, we ask the reader to exercise caution when considering data from the recalled depression histories.

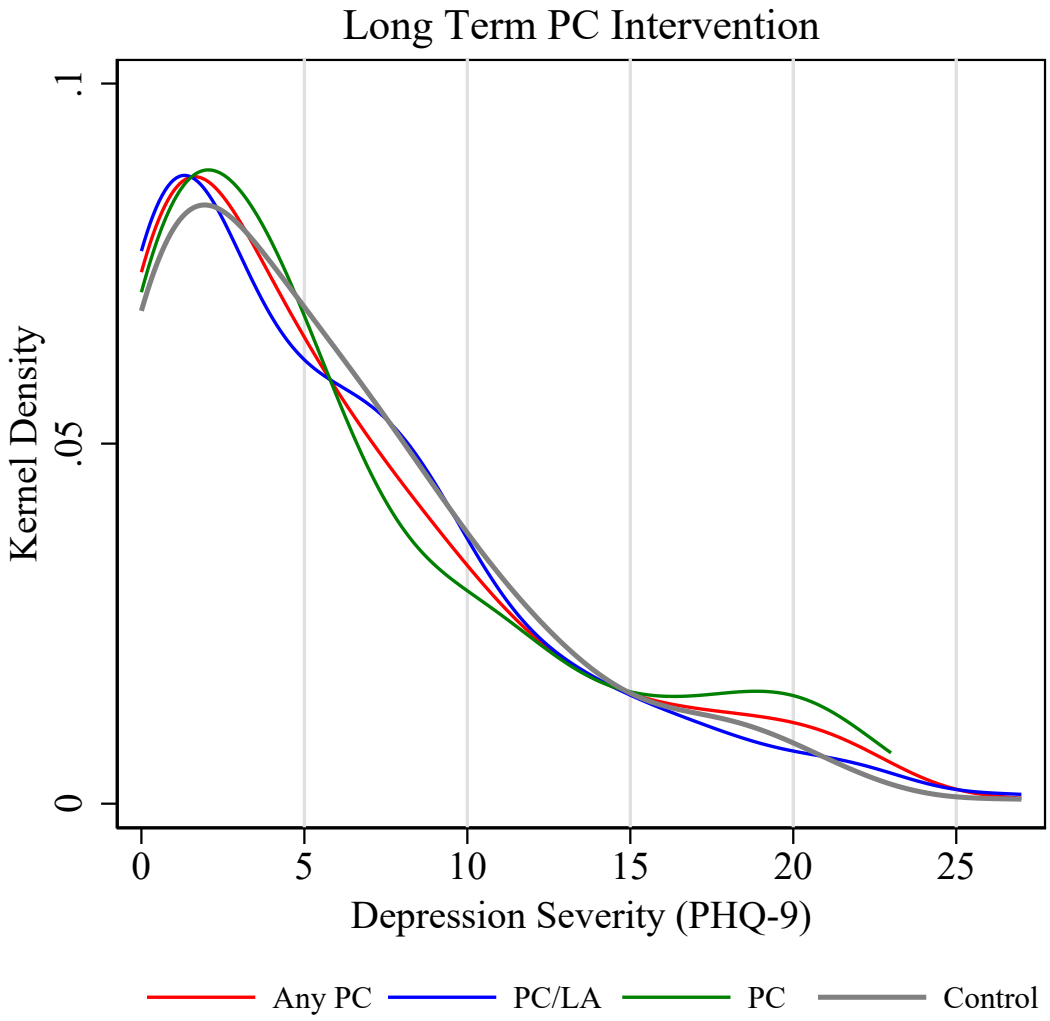
[15]The recall time differs for concurrent and recalled depression. For concurrent depression, the relevant time frame is the previous two weeks. For recalled depression, the time frame is one year. Thus, some people who were not depressed at the time of the interview may have been depressed earlier or later in the year. Subsequently, the recalled rates of depression exceed the concurrent rates of depression even with perfect recall. Moreover, the measurement of concurrent and recalled depression differs. Surveyors measured concurrent depression through the PHQ-9, which has a high diagnostic validity (Kroenke, Spitzer and Williams, 2001*b*). Thus, almost all people with a PHQ-9>9 had Major Depressive Disorder. Conversely, we measure recalled depression by asking participants if and for how long they were depressed in a given year. Thus, some people may believe that they were depressed when they in fact were not, and *vice versa*. These differences complicate our assessment of recall bias. Nevertheless, we expect the recalled and concurrent depression prevalence to be similar.

