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Pesticides and health: A review of evidence on health effects, valuation of risks, and benefit-cost analysis

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Abstract
In this paper, we provide reviews of recent scientific findings on health effects and preference valuation of health risks related to pesticides, and the role of benefit-cost analysis in policies related to pesticides. Our reviews reveal that whereas the focus of the health literature has been on individuals with direct exposure to pesticides, e.g. farmers, the literature on preference elicitation has focused on those with indirect exposure, e.g. consumers. Our discussion of pesticides policies emphasizes the need to clarify the rationale for regulation and the role of risk perceptions in benefit-cost analysis, and stress the importance of inter-disciplinary research in this area.

Keywords: Benefit-cost analysis; Health; Pesticides; Willingness to Pay

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1 Introduction

The use of chemicals in modern agriculture has significantly increased productivity. But it has also significantly increased the concentration of pesticides in food and in our environment, with associated negative effects on human health. Annually there are dozens of million cases of pesticide poisonings worldwide (Richter, 2002). Moreover, it is now better understood that pesticides have significant chronic health effects, including cancer, neurological effects, diabetes, respiratory diseases, fetal diseases, and genetic disorders. These health effects are different depending on the degree, and the type of exposure. Typically, the effects are different for farmers who are directly exposed to pesticides, compared to those for farmers' relatives or people living in rural areas who are less directly exposed. There are also effects on consumers through pesticide residues in food.

Pesticides use and regulation have long been controversial. The publication in 1962 of the famous *Silent Spring* by the biologist Rachel Carson made popular the risks associated to DDT (dichlorodiphenyltrichloroethane) (Carson, 1962). This was followed by the cancelation of this pesticide for agricultural uses by US authorities. Other prominent examples of pesticide cancelation include EDB (ethylene dibromide) in 1983 and methyl bromide in 2005. It is well known now that a significant fraction of pesticides are carcinogenic; for instance, 18% of all insecticides and 90% of all fungicides were found to be carcinogenic (NAS, 1987). It is also well known that pesticide residues remain for long periods of time, and that they are especially toxic to the young. Also, pesticides kill domestic animals, fishes and bees. Moreover, their use results in the development and evolution of pesticide resistance in insects, weeds and plant pathogens. Nevertheless hundreds of pesticides are used worldwide, and some pesticides are used in some countries but not in others. For instance, the main pesticide which is used in corn production in the US is atrazine, but this pesticide has been banned in the EU because of its toxicity since 2004 (Official Journal of the European Union 2004/248/CE).

Public decisions concerning pesticides have long been suspected of regulatory capture. One of the reasons for transferring in 1970 pesticides regulatory responsibility from the US Department of Agriculture to the Environmental Protection Agency (EPA) was to lessen the influence of farmers and pesticide producers. But this transfer of responsibility naturally increased the influence of consumers and environmentalists. Indeed, Cropper et al. (1992) showed that both grower and environmental groups' participation played a major role in explaining the EPA decisions to cancel a pesticide in the 1970s and 1980s. Risk assessment practices also play a role in pesticides regulation. The zero-risk or "de minimis risk" target has long been the advanced objective of regulators. But this objective is overly ambitious, and often not implemented as a result. There is a lot of evidence for instance that a significant portion of food samples still exceeds the maximum residue limits set by regulators both in the US and in Europe. Finally, risk perceptions may also influence pesticide regulation. Indeed, there is evidence that people underestimate the risks from natural carcinogens but that they nevertheless overestimate the risks from carcinogenic pesticides (Slovic, 2000).

These observations illustrate the challenge of regulating pesticides. At the root of this challenge there is the immense difficulty of producing more food with less pesticide, and the uncertainty about health effects of pesticides. Zilberman and Millock (1997) talk about a regulation nightmare. Given the growing health concerns of the population, some drastic actions to curb the use of pesticides have

been decided in some countries. For instance, Denmark decided as early as in 1986 to reduce by two the pesticide treatment frequency in agriculture. More recently, France announced in 2008 a reduction by two of pesticide use by 2018 in its "Ecophyto 2018" plan (MAP, 2009). A major problem with such ambitious policy targets is that they need not reflect an appropriate balance of benefits and costs induced by pesticides use in our societies. Also, these policy targets are difficult, if not impossible, to implement in practice, in part because of the opposition of farmers. In this difficult political context, the US EPA has long noticed that producing sound comparisons of benefits and costs of proposed pesticides regulations might be helpful to policy makers in order to identify and design a coherent pesticide regulatory policy (EPA US, 1992). However, only few studies based on benefit-cost analysis (BCA) concerning pesticides have been produced so far (Pimentel, 2005).

