

Research Article

Pesticides and Health in Vegetable Production in Kenya

Ibrahim Macharia

Department of Agribusiness Management and Trade, Kenyatta University, P.O. Box 43844–00100, Nairobi, Kenya

Correspondence should be addressed to Ibrahim Macharia; macharia.ibrahim@ku.ac.ke

Received 10 September 2015; Accepted 16 November 2015

Academic Editor: Fernando Barbosa Junior

Copyright © 2015 Ibrahim Macharia. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

This paper investigates the determinants of pesticide-related cost of illness (COI) and acute symptoms, using a balanced panel of 363 farmers interviewed from seven major vegetable producing districts of Kenya. Finding shows that the incidences of pesticide-related health impairments have increased. Variation in number of symptoms and symptom severity significantly explained COI. The personal protective equipment (PPE), education level, record keeping, and geographical location considerably determined health impairments. Encouraging the proper use of PPE and record keeping of pesticide use could greatly reduce poisoning cases and COI.

1. Introduction

The health effects of pesticide use have become one of the major public health problems worldwide. In developing countries, frequent exposure to pesticides by farmers and farm workers is very common [1–3]. The frequent exposures to pesticides result in both short-term (acute) and long-term (chronic) illnesses. Scientifically confirmed pesticide-related acute illnesses include headaches, stomach pains, vomiting, skin rashes, respiratory problems, eye irritations, sneezing, seizures, and coma [4]. The chronic illnesses include cancer, asthma, dermatitis, endocrine disruption, reproductive dysfunctions, immunotoxicity, neurobehavioral disorders, and birth defects [5–13]. Furthermore, deaths resulting from direct exposure to pesticides are also common [14].

The World Health Organization (WHO) and the United Nations Environment Program (UNEP) estimate pesticide poisoning rates at 2–3 per minute [14]. The largest numbers of pesticide poisonings and deaths are said to occur in developing countries [15]. It has been argued that pesticide-related health issues constitute a serious threat to development and can easily reverse or undermine the gains made in agricultural growth [16]. Poor access to health services and the inability of medical professionals to recognize pesticide-related morbidity raise further concerns [17]. Although pesticide-related poisoning is still not as high or more pronounced in Africa as in Asia, it is a growing problem as the increasing intensification of agricultural production

with more widespread use of pesticides will result in possible increase in pesticide poisoning [18].

In Kenya, pesticide use and farmers health have been documented by some empirical studies [19–21]. However, these studies were based on a snap shot of cross-sectional surveys and a clear trend of poisoning is not well understood. In addition, only two studies looked at the determinants of pesticide-related acute poisoning symptoms among farmers [19, 20]. However, the problem is that pesticide poisoning effects on human are not random but rather depend on other unobserved characteristics such as genetic characteristic. Such effects cannot be captured with cross-sectional data as utilized in the above studies. Thus the true underlying causal relations may be very different, either larger or smaller, compared with those noted in those researches.

The objective of this paper therefore is to examine the incidences and the determinants of acute pesticide poisoning among vegetable farmers in Kenya controlling for unobserved heterogeneity.

2. Methods

2.1. Surveys and Data. The study was conducted in seven major vegetable producing districts of Central Province (Kiambu, Kirinyaga, Murang'a, Nyandarua, and Nyeri North) and Eastern Province (Makueni and Meru Central) of Kenya (Figure 1) in the year 2005 with follow-up visits in 2008.