Table A.1: Recalled (RE) and Concurrent (CO) Depression Prevalence by Arm and Year

	2018			**2019**		
	Concurrent	**Recalled**	**RE/CO**	**Concurrent**	**Recalled**	**RE/CO**
PC/LA	0.32	0.24	0.76	0.26	0.26	1.01
PC	0.38	0.17	0.45	0.33	0.23	0.69
LA	0.43	0.18	0.40	0.32	0.23	0.70
Control	0.46	0.18	0.39	0.29	0.23	0.77

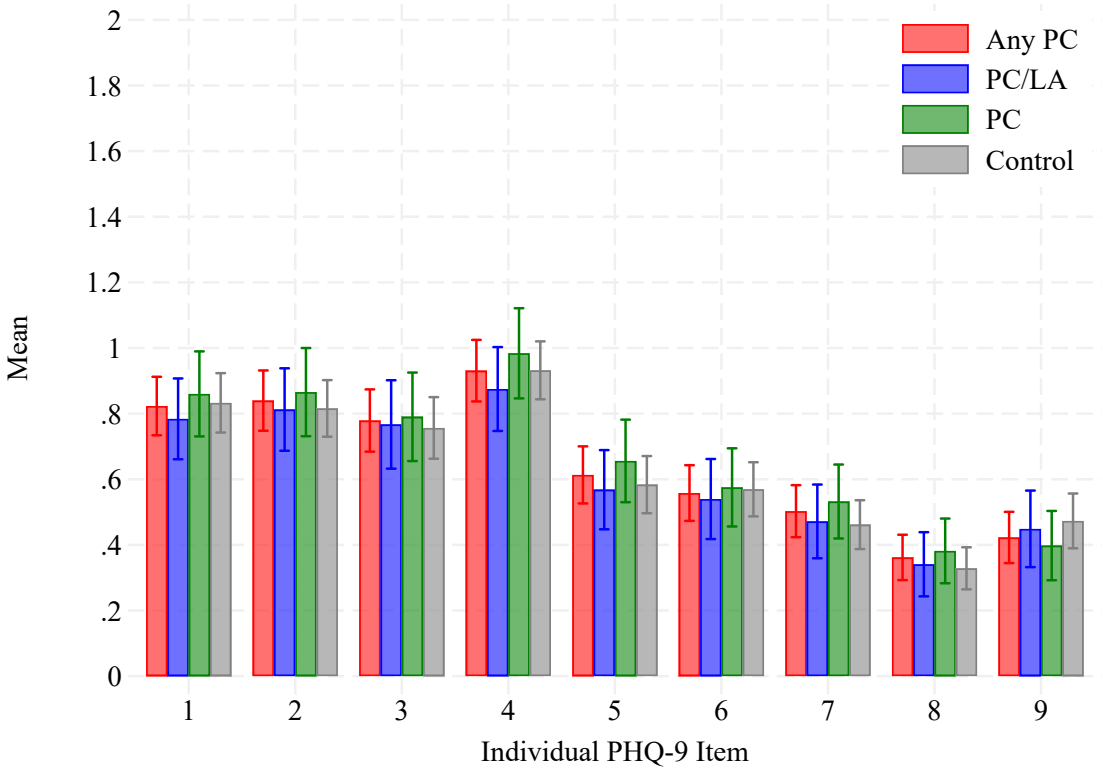
Notes: Prevalence of recalled depression from retrospective depression histories. Prevalence of concurrent from the PHQ-9 (PHQ-9$\geq$10). RE/CO = ratio of recalled over concurrent prevalence.