The aim of this study is to contribute to the understanding of the health effects of pesticides exposure and of how pesticides have been and should be regulated. Our conceptual framework is BCA, namely the most common economic method to evaluate a public policy. In the context of pesticide regulation, this framework requires evidence on health effects on those exposed directly and indirectly, valuation of risks, consideration of market and nonmarket failures, policy alternatives, and estimates of costs. As a first step we provide literature reviews on recent scientific findings on health effects. As a second step, we provide evidence on preference valuation of health risks related to pesticides use. Finally, we provide an overview and discussion of different policies and of difficulties of evaluating them using BCA. One objective of these reviews is to relate the findings from the different disciplines (health, economics and public policy) to each other, and to identify gaps and needs for future research.

The paper is organized as follows. The following section contains the review of the finding on the health effects related to pesticides use. Section 3 thereafter, after a brief introduction on preference elicitation, summarizes the findings from the literature on individual preferences and pesticides. Both section 2 and 3 end with some concluding remarks on each topic. A review and discussion on how pesticides use has been regulated and evaluated are then provided in section 4. We end the paper with some concluding remarks in section 5. Moreover, the Appendix contains a set of tables in which we briefly present the results of the scientific publications on health effects and valuation of risks selected in our sample.

2 Health effects of pesticides

The following section is dedicated to a survey of the existing literature on the health effects of pesticides. It focuses on agriculture exposure, so potentially harmful substances other than pesticides and different frameworks are out of the scope of this review. The analysis distinguishes between three types of studies depending on the people exposed: (i) people with direct exposure, (ii) members of a community with indirect exposure, and (iii) consumers.

Previous literature on potential health effects of pesticides first focused on the risks of acute intoxication among people with direct exposure. The availability of longitudinal data shifted the main concern to the risks of chronic intoxication and environmental contamination. More recently the concern moved to diseases such as cancer for which the risk is not associated with instant effects of pesticides but with chronic exposure. Nowadays, the concern about latent effects is not only on people

with direct exposure but also on subjects with indirect exposure such as consumers or residents of rural communities. Different risks associated with pesticides are often classified based on whether they have short-term effects (such as diarrhea, abdominal pain, headaches, nausea, vomiting, etc.) or long-term effects (such as skin diseases, cancer, depression, neurological deficits, diabetes, genetic disorders, or even death).

Since the existing literature on this topic is quite extensive and some other reviews have already been conducted (see, e.g., Alavanja et al., 2004; Florax et al., 2005; Cocco, 2002; Garcia, 2003), we focused on studies from 2000 to the present. The literature review was conducted in March 2013, and we included only published studies with "pesticide" in the title and "health" in the title or abstract, or with "pesticide exposure" in the title.[1] The search tool was EBSCOhost, which includes Business Source, EconLit, Medline, GreenFile, among others sources. We found 527 original texts, and after excluding news articles, books, and non-related or repeated results, we ended up with a sample of 191 studies.

Not including studies published before 2000 leads to an underrepresentation of certain health effects that are not in the agenda anymore such as skin reactions. On the other hand, chronic diseases have received more attention lately and this is reflected in the high number of publications analyzing these types of disease.

2.1 Health effects of pesticides on people with direct exposure

A wide range of subjects are included in this category. We considered studies whose subjects are not only people who spray pesticides, but also who mix and load the pesticides, sow pesticide-seeds, weed and harvest sprayed crops, and clean and dispose of containers. A total of 122 studies were found (63.9% of the total), and Table 1 in the appendix presents their most relevant features. Most of the statistically significant results are pesticide-specific so it is not possible to generalize regarding health effects of pesticides.

