Using a quasi-experiment design, the economic impact of schistosomiasis was studied in 412 rice-grower households in Mali. Two groups of seven villages were formed, one a treated group and one an untreated group, after pair-matching by geographical zone and irrigation type. Effect of treatment was assessed according to economic output (paddy yield) and five resource variables (family and hired labour productivity, family and hired labour intensity and farm size). This study shows that changes in health have no direct effect on rice production, but affect the household's use of its labour resources and its ability to utilise other resources: increases of 69 man-days available per hectare (for family workers) and of 0.47 hectares in farm size were observed in the treated group relative to the untreated group. These results illustrate the key role of the coping process in masking the direct economic effects of disease. The benefit of reducing the burden of disease in rice production areas was confirmed through provision of additional utility to households by increasing the time available for leisure activities or for work.

1 This research was supported by the Fonds d'Aide et de Coopération, Caisse Française de Développement (France), GTZ (RFA) and European Fund for Development (EEC).

We would like to thank Dr Christian Werler and Dr Abdoulaye Diarra from the GTZ/Malian Schistosomiasis Control Program and the lab technicians from INRSP (National Institute for Public health Research) and ENMP/DEAP (Department of Parasitic Disease Epidemiology at the Medical School in Bamako). We would also thank Bino Teme, Didier Cebron and Moctar Traore from the Institut d’Economie Rurale and the team’s investigators for the economic survey. Finally, we are greatly indebted to the staff of the Office du Niger and particularly to the villagers of the survey zone. This paper benefited from comments of Eric Brenner, Joseph Brunet-Jailly and two anonymous referees.

1. Introduction

Although it is generally assumed that disease in a community will have a measurable and possibly dramatic negative economic impact, the assessment of this impact remains difficult. It would be desirable for policy-makers to have at their disposal methods to assist in decision making and in the assessment of the health effects of the policies they promulgate. Research into the elaboration of such instruments falls into three broad areas. The first one concerns measurement of the burden of disease. Those studies measure the effects of illness in reducing output (Foster, 1967; Gateff et al., 1971; Weisbrod, 1974; Barbosa and da Costa, 1981; Audibert, 1986) or productivity (Weisbrod and Helminiak, 1977; Workneh et al., 1993), in the reallocation of labour and land (Fenwick and Figenshou, 1972; Conly, 1975), or in reducing physical ability (Collins et al., 1976; Spurr et al., 1977; Brohult et al., 1981; Van Ee and Polderman, 1984). However, most of these studies have failed to show any economic effect. Reasons for this failure may be attributed to lack of data regarding morbidity (Guyatt et al., 1992; Bennett, 1993), the specific distribution of the infection within the population (Bundy and Guyatt, 1992; Hammer, 1993; Guyatt et al., 1994) or methodological problems resulting from the lack of innovation in statistical analysis (Rosenfield et al., 1984) indicating how the burden of morbidity may be measured (Evans and Murray, 1987; Over et al., 1992; World Bank, 1993; Robine et al., 1993).

The second research area has focused on the cost-effectiveness or cost-benefits of disease control efforts. In the case of analysis of cost-effectiveness, output may be quantified in terms of cases prevented, years of life saved, days of healthy life saved (Ghana Health Assessment Project Team, 1981; Barnum, 1987) and, more recently, quality-adjusted life-years (World Bank, 1993). One considerable difficulty arising from this approach is the great variability of the results obtained by different studies, as illustrated, for example, by studies concerning malaria and its control (Hammer, 1993).

The third area has worked with models, such as general equilibrium models (Barlow, 1987; Jones and O'Neil, 1993), studying the complex interrelations between disease, prices, labour productivity and land use, or ecology (Wiemer, 1988) in order to estimate the expected rate of productivity loss per person depending, for example, on different values for parameters concerning parasite distribution. However, these models remain theoretical; they are based on simulations, and
require empirical confirmation. Furthermore, they generally fail to take into account the coping process\textsuperscript{2} in which labour may be reallocated, thus masking any possible economic losses.

In fact the relationship between infection and production (or income) is weakened by the existence of several modifying factors at different levels of the process: disease $\rightarrow$ health status $\rightarrow$ functional capacity $\rightarrow$ productivity $\rightarrow$ income. Taking these links into account should assist assessment of the impact of disease on income (Over et al., 1992).