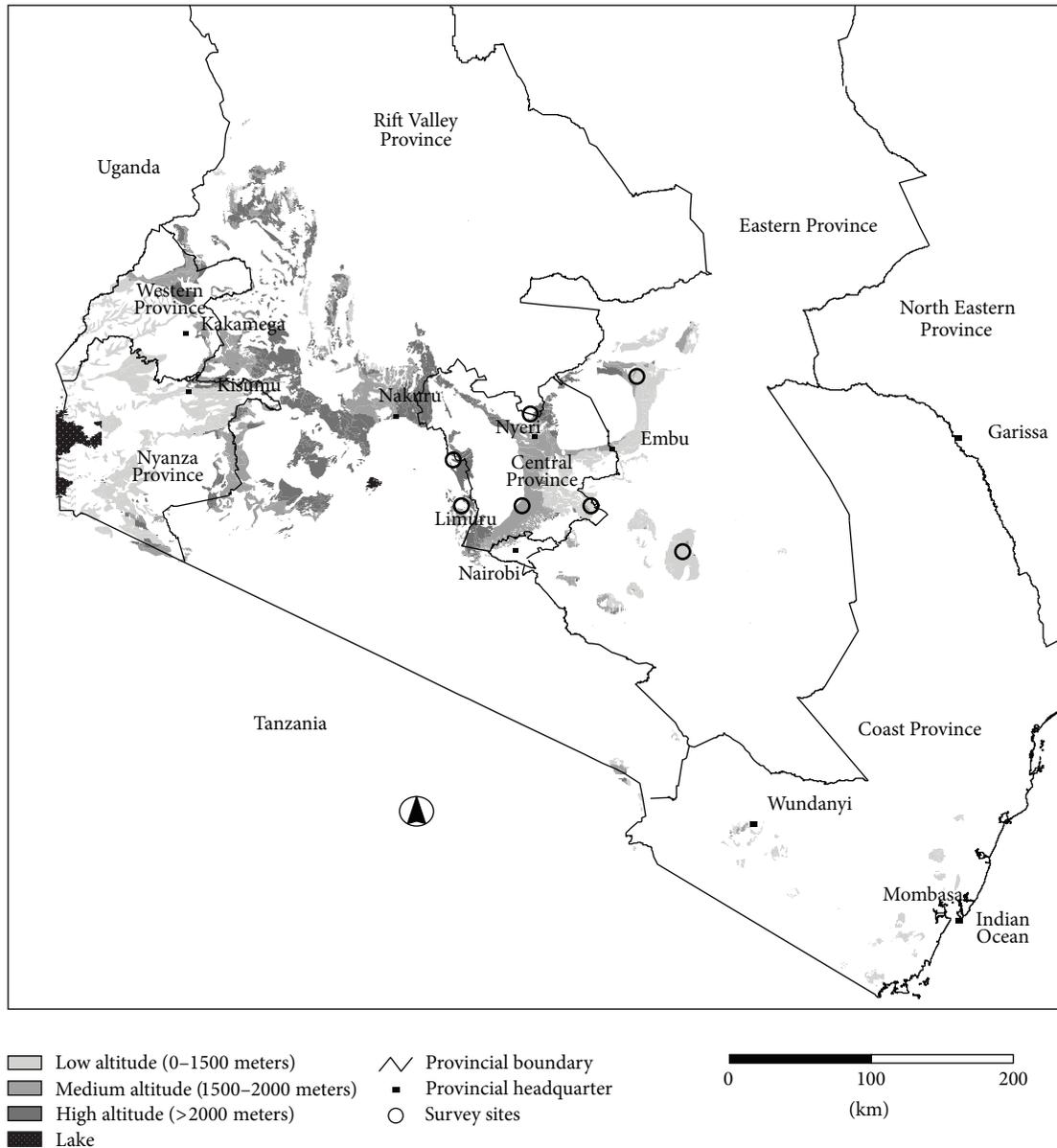


FIGURE 1: Study sites. Source: this study based on GIS mapping of potential vegetable production areas.

The 2005 survey comprised 839 interviews from the Diamondback moth biological control impact assessment survey (“DBM” with 295 farmers) and the Global Good Agricultural Practices (“GLOBALGAP” with 544 farmers) assessment survey. GLOBALGAP (formerly known as EUREPGAP) is a private sector body that sets voluntary standards for the certification of agricultural products around the globe. In both surveys, a multistage sampling procedure was used to select districts, sublocations, and farmers, respectively. First, districts were purposely sampled according to intensity of vegetable production and agroecological zones. Lists of farmers that were compiled by extension workers at sublocation level served as sampling frame from which 839 farmers were randomly sampled by probability proportional to size (PPS) procedure.

Sampled farmers were then monitored in one cropping season and were trained in record keeping of their production activities by trained enumerators. The trained enumerators under direct supervision of the researcher visited the farmers to check the records and transferred the information to the survey questionnaire.

Due to budget constraints, the 2008 survey was a recall survey of a random subsample of 425 farmers among the 839 farmers. However, we only obtain 363 balanced data set after the 2 years of study. Table 1 displays the distribution of farmers in the sampled districts.

The semistructured questionnaires employed covered a wide range of topics, such as cropping systems, demographics, common farming practices, pesticide use and handling practices, and type and quantities of pesticides sprayed.

TABLE I: Regional distribution of survey respondents.

| Province | District | Main vegetable crops | | Previous surveys (2005) | Number of farmers sampled (2008) | Balanced data (farmers) |
|----------|--------------|-------------------------------------|-------------------------------|-------------------------|----------------------------------|-------------------------|
| | | Domestic | Export | | | |
| Central | Kiambu | Cabbages, kales, and spinach | | 48 | 27 | 19 |
| | Kirinyaga | Peas, tomatoes | French beans | 155 | 74 | 66 |
| | Muranga | Tomatoes, kales | French beans | 51 | 24 | 21 |
| | Nyandarua | Cabbages, potatoes | | 119 | 52 | 49 |
| | Nyeri North | Peas, cabbage, onions, and carrots | French beans | 277 | 116 | 107 |
| Eastern | Makueni | Cabbages, kales | Asian vegetables ^a | 49 | 22 | 8 |
| | Meru Central | Peas, tomatoes, cabbage, and onions | French beans | 140 | 110 | 93 |

^aBrinjals, karella, dudhi, okra, turia, valore, and aubergine. Source: this study.

Health symptoms investigated were specified as those that only began during the spraying operation or within 24 hours after spraying. Additional information collected included the following: number of times the symptom occurred, workdays lost partially or completely due to the health impairment, medication taken by victims, and direct costs due to the symptoms, that is, pharmacy cost, consulting fees, and indirect costs such as travel expenses to and from health centre and dietary expenses resulting from illness like drinking milk or taking honey.

2.2. Analytical Framework. As discussed earlier panel data setup was used to control for the unobserved heterogeneity. In general, panel data model offers some distinct advantages over the cross-sectional data analysis. Greene [22] concluded that the main advantage of panel data is that one can formally model the heterogeneity across groups that are typically present in panel data. Baltagi [23] confirms this in his statement that the first benefit of panel data is “controlling for individual heterogeneity.” Additional benefits of using panel data are the ability to capture both cross section and time-series variation in the dependent variable and measure not only the effects that observable variables have on the dependent variable, but also the effects of relevant unobservable or nonmeasurable influences.

A general panel regression model is presented as

$$\begin{aligned}
 y_{it} &= \alpha_0 + \beta X_{it} + \gamma Z_i + V_{it}, \\
 V_{it} &= \varepsilon_i + \mu_{it},
 \end{aligned}
 \tag{1}$$

where y_{it} is the response of the dependent variable (in our case this is the cost of illness (COI) or number of acute symptoms) for the i th farmer in the sample at the t th year. α_0 is an intercept that may be different for each point in time, and β and γ are vectors of coefficients. X_{it} is the set of K -vector of time-variant covariates for the i th farmer at the t th year and Z_i is another set of predictor variables that do not vary over time (time-invariant), for example, gender

and location. V_{it} is the error term, which is decomposed into ε_i and μ_{it} . ε_i is regarded as the combined effect on y of all unobserved variables that are constant over time (time constant unobserved heterogeneity such as cognitive ability and motivation), and μ_{it} represents the idiosyncratic error term (what is unaccounted for in the model) and varies over individual farmers and over time.

The two main methods of dealing with ε_i are to make the random effects (RE) or fixed effects (FE) assumption. Random effects assumed that ε_i are random variables that is, ε_i is i.i.d. $(0, \sigma_\varepsilon^2)$ and that $\text{Cov}(x_{it}, \varepsilon_i) = 0$, while with fixed effects, ε_i are assumed to be potentially correlated with X_{it} . In fixed effect regressions we cannot estimate the effects of time constant covariates as these are normally cancelled out by the within transformation. Thus, classic fixed effects approaches do not produce any estimates of the effects of variables that do not change over time. Moreover, in some cases fixed effects estimates may have substantially larger standard errors than random effects estimates, leading to higher p values and wider confidence intervals. In addition, fixed effects estimates use only within-individual differences, essentially discarding any information about differences between individuals unlike random effects that used information both within and between individuals. Thus, if predictor variables vary greatly across individuals but have little variation over time for each individual, then fixed effects estimates will be rather imprecise. When neither the cross-sectional unit nor times have significant effects, all of the data can be pooled and one can have the constant coefficients model.