2.1.1 Cancer

Cancer associated with pesticide exposure is one of the most studied topics related to pesticides' toxicity during the last decade. Of the studies found, 43 analyze the relation between direct exposure to pesticides and the risk of cancer. Most of the studies use the Agricultural Health Study (AHS) data. No unanimous agreement has been reached: 12 of the studies report no significant evidence of increased risk of cancer among farmers exposed to pesticides compared with the risk of the general population (see, e.g., De Roos et al., 2005; Greenburg et al., 2008; Lynch et al., 2006), while the rest of the studies (31) conclude that exposure to certain pesticides significantly increases the risk of cancer (see, e.g., Lynch et al., 2009; Alavanja et al., 2003; Beane et al., 2005). The heterogeneity of the results is related to the type of cancer being analyzed as well as the nature of the pesticides.[2]

[1] More general searches were tested, but mostly the number of non-related papers increased substantially.

[2] Weichenthal et al. (2010) reviews in a simplified way the epidemiologic evidence related to occupational pesticide exposures and cancer incidence in the AHS cohort. Some of these studies were not included in our review since they were published in journals not considered by our search tool.

2.1.2 Depression and neurological deficits

The evidence found in 3 studies suggests that high-intensity and cumulative pesticide exposure contributes to depression among pesticide applicators (see, e.g., Beseler et al., 2008). Different studies have been carried out analyzing the effects of pesticide exposure on neurological function. Evidence relating long durations of farm work with decreasing levels of performance has been found, and one relevant factor might be chronic exposure to pesticides (Kamel et al., 2003). Results are mixed depending on the pesticide being analyzed: some of them suggest that neurological symptoms are associated with cumulative exposure, but this is true only for some fumigants and insecticides (Kamel et al., 2005), and some pesticides, such as triallates, have no effect at all (see, e.g., Sathiakumar et al., 2004).

2.1.3 Diabetes

More recent studies look for a link between diabetes risk and pesticide exposure. Exposure to organochlorine compounds is associated with increased prevalence of diabetes (Cox et al., 2007) as well as handling organophosphate insecticides (Montgomery et al., 2008). All the studies in this section find a significant association between pesticide exposure and diabetes, but the number of studies (3) is not large enough to formulate a general conclusion.

2.1.4 Respiratory diseases

There were 13 studies about respiratory diseases and its relation to pesticide exposure. Most of them suggest an increased risk of respiratory diseases such as rhinitis (see, Slager et al., 2009), asthma (Hoppin et al., 2009), bronchitis (Hoppin et al., 2007c), farmer's lung[3] (Hoppin et al., 2007a), and wheeze (Hoppin et al., 2002). However, Fieten et al. (2009) highlights the fact that it is not possible to establish a causal relationship, and 2 studies do not support a significant association (see, e.g., Boers et al., 2008).

2.1.5 Women specific disorders

There were 5 studies in which the authors analyze the relationship between pesticide exposure and women specific disorders. Regarding the age at menopause the results are mixed: Farr et al. (2006) finds that exposure is associated with a higher age at women's menopause, while Akkina et al. (2004) reports the opposite result. The rest of the studies find that pesticide exposure is a cause of hormonal disorders (see e.g., Farr et al., 2004), but it has no effect on delayed conception in pregnant women (Lauria et al., 2006).

2.1.6 General health, multiple diseases, and others

In this section we included studies that analyze the impact of pesticide exposure on general health or on several diseases (33 studies in total). There are studies of diseases such as hepatitis, dyspnea (Azmi et al., 2006), hearing loss (Crawford et al., 2008), myocardial infarction (Dayton et al., 2010), thyroid disease (Goldner et al., 2010), sperm quality (see, e.g., Perry et al. 2011), and of general measures such

[3] Farmer's lung is a hypersensitivity pneumonitis induced by the inhalation of biologic dusts coming from hay dust or mold spores or other agricultural products.

as human health hazard levels (Murphy et al., 1999) and even suicides (Beard et al., 2011). Some studies highlight that precautionary measures have a significant impact on the relationship between pesticides and disease (Sekiyama et al., 2007), while others find significant interaction effects on health of pesticide exposure and genetic polymorphisms (see, e.g., Lacasaña et al. 2010b). New studies include clever techniques to analyze the effects of pesticides, such as invitro tests (Orton et al., 2011), that enhance the identification of pesticides' effects.[4]

2.2 Health effects of pesticides on members of a community with indirect exposure

There were 64 studies (33.5% of the total) analyzing the impact of pesticides on the health of people with indirect exposure. The subjects of these studies include farmers' family members or people living in rural areas where there is an intensive use of pesticides. The main features of these studies are summarized in Table 2 in the appendix.