Several studies agree that the coping process and non-intra-family substitution of time must be taken into account when studying household production in order to understand the consequences of changes in health in the farm sector. Conly (1975), studying the effect of malaria on productivity, observed that the disease did not reduce farm production but increased family working time so as to cope with the reduction of labour productivity. Parker (1992), intending to gauge the impact of Schistosoma mansoni infection, found the same effect among female cotton pickers. Koopmanshap and Van Ineveld (1992), using the concept of friction cost, considered that real production losses may be much smaller than potential losses because sick people could frequently be replaced quickly at little or no cost.

In Indonesia, although the amount of labour available to the farmer is significantly decreased when the spouse becomes ill, no effect on farm profits was noticeable (Pitt and Rosenzweig, 1986). Hypothesising that the level of health may affect the productivity of farm inputs, and may have no direct effect on productivity, Pitt and Rosenzweig (1986) assessed the separability of production and health, and consequently the perfect substitution between hired and family labour. On the other hand, Grossman (1972) suggested a complementarity between health and work, and also between health and leisure. In fact, work reallocation may operate within the household. In Sudan, all the work hours lost in agriculture due to malaria, are made up by family members (Nur, 1983). In Colombia, non-wage-earner family members (rather than hired workers) compensate for the reduction in labour resulting from ill or injured workers (Bonilla and Rodriguez, 1993). Thus family members sacrifice their other activities (Conly,

\textsuperscript{2} The coping process is the process by which the household succeeds in compensating for the loss of family worker productivity due to ill-health.
1975) or their leisure (Castro and Mokate, 1988) in order to compensate for the decreased productivity of a family member who may be ill. In Africa, as noted by Mazur (1991), households dealing with opportunities and constraints are generally flexible and dynamic in allocating their resources such as land, capital and labour. Whilst each able household member is required to work on the extended household’s main plot, women and unmarried men may also cultivate small individual plots. However, the amount of land that households cultivate is also determined, in part, by their ability to hire external individual or group labour (Mazur, 1991).

Our efforts have been to develop another approach in estimating the costs of disease which would integrate economics with epidemiology, avoiding measures of morbidity, but taking into account the effects of the disease and the coping process by looking at economic responses to disease control efforts.

2. Quasi-experimental Design Model to Assess the Consequences of Changes in Health

The economic effects of health are felt in both the short and the long term. Long-term effects will not be considered since assessment requires an extended longitudinal study. In the short term, the level of health may affect production if compensation by family members is inadequate. A change in health may affect labour efficiency and productivity of farm inputs or the ability to utilise resources. The level of health might also directly affect the quality of the labour input.

Even if the main crop is considered to be the most important activity, readjustment of labour allocation may occur in less essential farm production activities. Because health affects family labour efficiency and productivity in fields devoted to main crops, every improvement in health provides direct additional utility to the household by increasing the time available for leisure or work in other activities. Whilst a reduction in family labour intensity (defined by the number of man-days of labour per hectare) may have no effect on hired labour, the additional time available to the family may be spent in increasing the cultivation of other crops.

The model uses a procedure, commonly used in evaluation research, in which the relationship between a primary factor (such as a treatment), a response variable (such as production or labour productivity, corresponding to disease status in epidemiology) and
intervening variables (or covariates) which may influence the factor-
response relationship (Kuritz et al., 1988) may be studied. The purpose
is to determine the effect of the treatment in comparison with a
situation in which no treatment is offered.

We use here a quasi-experimental design with two non-equivalent\(^3\) groups: experimental (treated) and control (untreated), with both
pretest and post-test measurements. Assuming that health status will
be modified as a result of the treatment, we then assess the change in
health by comparing factor responses (or post-test scores, \(Y\)) between
the two groups after treatment. As differences may occur at the outset
which give rise to post-test differences between the groups — even in
the absence of treatment — the analysis must check for the effects of
these initial differences and then justify the introduction of those
intervening variables in the pretest measurement\(^4\) (Cook and
Campbell, 1979). Analysis of covariance (ANCOVA), with one or
several covariates (pretest measurements), is then used to separate the
treatment effect from that resulting from selection differences.

The principle of this model can be illustrated graphically. Consider
a horizontal axis for pretest and a vertical axis for post-test scores. Each
point of the graph is jointly determined by individual pretest and
post-test scores. The individual results for each group can be plotted
as two scatterplots, one for each group, whose distributions can be
assumed to be normal. A regression line can be drawn through each of
the scatterplots. If the coefficients of the covariates were the same, the
regression lines would be parallel. This parallelism or homogeneity
across groups would mean that the likelihood of a treatment effect
occurring by covariate interaction where the effect of the treatment
varies according to the individual’s pretest results can be rejected. This
interaction may be tested by means of the partial F statistic for the
significance of the addition of an interaction term \(X_1X_2\) to a model
already containing \(X_1\) and \(X_2\). The treatment effect is represented as the
difference between the two intercepts (at the mean of the covariate
scores) where the regression lines intersect the vertical axis.