Figure A.1: PHQ-9 Density by Arm



Notes: Kernel density estimates of depression severity (PHQ-9) by intervention group. Kolmogorov-Smirnov tests comparing distributions indicate statistically insignificant differences. For Any PC vs. Control: p=0.98; for PC/LA vs. Control: p=0.68; and for PC vs. Control: p=0.96.

Figure A.2: Impacts on Individual PHQ-9 Questions by Arm



Notes: Scores of each of the 9 PHQ-9 questions by arm measured at 90 months since the baseline. Each question is scored on a scale from 0 to 3. Higher values mean higher severity of specific symptoms of depression. 95% confidence intervals.

B Additional Tables and Figures – Not for Publication

The following Tables and Figures reproduce the paper's 5 exhibits adding the coefficient estimates for the LA arm. These estimates are never significant, with the exception of human capital investment for children aged 12 and younger. This outcome increases by 0.35 SD ($p < 0.05$). Figure B.1 is the CONSORT chart.

Table B.1: Impact on Depression Symptoms (PHQ-9) - All Arms

	PHQ-9 (STD)	
	(1)	(2)
Panel A: Pooled PC		
Any PC	0.042	0.046
	(0.079)	(0.079)
Panel B: PC/LA and PC		
PC/LA	0.014	0.012
	(0.091)	(0.091)
PC	0.067	0.072
	(0.105)	(0.106)
LA	0.050	0.072
	(0.083)	(0.080)
PC/LA = PC	0.652	0.613
PC/LA = LA	0.719	0.534
Any PC = LA	0.923	0.743
N	909	909
Specification	ANCOVA	LASSO

Notes: The PHQ-9 is standardized. The standardized control group mean is 0. The non-standardized control group mean is 5.75. Standard errors clustered by locality in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$. The ANCOVA estimator conditions on the baseline value of the dependent variable and strata fixed effects. The LASSO estimator of Belloni, Chernozhukov and Hansen (2014) chooses covariates from a list described in footnote 9.

Table B.2: Prevalence of Recurrence and Remission - All Arms

	Using PHQ-9 data only		Using PHQ-9 data & recalled depression history	
	Remission	Recurrence	Remission	Recurrence
	(1)	(2)	(3)	(4)
Panel A: Pooled PC				
Any PC	-0.000	0.007	-0.003	0.000
	(0.009)	(0.005)	(0.006)	(0.005)
Panel B: PC/LA and PC				
PC/LA	0.005	0.009	-0.005	0.004
	(0.012)	(0.007)	(0.008)	(0.007)
PC	-0.004	0.005	-0.003	-0.003
	(0.010)	(0.006)	(0.007)	(0.007)
LA	0.001	0.009	0.000	0.002
	(0.010)	(0.006)	(0.007)	(0.006)
Control Mean	0.074	0.020	0.075	0.052
PC/LA=PC	0.482	0.601	0.834	0.365
PC/LA=LA	0.763	0.945	0.589	0.835
AnyPC=LA	0.934	0.686	0.624	0.756
N	4385	4385	8005	8005
Specification	LASSO	LASSO	LASSO	LASSO

Notes: We estimate the parameters of the following equation: $Y_{ijr} = \phi_0 + \phi_1 AnyPC_j + \phi_2 LA_j + \phi_3 X_{ij} + \omega_{ijr}$, where the outcome $Y_{ijr} = 1$ for a person i in locality j at round $r > 1$ is, alternatively, depression remission and recurrence. We define a remission as $P(PHQ - 9 < 5, t | PHQ - 9 \geq 10, t-1) = 1$, and a recurrence as $P(PHQ - 9 \geq 10, t | PHQ - 9 < 5, t-1) = 1$. We select the X_{ij} coefficients through LASSO using the covariates described in footnote 9. Different versions of the above equations estimate separate coefficients for the PC/LA and PC arms. In columns 1-2, the outcomes are created using PHQ-9 data only (up to 6 observations per respondent), while in columns 3-4 we also consider the recalled depression history from 2020 to 2023 (up to 10 observations per respondent). Standard errors clustered by locality in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Table B.3: Impacts on Depression and Treatment Awareness, and Beliefs about Depression - All Arms (Positive sign = more awareness; less negative beliefs)