The analysis was implemented in two steps. First, the COI model was estimated to evaluate the determinants of health costs among the vegetable farmers. Cost of illness was computed as the sum of farmer-reported medical treatment costs to clinics and private physicians, the opportunity cost of workdays lost to illness, travel costs to and from health facility, time spent in traveling, and the cost of home-based health care. In the second stage, the principal factors associated with the pesticide poisoning symptoms were examined seeking ones that are relevant at policy recommendation.

2.3. The Models

2.3.1. Cost of Illness Model. In previous studies, the health costs of pesticides were modeled using a Logarithmic regression model [19]. In this study the estimation of the determinants of health costs was modeled using a censored random effects Tobit model (Xttobit), since zero costs from respondents who had suffered pesticide-related illnesses but incurred no costs were considered. Using a Logarithmic model would have required adding a small unity value as log of zero is undefined. Estimation of dependent variables results in biased estimators in linear models [24]. The structural equation in the Tobit model is represented as

$$y_{it}^* = x_{it}\beta + \varepsilon_i + u_{it}, \quad (2)$$

where $\varepsilon_i \sim N(0, \sigma_\mu^2)$ and y^* is a latent variable that is observed for values greater than T and censored otherwise. The observed y is defined by the following measurement equation:

$$y_{it} = \begin{cases} y^* & \text{if } y^* > T \\ T_y & \text{if } y^* \leq T. \end{cases} \quad (3)$$

In the typical Tobit model, we assume that $T = 0$; that is, the data are censored at 0.

For the empirical model, the explanatory factors for the model explaining health costs incorporate four broad classes of variables, namely, those related to health (number of acute symptoms and symptoms severity), farmer characteristics variables (age, education, and gender), farm management variables (farm size (proxy for wealth), GLOBALGAP certification, and record keeping), and location control (district dummies) (see (4)). Variable definitions and descriptive statistics are presented in Table 2.

It is hypothesized that the number of acute symptoms, symptom severity, age, and farm size are positively associated with the health costs, while a negative association is expected for level of education, GLOBALGAP certification, and record keeping. The direction of the effect of gender on health costs is not clear a priori.

It is anticipated that young farmers may have a higher tendency to protect against pesticides exposure and consequently reduce the pesticide-related acute symptoms and associated health costs. Increased education is also expected to reduce health costs because farmers are more likely to read pesticide labels and follow the recommendation, again reducing the exposure and the acute symptoms. Likewise, GLOBALGAP certification and record keeping can result in a more judicious use of pesticide and higher tendency to protect against pesticide intoxication resulting in reduced acute symptoms:

$$\begin{aligned} \text{HEALTHCOST} = f(\text{TACUTE, SEVERE, AGE,} \\ \text{AGESQ, EDUCATION, GENDER, FARMSIZE,} \\ \text{GLOBALGAP, RECORD, District Dummies,} \\ \text{YEAR 2008 Dummy}). \end{aligned} \quad (4)$$

Acute Symptoms Model. The determinants of the number of acute symptoms were modeled as random effects. A Negative Binomial Regression model (Xtnbreg) was chosen to account for overdispersion, since the equidispersion assumption that has to be met with the Poisson model was violated; that is, the variance was larger than the mean and just over two-thirds of the counts were zero. When there is overdispersion, the Poisson regression is not appropriate because the standard errors estimated are biased downward and the p values are small and spurious [25].

A Negative Binomial Regression model is a count data model and a good facet of the model is that the Poisson model is nested within it [25]. However, the assumption of the standard Poisson model that the variance of the dependent variable is equal to the mean is not binding for the negative binomial model [26]. Negative Binomial Regression model deals with the problem of overdispersion by assuming that y_{it} has a negative binomial distribution, which can be regarded as a generalization of the Poisson distribution with an additional parameter allowing the variance to exceed the mean. The negative binomial function can be presented as

$$\begin{aligned} f(y_{it} | \mu_{it}, \varepsilon_i) \\ = \frac{\Gamma(\mu_{it} + y_{it})}{\Gamma(\mu_{it}) \Gamma(y_{it} + 1)} \left(\frac{\varepsilon_i}{1 + \varepsilon_i} \right)^{y_{it}} \left(\frac{\varepsilon_i}{1 + \varepsilon_i} \right)^{\varepsilon_i}, \end{aligned} \quad (5)$$

where Γ is the gamma function, parameter μ_{it} is assumed as both the mean and the variance, and parameter ε_i is assumed constant over time for each individual, while the mean and variance of y_{it} are given by

$$\begin{aligned} E(y_{it}) &= \varepsilon_i \mu_{it}, \\ \text{var}(y_{it}) &= (1 + \varepsilon_i) \varepsilon_i \mu_{it}. \end{aligned} \quad (6)$$

Under this model, the ratio of the variance to the mean is $1 + \varepsilon_i$ that can vary across individuals but is constant over time. The basic idea for this model is that the predictor information is related to the rate of the response to increase or decrease in counts.

For the empirical model, the acute symptom model aggregates skin irritation, diarrhea, sneezing, headache, dizziness, vomiting, stomach poisoning, blurred vision, eye irritation, and backache episodes incurred by the farmer during and/or soon after spraying pesticide as the dependent variable. For the explanatory variables, the medical literature indicates that the type and severity of pesticide poisoning depend on the toxicity of the pesticides, amount of pesticides involved in the exposure, and route of exposure [27]. The model accounted for these factors. In addition, in order to understand farm management variables that can affect pesticide poisoning, GLOBALGAP certification and record keeping were included. Furthermore, following Antle and Pingali [4], Wilson and Tisdell [28], and Asfaw [19], other control variables under farmer characteristics, that is, age, gender, education, and geographical location, were also included (7).