\(^3\) Nonequivalent groups are those for which the expected values of at least one
  group characteristic are not equal even in the absence of a treatment effect.

\(^4\) Covariates are introduced when initial differences between the two groups are
  observed against variables which could affect the response despite the treatment.
  In any case, they are introduced in such a way as to fit a comprehensive model of
  the response variable.
Four components determine the level of post-test response. The first is the grand mean of the post-test score across all households. The second is the treatment effect, which is the average value that the treatment adds to (or reduces) the post-test score in the treatment group. The third is the set of all covariates which provides an adjustment for initial differences between groups. The fourth is the error (Cook and Campbell, 1979).

The model is:

\[ Y_{ij} = \mu + \alpha_i + \sum_{k=1}^{K} \beta_k (X_{kij} - \bar{X}_k) + \varepsilon_{ij} \]

for \( j = 1, \ldots, N, i = e \text{ or } c \) (experimental or control), \( k = 1, \ldots, K \), and where \( Y_{ij} \) is the post-test score for the \( j \)th household in the \( i \)th treatment group, \( \mu \) is the grand mean, \( \alpha \) (technically \( \alpha_e - \alpha_c \)) is the treatment effect, \( \beta_k \) is the regression coefficients of the within-group linear regression of the post-test \( Y \) on the pretests \( X_{kij}, \bar{X}_k \) is the overall mean on the pretest and \( \varepsilon_{ij} \) is the error, assumed to be normal and independently distributed with mean zero and constant variance, \( \sigma_e^2 \).

In the absence of interaction, the estimate of the treatment effect is as follows:

\[ \hat{\alpha}_e - \hat{\alpha}_c = (\bar{Y}_e - \bar{Y}_c) - \sum_{k=1}^{K} \hat{\beta}_k (\bar{X}_{ke} - \bar{X}_{kc}) \]

where the expression on the right gives the effect of treatment without initial differences (first term) minus the effect due to initial differences (second term).

3. The Data and Estimation Procedure

3.1 Study Area

Field work was carried out in the Office du Niger (ON), an irrigated rice-growing site in Central Mali. Rice fields are partitioned in five geographical zones each comprising eight sectors defined on a technical basis (agricultural vulgarisation and water management). Irrigation is provided by two canals, the Sahel Canal for the northern fields and the Macina Canal for the eastern fields. The irrigation
scheme includes main, secondary, tertiary and drainage canals. Three sorts of plots (fully, semi and unlevelled) are present. The rice-growing area comprises 149 villages and 9604 households (ON staff census, June 1989). Paddy is the main crop, spread over an area of 47,000 hectares with approximately 2 hectares per active male. Whilst ploughing is done by bullock traction, most other tasks are accomplished using simple hand tools. Family manpower is the principal source of labour, even though ancillary non-family labour may also be used. Hired labour is only used on the cash crop fields. Fertilisers are used across all households depending on their knowledge. Paddy production is bought by the Office at a fixed price, the rice market not being liberalised at the time of the study. Sorghum is also cultivated, but is tending to become less and less important with the development of new paddy technology (transplanting instead of direct seeding). Market-garden products are grown, but only on an individual scale. In 1987, S. haematobium and S. mansoni infections\(^5\) were present and prevalence rates exceeded 60 and 50% respectively (Brinkmann et al., 1988).

### 3.2 Study Population

The sample population was drawn from a multi-disciplinary study, the two main objectives of which were (i) to estimate the costs of paddy production and (ii) to assess the economic impact of schistosomiasis. A sample of 30 villages was drawn from all ON villages. It was made up of 16 non-randomly and 14 randomly selected villages. The first 16 were part of an initial study (into paddy costs) carried out by the Institut d'Économie Rurale (Mali). In the first year of this study, the sample contained six villages from one area in which all three types of plots were represented. The sample was subsequently extended to a second area containing 10 villages. A further 14 villages were added when looking at the second objective: these were selected in order to give a sample equivalent to approximately 13% of the resident population.

\(^5\) Schistosomiasis is a disease due to a parasite of the genus *Schistosoma*. Two of the four species which affect humans are present in the studied area. The first results in urinogenital manifestations of the disease, while the second leads to an intestinal form which can give rise to liver disease. Infection may occur as a result of contact with infected water during domestic (washing clothes), recreational (swimming, bathing) or economic (fishing) activities in infected canals or drains.
households of each sector. Thirty households were then selected randomly with an equal probability in each of the 30 villages, using an enumeration list excluding non-resident households.