	Knowledge & Awareness (STD)			Beliefs (STD)			
	Direct Knowledge	Indirect Knowledge	Depression Awareness	Overall Beliefs	Depression Attributes	Societal Costs	Costs of Dep. Relative
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Panel A: Pooled PC							
Any PC	0.158**	0.009	-0.048	0.097	0.042	0.091	0.125*
	(0.078)	(0.083)	(0.070)	(0.069)	(0.071)	(0.071)	(0.074)
Panel B: PC/LA and PC							
PC/LA	0.122	-0.024	-0.015	0.005	-0.007	0.007	0.065
	(0.097)	(0.107)	(0.082)	(0.082)	(0.090)	(0.085)	(0.086)
PC	0.193**	0.023	-0.098	0.188**	0.081	0.185**	0.204**
	(0.091)	(0.095)	(0.085)	(0.086)	(0.085)	(0.088)	(0.096)
LA	0.049	0.094	0.045	0.018	0.004	-0.003	0.044
	(0.093)	(0.092)	(0.076)	(0.086)	(0.082)	(0.088)	(0.093)
PC/LA=PC	0.500	0.685	0.368	0.056	0.393	0.072	0.186
PC/LA=LA	0.494	0.300	0.469	0.891	0.917	0.922	0.843
AnyPC=LA	0.221	0.351	0.189	0.358	0.671	0.285	0.384
N	905	901	909	909	909	909	909
Specification	LASSO	LASSO	LASSO	LASSO	LASSO	LASSO	LASSO

Notes: The control group mean is 0. "Direct Med:" standardized first principal component of medical knowledge related to the intervention (knowing that psychiatrists can prescribe antidepressants and that antidepressants are an effective treatment for depression). "Indirect Med:" standardized first principal component of medical knowledge unrelated to the intervention (the efficacy or psychotherapy, the availability of antidepressants at local pharmacies, and local public hospitals' ability to prescribe antidepressants). "Dep awareness:" standardized first principal component of knowledge of depression and its core symptoms. "Overall beliefs:" standardized first principal component of the pooled components of the next three indices. "Depression attributes:" beliefs about depression attributes (e.g., whether people with depression are lazy or can take care of their children). "Societal costs:" beliefs about the societal costs of depression (e.g., whether people will want to hire, befriend, or marry people with depression). "Costs of Dep. Relative:" beliefs about the costs of having a depressed household member (e.g., whether it will be difficult for a daughter-in-law to take care of a depressed parent-in-law). Covariates are chosen through LASSO from the set in footnote 9. Standard errors clustered by locality in parentheses. $^{*}p < 0.1$, $^{**}p < 0.05$, $^{***}p < 0.01$.