A priori, it is anticipated that WHO class Ia, Ib, and II pesticides are positively correlated with incidences of acute poisoning whereas negative correlation can be expected with

TABLE 2: Descriptive statistics of variables used in empirical estimations ($N = 726$).

| Variables | Definition | Unit | Mean ^a | | <i>t</i> or <i>z</i> stat. ^b |
|---|---|-------------|-------------------|-------------------|---|
| | | | 2005 | 2008 | |
| Dependent variables | | | | | |
| TACUTE ^c | Number of symptoms | Count | 1.89 (0.13) | 1.09 (0.03) | -7.07*** |
| TACUTE | Number of symptoms | Count | 0.38 (0.48) | 0.37 (0.03) | -0.15 |
| HEALTHCOST ^c | Cost of illness | US\$ | 4.15 (1.70) | 7.98 (1.57) | 1.57 |
| HEALTHCOST | Cost of illness | US\$ | 0.84 (0.35) | 2.72 (0.58) | 2.80** |
| Farmer characteristics variables | | | | | |
| AGE | Age of the farmer | Years | 43.19 (0.66) | 46.18 (0.67) | 6.30*** |
| AGESQ | Age of the farmer (years squared) | Years | 2024.43 (62.80) | 2292.64 (66.85) | 65.21*** |
| EDUCATION | 0 = none; 1 = preprimary; 2 = primary; 3 = secondary; 4 = college | Ordinal | 2.45 (0.05) | 2.51 (0.04) | 1.09 |
| GENDER | Male | 1/0 | 0.70 (0.02) | 0.70 (0.02) | 0.00 |
| EXPERIENCE | Farming experience | Years | 18.42 (0.74) | 20.56 (0.07) | 2.38** |
| Health-related and pesticide exposure variables | | | | | |
| HEALTH | Farmer reported a symptom | 1/0 | 0.20 (0.02) | 0.34 (0.02) | 4.26*** |
| SEVERE | 1 = mild; 2 = severe; 3 = very severe | Ordinal | 1.11 (0.08) | 1.59 (0.36) | 1.22 |
| PWHOIab | WHO Ia and WHO Ib (extremely hazardous) | g | 8.79 (2.32) | 33.55 (12.79) | 1.92** |
| PWHOII | WHO category II (moderately hazardous) | g | 129.87 (10.15) | 432.63 (25.20) | 10.97*** |
| PWHOIII | WHO category III (slightly hazardous) | g | 18.95 (3.39) | 166.12 (19.23) | 7.45*** |
| PWHOU | WHO category U (no hazard) | g | 79.87 (7.47) | 167.79 (16.86) | 4.87*** |
| PESTHA | Total amount applied | g/ha/season | 1,473.00 (201.82) | 2,124.87 (118.28) | 2.97*** |
| NPEST | Pesticide products | Count | 2.89 (0.09) | 3.32 (0.08) | 3.37*** |
| COAT | Wear coat/apron | 1/0 | 0.49 (0.03) | 0.71 (0.02) | 6.06*** |
| GLOVE | Wear gloves | 1/0 | 0.26 (0.02) | 0.35 (0.02) | 2.49** |
| GUMBOOT | Wear boots | 1/0 | 0.26 (0.02) | 0.89 (0.02) | 17.35*** |
| MASK | Wear facemask | 1/0 | 0.24 (0.02) | 0.40 (0.02) | 4.36*** |
| TPPE | Protective equipment | Count | 2.81 (0.07) | 4.00 (0.11) | 10.85*** |
| Farm management variables | | | | | |
| FARMSIZE | Total farm size | ha | 1.46 (0.08) | 1.06 (0.05) | -4.46*** |
| GLOBAL-GAP | GLOBALGAP certified farmers | 1/0 | 0.07 (0.01) | 0.19 (0.02) | 0.15 |
| RECORD | Records keeping | 1/0 | 0.71 (0.02) | 0.32 (0.01) | -10.47*** |

All monetary variables for example health cost were adjusted (normalized) to US\$ of 2008 to take account of inflation. US\$ = 72 KSh (2005) and 75 KSh (2008).

^aFigures in parenthesis are standard errors.

^bStatistical significance at the 0.01 (***), 0.05 (**), and 0.1 (*) levels of probability. Categorical variables were analyzed using *z*-test.

^cWith only farmer who reported the health impairment.

Source: this study.

category III and U pesticide. Pesticides in WHO Ia, WHO Ib, and WHO II are very harmful, while WHO III and WHO U are less harmful [29].

Age could increase acute symptoms, as older farmers may be less concerned about health effects of pesticides. As already mentioned in cost of illness model it is expected

that pesticide-related acute symptoms decrease with the increase in level of education, GLOBALGAP certification, record keeping of production activities, and appropriate use of personal protective equipment:

$$\begin{aligned} \text{TACUTE} = f(\text{AGE, AGESQ, EDUCATION,} \\ \text{GENDER, GLOBALGAP, RECORD, NPEST,} \\ \text{PWHOIab, PWHOII, PWHOIII, PWHOU, COAT,} \quad (7) \\ \text{GLOVE, GUMBOOT, MASK, District Dummies,} \\ \text{YEAR 2008 Dummy}). \end{aligned}$$

The models were estimated using the random effect estimator as the Hausman test showed the fixed effects were not correlated with the regressors. All variables were cross-checked for the problem of multicollinearity, through the simple correlation matrix and variance inflation factor (VIF). The highest correlation coefficient was 0.32 and VIF were by far less than three, indicating that correlation between explaining variables could not affect the estimation of coefficients. Likewise, for endogeneity none of the independent variables was suspected to be explained within the equation in which they appeared. Misspecifications of the models were also checked using a regression specification error test [30]. In respect to the robustness of the Negative Binomial Regression model, a Poisson model was first fitted and the likelihood ratio test together with the statistical evidence of overdispersion indicated that the Negative Binomial Regression model was preferred to the Poisson model. In addition, to check the robustness of all the models other restricted models were estimated in which subsequently insignificant variables were dropped. The statistical quality of the models and the direction of the signs did not change, and the coefficients deviated only marginally.

3. Empirical Results and Discussion

3.1. Descriptive Statistics of Variables Used in Empirical Estimations. Table 2 summarizes the main descriptive statistics comparing 2005 and 2008 with *t*- and *z*-tests for the main variables investigated. The results showed that the incidences of pesticide-related acute illness had increased by over 70%. By cross-check although not indicated in the table the analysis showed that only 45% of the farmers consequently reported the effect once more in 2008 showing a high rate of new episodes cases. However, the number of symptoms per farmer dropped by almost half in 2008. In terms of frequency of symptom occurrence, headache and sneezing were reported as the main symptoms in both surveys. Dizziness as one of the major neurological effects of pesticide exposure was also found to have doubled in 2008. These symptoms have been associated with pesticides acute poisoning [27]. They are also consistent with other studies of pesticides exposure on farmers' health elsewhere [31–33].