2.3 Study Design and Data Collection

Two groups, $G_e$ and $G_c$, were formed after matching the villages on the basis of type of irrigation schemes and geographical zone, and after excluding all villages where the National Schistosomiasis Control Program had already been implemented. After this selection process, only 14 of the 30 villages initially included in the sample remained. However, this sub-group included 412 households, corresponding to about 4.5% of the ON households which collectively represented all the production patterns (in respect of environment and farming practices). Each of the two groups therefore contained seven villages.

One investigator was assigned to each village during two consecutive agricultural seasons (1989 and 1990) with responsibility for collecting economic data from June to December. Information relating to households' demographic characteristics, farm size, bullock labour and equipment, and harvested production was obtained from farmers by means of questionnaires administered daily. Labour time (number of hours spent in the rice fields on productive activities) for each family and non-family labourer was observed daily in the fields by the investigator. The investigators did not know their village group membership.

Two parasitological surveys were organised, one in December 1989, at the end of the first agricultural season, and the other in December 1990, after the end of the second season. Parasite egg counts were performed on single urine specimens (filtration method) and single stool specimens (Kato technique) at each survey. In December 1989, a mass treatment was offered to the experimental group, $G_e$, while subjects of $G_c$ received a placebo (vitamin D). In December 1990, a mass treatment was offered to the control, $G_c$, while infected $G_e$ subjects were selectively treated. Treatment was administered using a single oral dose of 40 mg/kg praziquantel under the supervision of the investigators. This protocol was approved by the National Schistosomiasis Control Program.

3.4 Internal Validity, Homogeneity, Adjustment and Estimation Procedures

Two major threats to internal validity pertaining to the non-equivalent
control group designs [selection-maturation\(^6\) and local history (Cook and Campbell, 1979)] were controlled for. A selection-maturation process was avoided by matching villages on the basis of geographical and environmental conditions and farming practices, and by not allowing the self-selection of individuals with respect to treatment. Plotting pretest scores against date of settlement and householder's age for each group showed no impact (on productivity) or difference in regression slopes (farm size), suggesting that a differential maturation process, due to past experience, was not present. No other external effects, such as new levelling of fields, occurred during the time of the study.

The relevance of the chosen pretests (covariates) was verified for each model using a significant non-parametric test (Mann-Whitney and Kolomogorov-Smirnov) or t-test at the 15% level. The Hausman test in the Stata package was used to ascertain that there were no significant differences in regression coefficients between fixed and random effects. Finally, an ANCOVA was performed using BMDP 1V and 2V procedures.

As the rates of non-participation in treatment were low (Table 1), no selection bias could be imputed to non-participation.

**4. Econometric Models**

Hypotheses for direct and indirect consequences of changes in health were tested by applying six models. The focus was then to determine the effect of a treatment for schistosomiasis on farm output, on productivity of inputs and on household ability to use resources (response variables). The models were the following:

Model I, where \( Y_x \) is the post-test score of paddy yield, tests the hypothesis that a change in health would affect farm output. The hypothesis that the productivity of farm inputs may be affected is investigated in Model II, where \( Y_x \) is the family labour productivity in the rice field. Model III, hired\(^7\) labour productivity, checks that there

\(^6\) Selection-maturation occurs when the treatment group outperforms the control group or, more generally, when the two groups grow in a common direction but at different rates because one group is more experienced, brighter or intrinsically more capable of change. In this case, the observed change has nothing to do with the effects of the treatment.

\(^7\) Permanent and daily wage-earners included.
are no post-test differences in hired labour productivity between the two groups. The hypothesis that improving health may also modify the intensity of labour in rice fields and thus lead to a more efficient use of time is investigated in Model IV, where $Y_z$ is the family labour intensity. Model VI, where $Y_z$ is the intensity of labour for hired workers, is used in order to check that there are no post-test differences in hired labour intensity between the two groups. The effect of health on household ability to utilise resources is studied in Model VI, where $Y_z$ is the farm size (defined as the total cultivated acreage).

Logarithmic transformation of the variables is used to normalise the distribution of the data as well as to reduce the heterogeneity of the variances.

### 4.1 Checking Potential Bias

Four hundred and twelve households were enrolled the first year (Table 1). Three households left the area before the second rice season. In addition, economic data for three households were missing when applying Model I, and for four households when applying Model IV. Participation rates for the 1989 mass treatment in $G_e$ was high (Table 1). Table 2 shows that the two groups did not differ at the time of the baseline survey with respect to prevalence and density of *S. haematobium* or *S. mansoni* infection. The effect of treatment in the experimental group was demonstrated by the decrease in prevalences and densities observed during the second survey. Table 3 displays descriptive statistics of the variables used in the analysis.