Figure B.1: CONSORT Chart

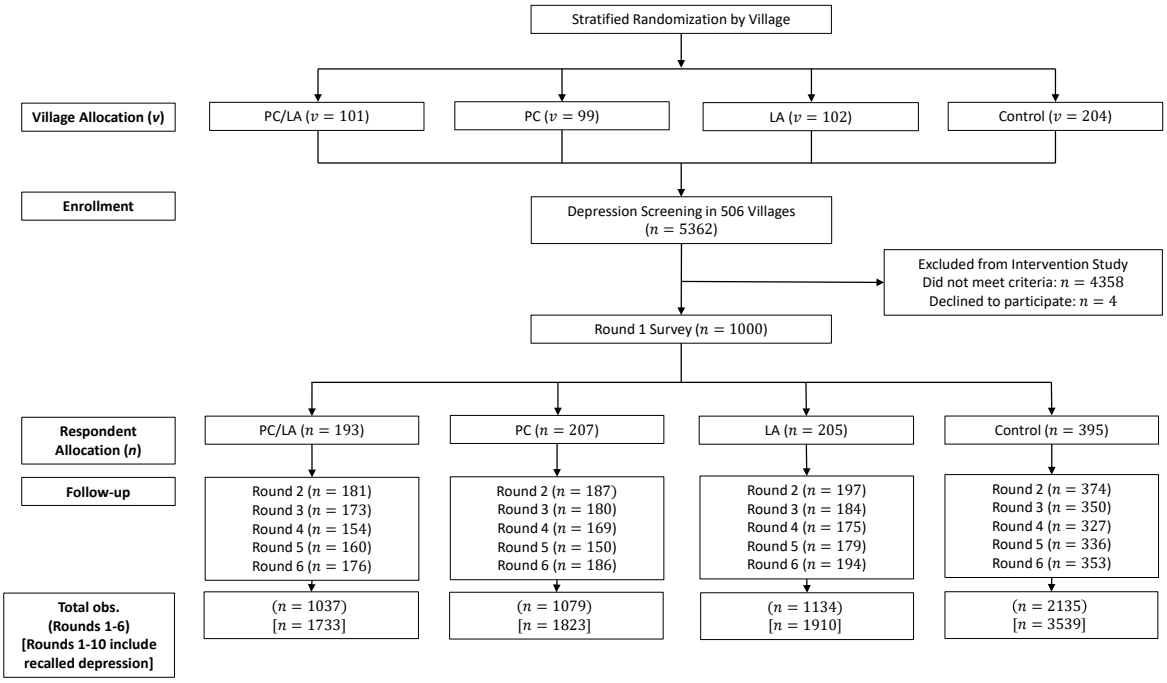