A total of 62 pesticides products, comprising 36 active ingredients formulated singly or in mixture, were used to control various vegetable pests in 2005. The number increased

slightly to 66 products in 2008 with 44 active ingredients in the formulations. However, close analysis showed that 19 new products were applied in 2008, implying that 15 products of those used in 2005 were dropped. The commonly used products included dimethoate (WHO II), used by 48% of farmers, lambda cyhalothrin (WHO II, 27%), cymoxanil (WHO II, 22%), cypermethrin (WHO II, 22%), cyfluthrin (WHO Ib, 20%), mancozeb (WHO U, 18%), and deltamethrin (WHO II, 14%).

For minor poisoning, many farmers used home remedies such as milk, lemon juices, honey, and herbs. The medicines from the local pharmacy shops which were sometimes painkillers were bought in cases where the symptoms of illness were mild and farmers visited the health clinic if the symptoms either persisted or became serious; that is, the victim was unable to talk, walk, or see or vomited continuously. This evidence seems to suggest that many farmers treat acute pesticide effects as minor problems that do not warrant medical attention. Although in only about a quarter of the poisoning cases a physician was consulted, this cost component accounts for the largest share of the total cost of treatment.

The health cost almost doubled in 2008 as compared to 2005. On average, health cost was estimated at US\$ 6.55/farmer/season for 28% of the farmers who reported pesticide-related illnesses. These costs equal 47% of mean household pesticides expenditures in 2008. Considering all the farmers this translates to a mean of US\$ 1.77/farmer/season and assuming two crop seasons per year the costs amount to US\$ 3.54/farmer/year. However, the true health costs are likely to be much higher because the costs arising from chronic diseases resulting from long-term pesticides exposure were not considered, as this would have required more detailed medical assessments. Moreover, only costs directly involving family members were reported; costs occurring to hired farm laborers were not included. Furthermore, other "costs" to restore health status completely and nonmonetary costs like suffering and income lost by family members assisting in seeking treatment were not captured [34, 35]. In addition, preventive costs associated with precautions taken to reduce exposure such as wearing protective equipment were not considered because they were mainly improvised from old clothing or pieces of cloth wrapped around the nose and mouth to reduce inhalation exposure. The cloths were also used for other purposes like spraying on coffee and other farm work and it was difficult to specify those used for spraying pesticides on vegetable crops alone. However, the combined mean of personal protective equipment used increased by 43%, with the largest increment noted for gumboots. Over 20% of farmers also paid wage premiums of up to 32% above the normal wage to hired labor for spraying pesticides, which were normally paid in cash.

Comparison with other studies conducted in developing countries shows similar results. Pingali et al. [36] showed that 58%–99% of the farmers exposed to pesticides had at least one health effect symptom in Indonesia, Philippines, and Vietnam. In Tanzania farmer spending on health due to pesticide and exposure ranges between US\$ 0.018 and 116 in

TABLE 3: Tobit model for cost of illness estimations.

| Model Variables | Unrestricted | | Restricted | |
|----------------------------|----------------------------|---------|----------------------------|---------|
| | (Coefficient) ^a | z-value | (Coefficient) ^a | z-value |
| TACUTE | 7.45 (4.08)** | 1.83 | 6.20 (2.00)*** | 3.10 |
| SEVERE | 9.01 (2.52)*** | 3.58 | 11.07 (2.17)*** | 5.12 |
| AGE | -0.48 (1.09) | -0.44 | | |
| AGESQ | 0.01 (0.01) | 0.61 | | |
| EDUCATION | 1.46 (2.36) | 0.62 | | |
| GENDER | -2.84 (4.33) | -0.66 | | |
| FARMSIZE | 3.25 (2.88) | 1.13 | | |
| GLOBALGAP | -21.75 (3.40)* | -1.62 | -18.71 (7.47)** | -2.50 |
| RECORD | -1.08 (4.89) | -0.22 | | |
| KIAMBU | 2.50 (10.31) | 0.24 | | |
| MAKUENI | -15.08 (17.25) | -0.87 | | |
| MERU CENTRAL | 1.65 (8.71) | 0.19 | | |
| MURANGA | -5.48 (11.75) | -0.47 | | |
| NYANDARUA | -5.61 (8.81) | -0.64 | | |
| NYERI NORTH | 6.62 (8.47) | 0.78 | | |
| YEAR 2008 | 7.93 (9.15) | 0.87 | | |
| Constant | -23.23 (29.41) | -0.79 | -19.54 (4.45)*** | -4.39 |
| Log likelihood | -464.10 | | -549.55 | |
| Wald χ^2 /LR χ^2 | 40.18*** | | 43.22*** | |

^aFigures in parenthesis are robust standard errors, statistically significant at the 0.01 (***) , 0.05 (**), and 0.1 (*) levels of probability.

Source: this study.

a year [37]. In West Africa, the economic value of pesticide-related health costs equals US\$ 3.92/household/season in the case of cotton-rice systems [38]. Zimbabwe cotton growers incur a mean of US\$ 4.73 in Sanyati and US\$ 8.31 in Chipinge on pesticide-related direct and indirect acute health effects [2]. In Sri Lanka, cost to farmers from pesticide exposure equals 10 weeks' income [39], while in India the average annual welfare loss to an applicator from pesticide exposure amounts to US\$ 36 [40]. The immediate costs of a typical intoxication (medical attention, medicines, and days of recuperation) equaled the value of 11 days of lost wages in Ecuador [41].

Pesticide application rate/hectare/season also increased by 47%. Comparison between the years for the specific farmers who participated in the DBM survey showed that many farmers had reduced the pesticides application rate by 8%, while the GLOBALGAP surveyed farmer had increased by 40%. Similar findings in support of the reduction of pesticide use were reported by Jankowski [42] and Löhr et al. [43] where farmers in the study areas with DBM biocontrol (*Diadegma semiclausum*) reduced pesticide applications with others even stopping spraying altogether. The increase in application rate by GLOBALGAP farmers can partially be explained by the low number of farmers who were certified at the time of survey and the failure of the farmers certified in 2005, to maintain their certification status; that is, certified farmers dropped from 18% to 7%, with only 31% of the farmers maintaining their certification for 2008.