#### Table 1: Population Study and Participation Rate for Mass Treatment by Group

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of Households</th>
<th>Number of Individuals</th>
<th>Participation Rate (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Active 1</td>
<td>Dependent 2</td>
</tr>
<tr>
<td>$G_e$ 3</td>
<td>206</td>
<td>1689</td>
<td>1004</td>
</tr>
<tr>
<td>$G_c$ 3</td>
<td>206</td>
<td>1704</td>
<td>852</td>
</tr>
</tbody>
</table>

1Active = 10–55 years.

2Dependent = 0–9 and >55 years.

3$G_e$ = experimental group, $G_c$ = control group.
Impact of Schistosomiasis in Mali 195

Table 2: Prevalence Rates and Density of Schistosomiasis Infections in the Two Groups in 1989 and 1990

<table>
<thead>
<tr>
<th>Infection</th>
<th>Group</th>
<th>Year</th>
<th>Number Examined</th>
<th>Prevalence (%)</th>
<th>GMD&lt;sup&gt;1&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>S. haematobium</td>
<td>Ge</td>
<td>1989</td>
<td>1825</td>
<td>44.6</td>
<td>3.97</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1990</td>
<td>1620</td>
<td>22.7</td>
<td>1.62</td>
</tr>
<tr>
<td></td>
<td>Gc</td>
<td>1989</td>
<td>1734</td>
<td>41.2</td>
<td>3.33</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1990</td>
<td>1402</td>
<td>40.9</td>
<td>3.12</td>
</tr>
<tr>
<td>S. mansoni</td>
<td>Ge</td>
<td>1989</td>
<td>1326</td>
<td>52.5</td>
<td>12.3</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1990</td>
<td>942</td>
<td>24.7</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Gc</td>
<td>1989</td>
<td>1246</td>
<td>52.4</td>
<td>13.4</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1990</td>
<td>757</td>
<td>42.6</td>
<td>7.4</td>
</tr>
</tbody>
</table>

<sup>1</sup>Geometric mean density (infected and non-infected): S. haematobium = number of eggs/10 ml of urine; S. mansoni = number of eggs/g of stools.

<sup>2</sup>Ge = experimental group, Gc = control group.

4.2 Checking Basic Assumptions of the ANCOVA

The hypothesis that the covariates (fixed effects) and the error are independent of one another is proven in all the models except Model II. The ANCOVA analysis cannot be used and thus the hypothesis that family labour productivity may be affected by the treatment cannot be tested. The assumption that the slopes are equal holds true for all five remaining models (F-tests not significant at 5% level, Table 4). The ANCOVA analysis may therefore be used to assess the economic consequences of health.

The effect of the covariates is confirmed (β<sub>k</sub> is significant at the 1% level) except for Model I (paddy yield), where the coefficients of the covariates are not significant, therefore the null hypothesis of zero slope for these covariates cannot be rejected. For this model, the ANOVA analysis was used, and the results compared to ANCOVA analysis.

4.3 Effect of Treatment on Farm Output

The hypothesis that a change in health has a direct effect on main cash
Table 3: Definition, Mean and Standard Deviations of the Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Experimental Group (G_e)</th>
<th>Control Group (G_c)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N</td>
<td>Mean</td>
</tr>
<tr>
<td>Paddy yield (production in kg/ha)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Yield_0 at pretest</td>
<td>206</td>
<td>7.421</td>
</tr>
<tr>
<td>Yield_1 at post-test</td>
<td>203</td>
<td>7.683</td>
</tr>
<tr>
<td>Family labour intensity (man-days worked/ha)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fam_0 at pretest</td>
<td>206</td>
<td>5.850</td>
</tr>
<tr>
<td>Fam_1 at post-test</td>
<td>203</td>
<td>5.439</td>
</tr>
<tr>
<td>Hired labour intensity (man-days worked/ha)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hire_0 at pretest</td>
<td>200</td>
<td>6.35</td>
</tr>
<tr>
<td>Hire_1 at post-test</td>
<td>204</td>
<td>5.74</td>
</tr>
<tr>
<td>Family labour productivity (paddy production/man-days worked)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fprod_0 at pretest</td>
<td>206</td>
<td>1.58</td>
</tr>
<tr>
<td>Fprod_1 at post-test</td>
<td>203</td>
<td>2.25</td>
</tr>
<tr>
<td>Hired labour productivity (paddy production/man-days worked)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hprod_0 at pretest</td>
<td>193</td>
<td>2.57</td>
</tr>
<tr>
<td>Hprod_1 at post-test</td>
<td>193</td>
<td>2.55</td>
</tr>
<tr>
<td>Farm size (cultivated total acreage/ha)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Size_0 at pretest</td>
<td>206</td>
<td>1.743</td>
</tr>
<tr>
<td>Size_1 at post-test</td>
<td>205</td>
<td>1.774</td>
</tr>
<tr>
<td>Hired labour force (no. of permanent wage-earners in the household)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hwork</td>
<td>206</td>
<td>-1.717</td>
</tr>
<tr>
<td>Sorghum production (kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prod_m</td>
<td>206</td>
<td>2.049</td>
</tr>
<tr>
<td>Sorghum acreage (ha)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Acrem</td>
<td>20</td>
<td>-1.115</td>
</tr>
<tr>
<td>Phosphate (quantity of phosphate spread, kg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phos</td>
<td>206</td>
<td>4.875</td>
</tr>
</tbody>
</table>