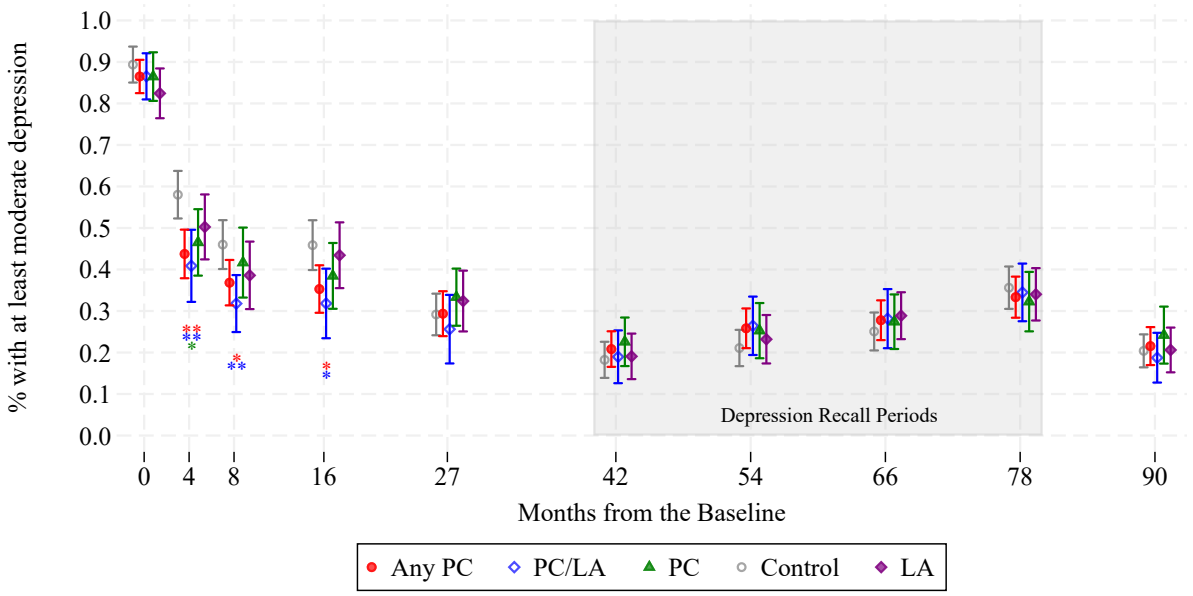
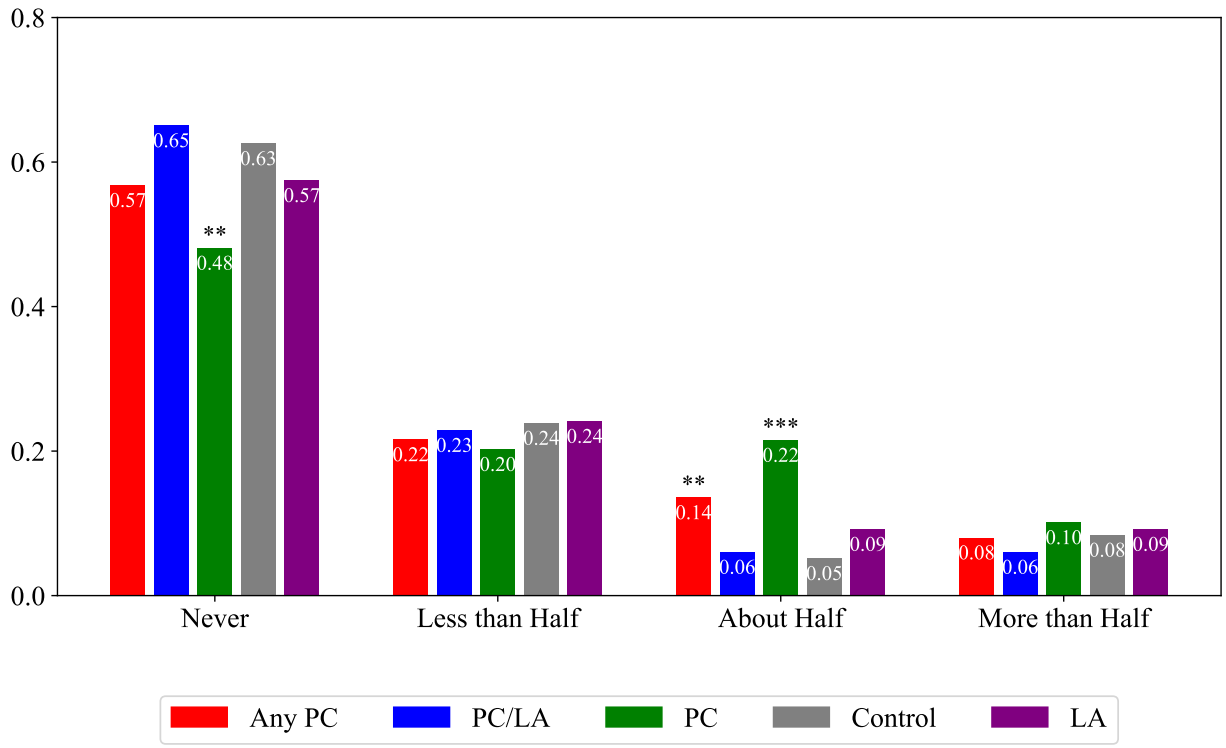