3.2. Model Estimations

3.2.1. Cost of Illness Estimation. The estimation results of the Tobit models with the health costs as dependent variable are reported in Table 3. Result shows that health costs are positively associated with number of symptoms and symptoms severity, which implies that an increase in any of these variables spontaneously influences positively the health costs, holding other factors constant.

The finding that the GLOBALGAP certification tends to decrease the health cost could indicate that the certified farmers use adequate safety precautions or use low toxic pesticides, which generally reduce the health impairments and thus decrease costs. It could also be that these farmers are able to use the minimum treatment possibilities.

Among the farmers' characteristics variables, that is, age, education, and gender, none had any discernible effect on health costs. In addition, farm size though considered as an indicator of wealth does not have a direct effect on health costs, though it has the correct sign. Perhaps it could be because farms do not present "liquid cash" that can be accessed immediately in time of need. In addition, no direct association was found between record keeping and the health costs.

District controls are insignificant, so location does not directly affect the health costs. When the model was reestimated (restricted) by dropping insignificant variables, the estimates of the coefficients were robust.

TABLE 4: Binomial Regression model for the acute symptoms estimations.

| Model Variables | Unrestricted | | Restricted | |
|-----------------|----------------------------|---------|----------------------------|---------|
| | (Coefficient) ^a | z-value | (Coefficient) ^a | z-value |
| AGE | 0.04 (0.04) | 1.13 | | |
| AGESQ | -0.00 (0.00) | -1.21 | | |
| EDUCATION | -0.16 (0.07)** | -2.13 | -0.14 (0.07)* | -1.94 |
| GENDER | -0.10 (0.16) | -0.67 | | |
| GLOBALGAP | -0.33 (0.29) | -1.11 | | |
| RECORD | -0.44 (0.17)*** | -2.57 | -0.55 (0.15)*** | -3.77 |
| NPEST | 0.09 (0.05)** | 1.88 | 0.10 (0.05)** | 2.39 |
| PWHOlab | 0.00 (0.00) | 1.28 | | |
| PWHOII | 0.00 (0.00) | 0.68 | | |
| PWHOIII | -0.00 (0.00) | -0.28 | | |
| PWHOU | -0.00 (0.00) | -0.13 | | |
| COAT | -0.29 (0.16)* | -1.82 | -0.29 (0.15)** | -2.03 |
| GLOVE | -0.26 (0.21) | -1.23 | | |
| GUMBOOT | 0.32 (0.23) | 1.36 | | |
| MASK | -0.35 (0.20)* | -1.74 | -0.39 (0.17)** | -2.30 |
| KIAMBU | 1.69 (0.36)*** | 4.67 | 1.63 (0.32)*** | 5.20 |
| MAKUENI | 1.74 (0.49)*** | 3.55 | 1.50 (0.46)*** | 3.35 |
| MERU CENTRAL | 1.18 (0.31)*** | 3.82 | 0.95 (0.25)*** | 3.77 |
| MURANGA | 0.64 (0.46) | 1.40 | | |
| NYANDARUA | 0.90 (0.34)*** | 2.66 | 0.80 (0.28)*** | 2.81 |
| NYERI NORTH | 0.93 (0.30)*** | 3.07 | 0.79 (0.24)*** | 3.26 |
| YEAR 2008 | -0.05 (0.21) | -0.23 | | |
| Constant | -1.22 (1.06) | -1.15 | -0.01 (0.48) | -0.02 |
| Log likelihood | -518.85 | | -535.52 | |
| Wald χ^2 | 73.74*** | | 60.96*** | |

^aFigures in parenthesis are robust standard errors, statistically significant at the 0.01 (* * *), 0.05 (**), and 0.1 (*) levels of probability. Source: this study.

3.2.2. Acute Symptoms Estimation. Given the critical contribution of pesticide-related acute symptoms to the health costs as indicated in Table 2, the principal determinants of these symptoms are reported in Table 4.

The model shows that pesticide-related acute symptoms increase significantly with the number of pesticide products handled. This is not surprising, given that different pesticide products require different application rates and have different levels of toxicity. In addition, handling different pesticide products can increase incidences of symptoms since an interaction between pesticides can lead to unknown toxic chemical reactions [44]. Likewise, although the coefficients for pesticides in WHO Iab and WHO II are insignificant, they are positively correlated with acute symptoms whereas negative correlation is observed with WHO III and WHO U pesticides. The significant negative sign of the variable “record keeping” suggests that the probability of pesticide-related illnesses is less for farmers who keep records. In general, record keeping of pesticide products handled, their application dosage, application techniques, and production activities enabled farmers to be more judicious on pesticides use and higher tendency to protect them. With records, a farmer can also see how well she/he is managing production

operations and can identify the strengths and weaknesses in those activities.

The level of education reduces the probability of reported symptoms, which implies that farmers with a higher education level are more knowledgeable and therefore have a better understanding of the dangers posed by pesticides. In previous studies, however, the contrary effect was found because respondents with higher knowledge were more likely to report more health symptoms [2].

The use of personal protective equipment particularly the use of a coat/apron and facemask significantly reduced the number of symptoms. Exposure to pesticides is often attributed to a failure to use protective equipment [34]. The positive sign of the use of boots although insignificant seems perverse and alarming at first glance. However, as the researcher had observed in the field, the improper use, that is, putting the trouser inside the boots, may offer a partial explanation of this apparently perverse result. This finding is analogous to that found by Ohayo-Mitoko et al. [45], where use of gumboots was associated with high *acetyl cholinesterase* inhibition. *Acetyl cholinesterase* is enzyme that breaks down acetylcholine (ACh) into choline and acetic acid. It is released onto the sarcolemma of muscle fibers and destroys ACh after

the ACh has combined with receptors on the muscle fiber. Thus, it prevents continued muscle contraction in the absence of additional nervous stimulation.