1All variables are in natural logarithm.

crop production is rejected, as shown by the corresponding mean post-test scores in the two groups (the t-test is not significant, Table 5). It may be that the effect of the coping process is at work here, giving rise to the following question: can it be that no change in productivity could be observed because other family members adequately com-
Table 4: Regression Coefficients of Covariates, Tests for Equality of Slopes and the Hausman Test

<table>
<thead>
<tr>
<th>Model</th>
<th>Regression Coefficients</th>
<th>F-test for Equality of Slopes</th>
<th>Hausman Test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Coefficient</td>
<td>$\beta$</td>
<td>SE</td>
</tr>
<tr>
<td>Model I: paddy yield</td>
<td>Prodm</td>
<td>0.000</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Acrem</td>
<td>-0.025</td>
<td>0.021</td>
</tr>
<tr>
<td></td>
<td>All covariates</td>
<td>0.76n.s.</td>
<td>1.61</td>
</tr>
<tr>
<td>Model II: family labour productivity</td>
<td>Fprod0</td>
<td></td>
<td>7.41</td>
</tr>
<tr>
<td></td>
<td>Hwork</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>All covariates</td>
<td>7.41</td>
<td>0.006**</td>
</tr>
<tr>
<td>Model III: hired labour productivity</td>
<td>Prodm</td>
<td>-0.02</td>
<td>0.01</td>
</tr>
<tr>
<td></td>
<td>Acrem</td>
<td>0.12</td>
<td>0.03**</td>
</tr>
<tr>
<td></td>
<td>All covariates</td>
<td>1.31n.s.</td>
<td>2.98</td>
</tr>
<tr>
<td>Model IV: family labour intensity</td>
<td>Fam0</td>
<td>0.83</td>
<td>0.07**</td>
</tr>
<tr>
<td></td>
<td>Hwork</td>
<td>-0.08</td>
<td>0.02*</td>
</tr>
<tr>
<td></td>
<td>All covariates</td>
<td>0.80n.s.</td>
<td>0.02</td>
</tr>
<tr>
<td>Model V: hired labour intensity</td>
<td>Prodm</td>
<td>0.017</td>
<td>0.015</td>
</tr>
<tr>
<td></td>
<td>Acrem</td>
<td>-0.16</td>
<td>0.029**</td>
</tr>
<tr>
<td></td>
<td>All covariates</td>
<td>2.1n.s.</td>
<td>2.26</td>
</tr>
<tr>
<td>Model VI: total farm size</td>
<td>Siz0</td>
<td>0.92</td>
<td>0.03**</td>
</tr>
<tr>
<td></td>
<td>Hwork</td>
<td>0.03</td>
<td>0.01*</td>
</tr>
<tr>
<td></td>
<td>Prodm</td>
<td>0.02</td>
<td>0.005**</td>
</tr>
<tr>
<td></td>
<td>Acrem</td>
<td>-0.06</td>
<td>0.01**</td>
</tr>
<tr>
<td></td>
<td>Phos</td>
<td>0.03</td>
<td>0.009**</td>
</tr>
<tr>
<td></td>
<td>All covariates</td>
<td>1.88n.s.</td>
<td>0.0</td>
</tr>
</tbody>
</table>

*Significant at the 1% level; **significant at the 0.1% level; n.s. = not significant.
Table 5: Adjusted Means (m) and Treatment Effect (a)\(^1\)