2.2.1 Cancer

We found 16 studies on cancer risk and indirect pesticides exposure among which no consensus is reached. For example, while 5 studies find evidence associating pesticide exposure (environmental, or prenatal) with increased risk of childhood leukemia (see, e.g., Ferreira et al. (2013)), Pearce et al. (2006) reports no significant association. In total, there are 3 studies whose evidence suggests no significant association between increased risk of cancer and indirect pesticide exposure (see, e.g., Carreon et al. (2005)), while the rest conclude the opposite, at least for some specific pesticides.

2.2.2 Depression and neurological deficits

There were 11 studies examining whether neurological effects are related to indirect pesticide exposure. The studies look at the risk of depression for spouses of pesticide applicators (Beseler et al., 2006), decreases in neurobehavioral development (see, e.g., Bouchard et al., 2011; Eskenazi et al., 2010; Harari et al., 2010), Parkinson's disease (Yesavage, et al., 2004), and the effect on children's IQ scores (Rauh et al ., 2011) . Even when exposure is indirect the risks of neurological damage may increase, especially for children whose exposure takes place during early stages of fetal development (Eskenazi et al., (2007)).

2.2.3 Diabetes

Only 2 studies were found looking at increased risk of diabetes, and both conclude that pesticide exposure can be associated with an increased risk of diabetes (Everett et al., (2010), Son et al. (2010)). The number of related studies is too small to end up with a general conclusion, but this should motivate more research regarding this type of risk.

2.2.4 Respiratory diseases

It was surprising to find only 1 study looking at the risk of respiratory diseases (Balluz et al. (2000)). This study suggests that the health complaints reported by employees at a health center whose first floor

[4] These laboratory methods allow the researcher to recreate the exposure of specific cells to pesticides, isolating the pure-pesticide effect.

was used as a mixing area for pesticides 20 years before, were precipitated by environmental and psychological factors, more than actual exposure to pesticides. More evidence is needed to come up with a general conclusion.

2.2.5 General health, multiple diseases, and others

There were 32 studies gathered in this section. While some studies find no long-term health risks from potential inhalation of pesticides (Murphy and Haith, 2007), or no association between birth weight and pesticide related activities during early pregnancy (Sathyanarayana et al., 2010), others find significant effects over certain variables, such as body mass index (Burns et al., 2012), endocrine performance (Cecchi et al., 2012), and fetal growth (Wickerham et al., 2012). Recent studies look at the effect of indirect exposure on health outcomes taking into consideration genetic heterogeneity among subjects (see, e.g., Andersen et al., 2012).

2.3 Health effects of pesticides on consumers

Although we expected to have more studies in this section, only 5 studies were found (2.6% of the total) and most of them related to cancer risk. Main results are presented in Table 3 in the appendix.

2.3.1 Cancer

The studies on cancer analyze the risks associated with the consumption of specific products which have some pesticide residues. These consumption products include: fish (Li et al., 2008), water (Buczynska and Szadkowska, 2005), seafood (Moon et al., 2009), and milk or other dairy products (Pandit and Sahu, 2002). In general these studies find a small but statistically significant association between cancer risks and some specific pesticide residues, such as DDT and DDD (dichlorodiphenyldichloroethane), but not for other organochlorines. Specifically PCBs (polychlorinated biphenyls) present a higher risk for consumers (see e.g., Li et al., 2008).

2.3.2 General health, multiple diseases, and others

Only 1 study was found analyzing pesticide residue concentration in vegetables and finds that the risk posed to consumers varies with the season (Bhanti et al., 2007). The winter season has the highest pesticide concentrations in vegetables that might accumulate in the person's body and lead to fatal consequences in the long run. However, only methyl parathion residues have a significant hazard index, so this result is also pesticide-dependent.