Figure B.2: Prevalence of Moderate Depression over Time - All Arms



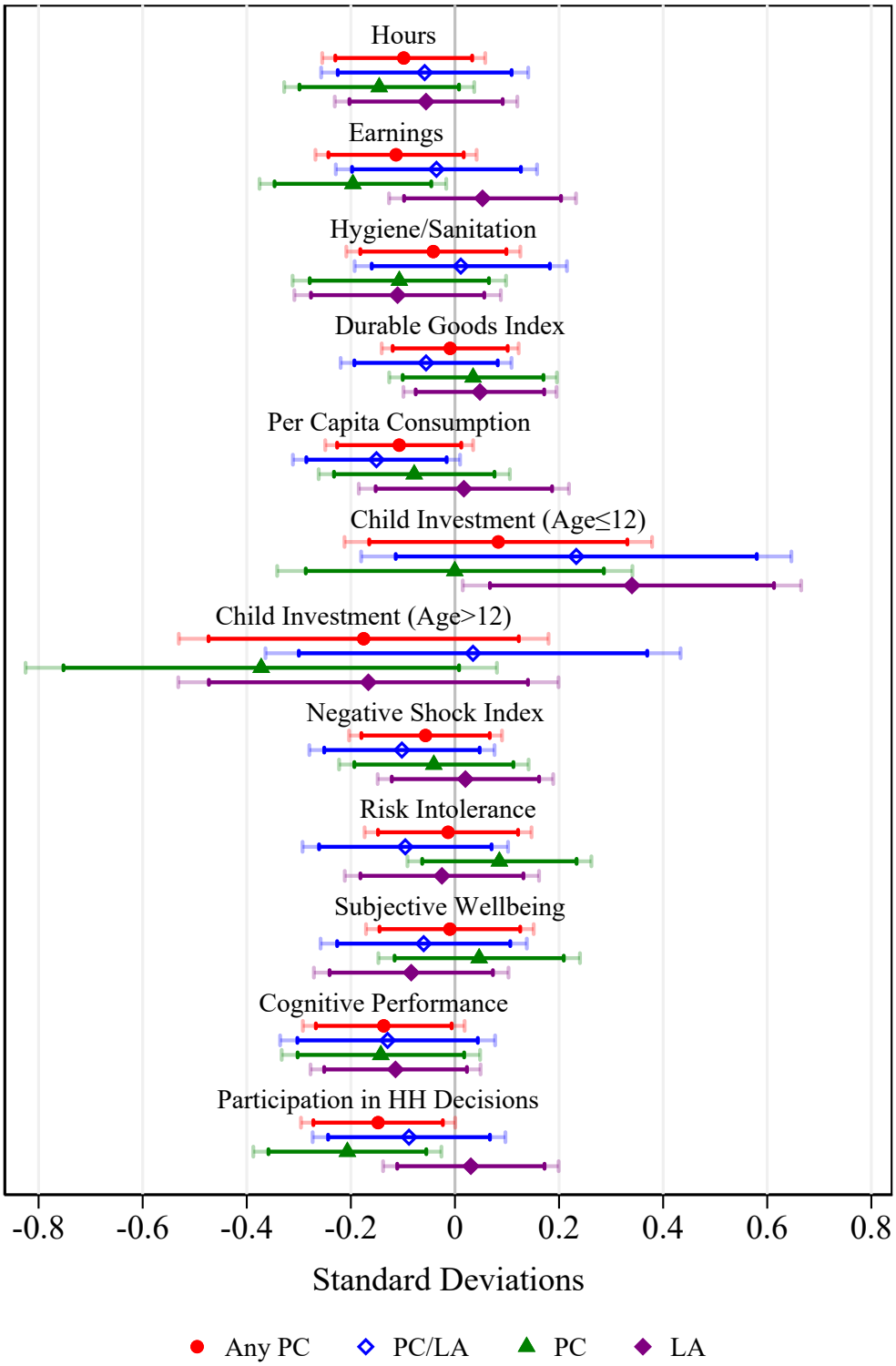
Notes: ***, **, and * represent significant differences from the control group at the 1, 5, and 10% level. Red stars indicate the significance of the Any PC arms, blue stars indicate the significance of the PC/LA arm, and green stars indicate the significance of the PC arm. These estimates are from versions of equation 1, estimated by round through LASSO, with covariates from footnote 9 and clustering standard errors by locality. Any PC pools the PC and PC/LA arms. Depression is measured concurrently from the PHQ-9 (PHQ-9≥10) up to 27 months since the baseline and then at 90 months. Depression prevalence in the shaded area (42 to 78 months after the baseline) is estimated from depression histories recalled at 90 months. For those time periods, PHQ-9 data are unavailable. The recalled depression years, 2020 to 2023, overlap with the COVID pandemic. 95% confidence intervals.

Figure B.3: Impact on seeking depression treatment (conditional on being depressed) - All Arms



Notes: We tested the hypothesis that care seeking differs from the control group's in each arm by estimating versions of equations 1 and 2, selecting covariates through LASSO from the list in footnote 9 and clustering standard errors by locality. The dependent variables are 4 indicators for how often people sought treatment when depressed between 2018 and 2023 (never, less than half the time, about half the time, and more than half the time). Any PC pools the PC and PC/LA arms. ***, **, and * represent statistically significant differences from the control group at the 1, 5, and 10% level.

Figure B.4: Impacts on Standardized Socioeconomic Outcomes and Pathways - All Arms



Notes: Standardized impacts for socioeconomic outcomes and pathways. Light (dark) bars indicate 95 (90) percent confidence intervals. Standard errors clustered by locality. Estimates from equations 1 and 2 using a post-double-selection LASSO estimator with covariates from footnote 9 and clustering standard errors by locality.

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