<table>
<thead>
<tr>
<th>Model</th>
<th>Experimental Group m</th>
<th>Control Group m</th>
<th>t-test</th>
<th>α(^2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>I Paddy yield</td>
<td>2271 (2091–2446)</td>
<td>2175 (1989–2355)</td>
<td>-0.72</td>
<td>n.s.</td>
</tr>
<tr>
<td>III Hired labour productivity</td>
<td>12.64 (11.27–14.26)</td>
<td>11.21 (9.89–12.52)</td>
<td>-1.4n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>IV Family labour intensity</td>
<td>249.86 (228.56–272.65)</td>
<td>318.90 (290.56–347.99)</td>
<td>3.77*</td>
<td>-69.0</td>
</tr>
<tr>
<td>V Hired labour intensity</td>
<td>181 (160.99–203.69)</td>
<td>192 (170.79–216.92)</td>
<td>0.72n.s.</td>
<td>n.s.</td>
</tr>
<tr>
<td>VI Farm size</td>
<td>5.72 (5.61–6.07)</td>
<td>5.26 (5.06–5.47)</td>
<td>-2.9*</td>
<td>+0.47</td>
</tr>
</tbody>
</table>

\(^1\)Adjusted means and treatment effect are given in original units (man-days by hectare for labour intensity, and hectares for farm size).
\(^2\)α = m_e – m_c.

*pSignificant at the 0.1% level.

Compensated for the loss of work time through illness? In other words, is the reduction in efficiency due to disabled family workers compensated for by family members, who then work more? Let us turn to the family labour intensity.

4.4 Effect of Treatment on Family Labour Intensity

Improvements in health significantly reduce family labour intensity in rice-growing, but have no effect on non-family labour intensity in the same activity. The difference between the post-test scores (adjusted means after treatment) of the family labour intensity in the two groups is significant (Model IV, \(t_{[401]} = 3.77\), Table 5). The adjusted mean of family labour intensity is lower in the experimental group than in the control group (P < 0.0001). The observed reduction of family labour intensity in rice fields is not followed by a reduction in hired labour intensity: no difference in post-test scores can be observed between the two groups (Model V, \(t_{[382]} = 0.72\) is not significant). At the same time,
no effect on hired labour productivity can be observed (Model III, $t_{378} = -1.4$ is not significant).

These results, i.e., no change in production but a decrease in family labour intensity while no changes in hired labour (productivity and intensity) are observed, suggest that family members made up for the work lost due to illness. The reduction in efficiency due to disabled family workers (and not that of non-family workers) is compensated for by family members who then work more: an improvement in health provides an increase of time available of 69 man-days for family workers; no such changes are observed for non-family workers. Thus, family labour intensity gains in the experimental group result in more time becoming available.

4.5 Effect of Treatment on Ability to Use Resources

Our findings confirm the suggestion that treatment has an effect on farm size and the allocation of some available time to crops other than rice. The adjusted mean of the post-test farm size score is higher in the experimental group than in the control with $P < 0.001$ (Model VI), resulting in an additional of 0.47 hectares in total in the former group at the post-test level.

Two important results have been identified: treatment reduces family labour intensity in rice fields without modifying hired labour intensity and hired labour productivity, and treatment increases the total cultivated area. This suggests that a portion of the gain in family labour productivity (indirectly measured by family labour intensity) is invested in extending other crops; the rest may be invested in leisure or non-agricultural or social activities.

5. Discussion

Measuring the economic effects of disease has not been always obvious. In the case of schistosomiasis, earlier works often failed to show a loss of production or labour productivity. Gateff et al. (1971) found no differences in weekly earnings between infected and non-infected cane-cutters. Weisbrod and Helminiak (1977), using the same approach but in banana fields, also failed to find differences in weekly earnings between infected and non-infected workers. Therefore, Weisbrod and Helminiak (1977), in assuming that disease would affect not only weekly earnings, but also labour productivity and then showing that infected workers worked more (in terms of number of
days worked per week) than non-infected workers, seem to have indicated, even if this was not their interpretation, the way in which sick workers might adjust to disability. Conly (1975) gave evidence of this adjustment when she showed that malaria-infected workers had to work longer than those not infected in order to receive the same weekly earnings.

In fact, studies which seek to demonstrate and measure the economic effects of disease are confronted with three difficulties: the individual economic behaviour patterns of wage-earner workers in plantations, the need for innovation in statistical methodologies and analysis (Guyatt and Evans, 1992), and the way in which households organise themselves in order to cope.