2.4 Concluding remarks on health effects and pesticides

Most of the studies found in our literature review are related to the health effects on people with direct exposure (63.9%). Research on people with indirect exposure is harder to find, while research on the risk of pesticide residues for consumers has not really been part of the agenda. Therefore, more research is needed to analyze the potential health effects of pesticides on these subjects.

Regarding data availability, the AHS is the main database used in a large number of the studies (in more than 40% for direct exposure and almost 20% for indirect exposure). More studies collecting data should be conducted to validate the results since the AHS might be influenced by geographical

conditions (only two states are represented: Iowa and North Carolina). Moreover, in the AHS the enrollment process was voluntary and the first approach was made during the training to obtain a license to apply pesticides; as a result, selection bias is one concern. Nevertheless, this study compares favorably with enrollment rates of previous prospective studies, so more initiatives like this should be considered to improve research in this field.

Previous literature surveys (published before 2000) have analyzed only the hazard effects of direct exposure. Do Pico (1992) provides similar conclusions to those we find regarding respiratory diseases: hazardous exposures (including pesticides) can develop into respiratory disorders such as asthma, bronchitis, solo filler's disease, and neuromuscular respiratory failure. Eyer (1995) finds that exposure to high doses of organophosphates with clinically significant intoxications can affect behavior, as well as mental and visual functions, whereas asymptomatic exposure is not connected to neuropsychopathological disorders. These results are similar to what can be concluded from our review: results are pesticide-specific and sometimes depend on the length of the exposure period.

Moreover, Blair et al. (1995) suggests an association between pesticide exposure and cancer. Our review provides stronger conclusions due to new findings on some pesticides increasing the risk of cancer (such as metolachlor and diazinon on lung cancer). The review by Dich et al. (1997) on cancer risk links phenoxy herbicides with soft tissue sarcoma (STS), organochlorine insecticides with STS, non-Hodgkin's lymphoma (NHL), leukemia and lung and breast cancer; organophosphorous are linked with NHL and leukemia; and triazine herbicides with ovarian cancer. This review highlights that "few, if any, of these associations can be considered established and causal" which is a concern that can be extended to many of the studies included in our review.

Arbuckle et al. (1998) suggests an increased risk of fetal deaths associated with pesticides and maternal employment in the agricultural industry. Similarly to one of the studies on pesticide exposure during early pregnancy found in our review (Sathyanarayana et al., 2010), the authors face problems because of the lack of data and methodological issues.

We consider the results of this review consistent with previous ones. The links between health risks and pesticide exposure are pesticide-dependent, the existing findings often disagree, there is a lack of data in order to obtain more reliable results, and there still exist some methodological issues that could be improved to deal with the low number of cases, and potential regional effects or biases.

3 Preference elicitation

In this section we first briefly introduce the monetizing of preferences for health risks. Next we present recent empirical findings on the evaluation of reduced pesticides use. The section ends with some conclusions regarding the empirical research that has been conducted and the way to proceed.

3.1 Health risk valuation

Valuation of pesticide risks is based on the notion that individuals are the best judges of their own welfare. The monetary value of a change in the risk level should therefore reflect the preferences and perceptions of those individuals who are exposed to the risk. This includes both groups who have direct contact with the pesticides, such as producers and farmers, and those who are indirectly

affected, such as consumers of products that contain pesticide residue, people who are exposed when visiting the countryside for recreational purposes, etc.

Since no directly observable prices exist for pesticide health risk reductions, analysts have to rely on non-market evaluation techniques to monetize individuals' preferences.[5] The monetary values obtained using these techniques reflect the individuals' willingness to pay (WTP) and willingness to accept (WTA) for a reduction and increase in the risk level. Thus, since the individual WTP and WTA reflect how much individuals are prepared to give up or how much they require in compensation for a risk change, they reflect individual preferences. Non-market evaluation techniques can, broadly speaking, be classified as revealed- (RP) or stated-preference (SP) techniques. This classification is based on the approach used to obtain information about individuals' preferences. In the former, the RP approach, analysts use the information from individuals' actual decision on existing markets to derive the monetary value of a change in risk. In contrast, in the SP approach, respondents are asked to make decisions in constructed markets where the scenarios are hypothetical. Both approaches have their strengths and weaknesses.