Location control for agroecology and differences in institutional settings shows that farmers in the districts of Kiambu, Meru Central, Makueni, Nyandarua, and Nyeri North experience significantly high cases of pesticide ascribed health symptoms as compared to Kirinyaga (base). Perhaps this is due to the use of protective equipment by farmers located in Kirinyaga.

Contrary to the expectations, the analysis does not support the hypothesis of a significant influence of GLOBALGAP certification on the outcome of health, but the variable has the correct signs. Once again, the low number of farmers who were certified and the failure of the certified farmers to maintain their certification may be the cause of the insignificance. The hypothesis that gender and age have a stronger relation to the acute symptoms is also not supported by the results.

The likelihood ratio test used to assess the statistical quality of the model showed that the model was statistically valid, that is, dispersion parameter alpha was greater than zero. The reduced model with only the variables that had a significant effect on the dependent variable shows that the statistical quality of the model does not differ much and the directions of the coefficient are identical, suggesting the robustness of the model.

4. Conclusions and Recommendations

The results of the study give indications of increase of pesticide-related health impairments with over 70% new episodes. The most frequently reported symptoms were sneezing, dizziness, headache and blurred vision, and skin irritations. The result further shows that farmer loses on average US\$ 3.54/farmer/year on pesticide-related indirect health costs. These costs are significantly explained by variation in number of symptoms and severity of the symptoms. Pesticide-related acute symptoms increase significantly with the number of pesticide products handled and considerably reduce with level of education, use of PPE, and record keeping. These findings hint at some important points for policies aiming to reduce pesticide poisoning among vegetable farmers. Firstly, the results support the already widely known notion that proper use of PPE (coat/apron and facemask) reduces the pesticide-related impairment. Encouraging of PPE and record keeping of pesticide use activities by farmer is thus recommended.

Future efforts to measure pesticide-related health costs should cover the health costs of all individuals exposed to pesticides, for example, entire public, consumers, and hired workers, and also incorporate pesticide-induced chronic illnesses and deaths.

Conflict of Interests

The author declares that there is no conflict of interests regarding the publication of this paper.

Acknowledgments

This research was supported by DAAD and an ICIPE-GTZ/BMZ funded project. The author expresses appreciation to Professor Dagmar Mithöfer, Professor Hermann Waibel, farmers, and staff members of the Ministry of Agriculture and other individuals who provided valuable assistance.

References

- [1] J. M. Antle, D. C. Cole, and C. C. Crissman, "Further evidence on pesticides, productivity and farmer health. Potato production in Ecuador," *Agricultural Economics*, vol. 18, no. 2, pp. 199–207, 1998.
- [2] B. M. Maumbe and S. M. Swinton, "Hidden health costs of pesticide use in Zimbabwe's smallholder cotton growers," *Social Science and Medicine*, vol. 57, no. 9, pp. 1559–1571, 2003.
- [3] H. Garming and H. Waibel, "Pesticides and farmer health in Nicaragua: a willingness-to-pay approach to evaluation," *European Journal of Health Economics*, vol. 10, no. 2, pp. 125–133, 2009.
- [4] J. M. Antle and P. L. Pingali, "Pesticides, productivity, and farmer health: a Philippine case study," *American Journal of Agricultural Economics*, vol. 76, no. 3, pp. 418–430, 1994.
- [5] A. M. Raschke and A. E. C. Burger, "Risk assessment as a management tool used to assess the effect of pesticide use in an irrigation system, situated in a semi-desert region," *Archives of Environmental Contamination and Toxicology*, vol. 32, no. 1, pp. 42–49, 1997.
- [6] L. Horrigan, R. S. Lawrence, and P. Walker, "How sustainable agriculture can address the environmental and human health harms of industrial agriculture," *Environmental Health Perspectives*, vol. 110, no. 5, pp. 445–456, 2002.
- [7] M. C. R. Alavanja, J. A. Hoppin, and F. Kamel, "Health effects of chronic pesticide exposure: cancer and neurotoxicity," *Annual Review of Public Health*, vol. 25, pp. 155–197, 2004.
- [8] F. Kamel and J. A. Hoppin, "Association of pesticide exposure with neurologic dysfunction and disease," *Environmental Health Perspectives*, vol. 112, no. 9, pp. 950–958, 2004.
- [9] M. Kishi, "The health impacts of pesticides, what do we now know," in *The Pesticide Detox, Towards a More Sustainable Agriculture*, J. Pretty, Ed., Earthscan, London, UK, 2005.
- [10] F. D. Dick, G. DePalma, A. Ahmadi et al., "Environmental risk factors for Parkinson's disease and parkinsonism: the Geoparkinson study," *Occupational and Environmental Medicine*, vol. 64, no. 10, pp. 666–672, 2007.
- [11] M. Nasterlack, "Pesticides and childhood cancer, an update," *International Journal of Hygiene and Environmental Health*, vol. 210, no. 5, pp. 645–657, 2007.
- [12] F. Kamel, C. M. Tanner, D. M. Umbach et al., "Pesticide exposure and self-reported Parkinson's disease in the agricultural health study," *American Journal of Epidemiology*, vol. 165, no. 4, pp. 364–374, 2007.
- [13] D. B. Hancock, E. R. Martin, G. M. Mayhew et al., "Pesticide exposure and risk of Parkinson's disease; a family-based case-control study," *BMC Neurology*, vol. 8, article 6, 2008.
- [14] S. Dasgupta and C. Meisner, "Health effects and pesticide perception as determinants of pesticide use, evidence from Bangladesh," World Bank Policy Research Working Paper 3776, 2005.