Assessment of the economic effect of disease among wage-earners in plantations suffers from a double bias. The first is what is called in epidemiological studies 'the healthy worker effect', a selection bias arising when workers are in better health than non-workers. The second is linked with their economic behaviour. Workers in plantations have two strategies. One, which concerns temporary or daily workers, is to get enough money for discrete but pressing needs (Weber, 1978). The second, which concerns permanent but seasonal workers, is to gain a secondary, complementary income as they generally also work, during the rainy season, in their own fields. As earnings in plantations are guaranteed and known (in contrast to those from family farms), workers may work with a greater intensity than they would do if they were sick in order to fulfil their strategies.

The use of the ANCOVA technique, already applied in economic research (Kalirajan and Shand, 1985; Shaver and Reimer, 1991; Holden and Coppock, 1992) but not previously in the analysis of the economic consequences of disease, may be seen as a useful methodological innovation. More than the traditional approach which uses the concept of work-days or earnings lost to assess economic impact of disease, our approach, using a quasi-experimental design, might enhance the study process which focuses not only on economic output (as yields or earnings) but also on input productivity. Since schistosomiasis is a chronic disease which has been present in this area for a long time, farmers have had to adjust their economic behaviour to account for such a chronic disability and treatment against schistosomiasis should improve their well-being. In our study, we wanted to place more emphasis on improvement in health than has been the case in earlier
works, and to benefit from a new opportunity to establish a link between disease and production or productivity.

This study shows that change of health (following treatment) has an indirect effect on farm output in terms of both labour and land. Our findings support the concept of a coping process in which reallocation of tasks occurs within the household rather than outside it already observed in earlier works (Conly, 1975; Castro and Mokate, 1988; Bonilla and Rodriguez, 1993; Nur, 1993; Mills, 1994a), and that of a reallocation of time between activities, suggested by Becker (1975). Whilst treatment did not afford any change in production by improving the well-being of family workers, it did affect family labour intensity in rice fields. Family workers became more efficient, but they did not use their new-found efficiency to save on hired labour. Where additional non-family labour was employed, hired labour was used in rice fields rather than in other crop fields (hired labour intensity and productivity in rice fields had not been modified after treatment and was the same in the two groups). Further, reduction of family labour intensity, instead of reducing non-family labour intensity in rice fields, provided additional utility to the household in releasing free time to family workers. Our findings show that this free time was used to expand total farm acreage with traditional non-rice crops such as sorghum. This response has several explanations. At the time this study was conducted, farmers did not have the option of expanding rice acreage (attributions being made directly by the Office) or of changing their cultivation technique, so the benefits of the labour intensity reduction could not be reinvested here. Sorghum remains a well-liked food, and farmers would like to increase its production (by increasing acreage). However, the lack of information on market-gardening acreage does not permit verification of this assumption. As long as farmers cannot influence the market price of rice [and it is not sure that the liberalisation of the rice trade will allow them to do so as farming households are not organised in cooperative associations (Harre and Oyep, 1992)], market gardening seems more profitable: farmers can speculate in selling onions several months after the harvest. Had we the opportunity of assessing market-gardening acreage, the impact of the economic effects might have been higher. Thus, market-gardening will be an activity sacrificed early following the reallocation of tasks within the household to cope with reduction of labour productivity.

The cost of schistosomiasis in the ON context may be better assessed
in terms of household welfare and individual incomes than in terms of household rice income, as other non-rice activities are reduced. With the anticipated future expansion and intensification of rice-growing (wider adoption of transplanting and of double-harvesting) at the ON, it will become more difficult for family workers to cope with the labour productivity reduction implications of schistosomiasis, and the disease may have a more noticeable effect on rice production.

The economic impact of schistosomiasis might be underestimated since long-term effects have not been examined: disabled households may be less willing to invest in resources than healthy households. We know from unpublished data (Coulibaly et al., 1980) that capital goods, such as bullocks and ploughs, are determinants of ability to adhere to the rice-farming calendar, and thus of rice output. But assessing the effect of treatment on investment would require the type of quasi-experimental design described above to be carried out over several years and this would be not only very expensive but also unethical.

Our findings have two implications. The first may be useful to control programmes and in resource-allocation decisions. If gains in time due to treatment, valued at $69 per hectare (with a daily wage of one dollar), are higher than the cost of treatment (also valued per hectare), then such treatment of the disease may be economically justified. There remains a need to assess the treatment plans and the different components of their costs. This economic gain should also be considered as one of the output components (indirect economic benefit) of a cost-effectiveness, or cost-benefit, analysis. The second implication is epidemiological since the resulting gains in time form part of the measurement of the disease burden (Mills, 1994b; Murray, 1994) and should be considered as an indirect measure of the level of adult morbidity due to schistosomiasis which is still under debate (Tanner, 1989).

References


Cook, T. and D.T. Campbell (1979). *Quasi-experimentation, Design*


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