- [15] C. Wilson, "Exposure to pesticides, ill-health and averting behaviour: costs and determining the relationships," *International Journal of Social Economics*, vol. 32, no. 12, pp. 1020–1034, 2005.
- [16] H. P. Binswanger and R. F. Townsend, "The growth performance of agriculture in Sub-Saharan Africa," *American Journal of Agricultural Economics*, vol. 82, no. 5, pp. 1075–1086, 2000.
- [17] Pesticide Trust, *The Pesticide Hazard, A Global Health and Environmental Audit*, compiled by B. Dinarm, The Pesticide Trust Biddles Limited, London, UK, 1993.
- [18] L. London, A. J. Flisher, C. Wesseling, D. Mergler, and H. Kromhout, "Suicide and exposure to organophosphate insecticides, cause or effect?" *American Journal of Industrial Medicine*, vol. 47, no. 4, pp. 308–321, 2005.
- [19] S. Asfaw, *Global agrifood supply chain, EU food safety standards and African small-scale producers, the case of high value horticultural exports from Kenya [Ph.D. thesis]*, Leibniz University of Hanover, Hanover, Germany, 2008.
- [20] J. J. Okello, *Compliance with international food-safety standards, the case of green bean production in Kenyan family farms [Ph.D. Dissertation]*, Michigan State University, East Lansing, Mich, USA, 2005.
- [21] G. J. A. Ohayo-Mitoko, H. Kromhout, J. M. Simwa, J. S. M. Boleij, and D. Heederik, "Self reported symptoms and inhibition of acetylcholinesterase activity among Kenyan agricultural workers," *Occupational and Environmental Medicine*, vol. 57, no. 3, pp. 195–200, 2000.
- [22] W. Greene, *Econometric Analysis*, Pearson/Prentice Hall, Upper Saddle River, NJ, USA, 6th edition, 2008.
- [23] B. H. Baltagi, *Econometric Analysis of Panel Data*, Wiley, West Sussex, UK, 3rd edition, 2005.
- [24] J. Tobin, "Estimation of relationships for limited dependent variables," *Econometrica*, vol. 26, pp. 24–36, 1958.
- [25] J. S. Long, *Regression Models for Categorical and Limited Dependent Variables*, SAGE Publications, Thousand Oaks, Calif, USA, 1997.
- [26] C. A. Cameron and P. K. Trivedi, *Regression Analysis of Count Data*, Cambridge University Press, Cambridge, UK, 1998.
- [27] Extension Toxicology Network, *Cholinesterase Inhibition Toxicology Information Brief Cornell University*, 2004, <http://mpem.cce.cornell.edu/profiles/extoxnet/TIB/cholinesterase.html>.
- [28] C. Wilson and C. Tisdell, "Why farmers continue to use pesticides despite environmental, health and sustainability costs," *Ecological Economics*, vol. 39, no. 3, pp. 449–462, 2001.
- [29] WHO, "The World Health Organization (WHO) recommended classification of pesticides by hazard and guidelines to classification," 2009, http://www.who.int/ipcs/publications/pesticides_hazard_2009.pdf.
- [30] J. B. Ramsey, "Tests for specification errors in classical linear least-squares regression analysis," *Journal of the Royal Statistical Society—Series B: Methodological*, vol. 31, pp. 350–371, 1969.
- [31] S. A. Martin Jr., D. P. Sandler, S. D. Harlow, D. L. Shore, A. S. Rowland, and M. C. R. Alavanja, "Pesticide use and pesticide-related symptoms among black farmers in the Agricultural Health study," *American Journal of Industrial Medicine*, vol. 41, no. 3, pp. 202–209, 2002.
- [32] K. Atreya, "Health costs of pesticide use in a vegetable growing area, central mid-hills, Nepal," *Himalayan Journal of Sciences*, vol. 3, no. 5, pp. 81–84, 2005.
- [33] M. C. R. Alavanja, N. L. Sprince, E. Oliver et al., "Nested case-control analysis of high pesticide exposure events from the agricultural health study," *American Journal of Industrial Medicine*, vol. 39, no. 6, pp. 557–563, 2001.
- [34] A. C. Rola and P. L. Pingali, *Pesticides, Rice Productivity, and Farmers' Health*, International Rice Research Institute (IRRI), World Resources Institute (WRI), Washington, DC, USA, 1993.
- [35] A. M. Freeman, *The Measurement of Environmental and Resource Values: Theory and Methods*, Resources for the Future, Washington, DC, USA, 1993.
- [36] P. L. Pingali, C. B. Marquez, and F. G. Palis, "Pesticides and Philippine rice farmer health: a medical and economic analysis," *American Journal of Agricultural Economics*, vol. 76, no. 3, pp. 587–592, 1994.
- [37] A. V. F. Ngowi, T. J. Mbise, A. S. M. Ijani, L. London, and O. C. Ajayi, "Smallholder vegetable farmers in Northern Tanzania: pesticides use practices, perceptions, cost and health effects," *Crop Protection*, vol. 26, no. 11, pp. 1617–1624, 2007.
- [38] O. C. Ajayi, M. Camara, G. Fleischer et al., "Socio-economic assessment of pesticide use in Mali," Pesticide Policy Project Publication Series no. 6, University of Hanover, Hanover, Germany, 2002.
- [39] C. Wilson, *Cost and policy implications of agricultural pollution with special reference to pesticides [Ph.D. thesis]*, University of St Andrews, St Andrews, UK, 1998.
- [40] I. P. Devi, "Pesticide use in the rice bowl of Kerala, health costs and policy options SANDEE," Working Paper no. 21, South Asian Network for Development and Environmental Economics (SANDEE), Kathmandu, Nepal, 2007.
- [41] D. Yanggen, D. Cole, C. Crissman, and S. Sherwood, *Human Health, Environmental and Economic Effects of Pesticide Use in Potato Production in Ecuador*, International Potato Center (CIP), Lima, Peru, 2003.
- [42] A. Jankowski, *Economics of biological control of diamondback moth in vegetable production in East Africa [MSc Dissertation]*, Leibniz University of Hanover, Hanover, Germany, 2007.
- [43] B. Löhr, R. Gathu, C. Kariuki, J. Obiero, and G. Gichini, "Impact of an exotic parasitoid on *Plutella xylostella* (Lepidoptera: Plutellidae) population dynamics, damage and indigenous natural enemies in Kenya," *Bulletin of Entomological Research*, vol. 97, no. 4, pp. 337–350, 2007.
- [44] L. Yáñez, D. Ortiz, J. Calderón et al., "Overview of human health and chemical mixtures: problems facing developing countries," *Environmental Health Perspectives*, vol. 110, no. 6, pp. 901–909, 2002.
- [45] G. J. A. Ohayo-Mitoko, H. Kromhout, P. N. Karumba, and J. S. M. Boleij, "Identification of determinants of pesticide exposure among Kenyan agricultural workers using empirical modelling," *Annals of Occupational Hygiene*, vol. 43, no. 8, pp. 519–525, 1999.



Hindawi

Submit your manuscripts at
<http://www.hindawi.com>