When using market data to monetize preferences for non-market goods (or bads) the analysts observe behavior on markets related to the good of interest. Individuals' decisions then reveal their preferences for the good of interest. Economists often prefer the RP to the SP approach since it is based on actual decisions. Not only because an observed choice reveals that the individual prefers his choice to other available alternatives, but also because many decisions are made repeatedly. Thus, individuals learn by experience and have incentives to seek information and to be well-informed about the decision alternatives available. An example where the RP approach has been used to monetize health preferences is car owners' decisions on optimal car safety level (Atkinson and Halvorsen, 1990; Andersson, 2005). The premium paid for a safer car reflects the WTP to reduce risk exposure (everything else equal). Moreover, the RP approach has been used extensively to analyze and derive preferences for safety in the labor market where workers demand for compensation to accept riskier jobs (Viscusi and Aldy, 2003). However, even if the RP approach has its strengths it also has its weaknesses. It relies on the assumption that individuals make well-informed decision and that the analyst has knowledge about the information and decision alternatives that individuals face when making their decisions. Market data availability is crucial; obtaining market data for some goods may not be possible, while markets may not exist for others.

The weaknesses of the RP approach are the strengths of the SP approach. Since the SP approach is based on a hypothetical market setting, the analyst has the freedom to tailor the scenario to elicit preferences for the specific question he is interested in. This means that: (i) the analyst knows about the information and the decision alternatives the respondent has access to, (ii) any (at least in theory) market can be constructed, and therefore (iii) data will be available. This does not mean that the SP approach does not have its weaknesses, though. Its main weakness is the hypothetical nature of the market, which is also the reason why many economists prefer the RP approach, and the fact that

[5] There is now a huge literature on the evaluation of non-market goods. We aim at keeping the references in this section to a minimum, and for those interested in an introduction to the topic we suggest Bateman et al. (2002) and Haab and McDonnel (2003), but there are many other good textbooks on the subject, including books on BCA such as Boardman et al. (2011).

respondents are often asked to state their preferences for goods that are unfamiliar to them as a "market good", i.e. they are not used to trade them in a market. The combination of the hypothetic market and an unfamiliar good may lead to respondents not stating their "true" preferences. Due to the hypothetical nature respondents may not have incentives to make the effort to understand the scenario and its consequences, and may instead have incentives to answer strategically, i.e. in a way that is in their own interest. For instance, they may state a higher WTP than their true one since they know that they will not actually have to pay the amount. However, despite these weaknesses SP methods have been used in many studies to evaluate health risks in many areas due to the strengths mentioned above (Hammitt and Graham, 1999; Florax et al., 2005; Andersson and Treich, 2011; Kling, et al., 2012).

Health risks affect individual utility and therefore the WTP estimated, either with RP or SP techniques, reflects a use value to the individuals. In the literature on non-market evaluation the distinction between use and non-use value is an important one. The latter refers to goods for which individuals have a positive WTP, despite the fact that they have no personal direct gain from the goods. For instance, individuals may be prepare to pay some amount of money to save endangered species even if they have no commercial interest (e.g. organizing commercial safari trips), or have any chance of seeing the animal or benefit from it in any other way. The distinction is relevant for pesticide risks, since they affect both individual health and the environment. Regarding the latter, the effects from pesticide use can have both use value, such as commercial values for recreation, fishing, etc., but also non-use values, for instance a belief about an intrinsic value for species and habitats, or a WTP to preserve the environment for future generations. An important difference between the two is that non-use values are typically estimated with SP methods, since the RP approach relies on the information on individuals' behavior in markets.

In addition to RP and SP studies, WTP can be elicited in experiments. These experiments often take place in a laboratory with constructed markets where subjects must make a commitment, which usually involves a money transfer. Experiments are usually not used to derive monetary values for policy purpose, e.g. values to be used in BCA, but are instead used to examine hypothesis about behavior or methodological issues.

3.2 Evidence from the literature on the evaluation of pesticide risks

The risk of pesticides to human health has been of public concern since the 1970s. The benefits of pesticide use then started being weighed against their costs, which led to research being conducted monetizing the benefits and the costs. Most of the early work was conducted in the US and after only a few studies were carried out in the 1980s, there was a rapid growth in the number of studies in the 1990s. This early work was reviewed and analyzed in Florax et al. (2005) and Travisi et al. (2006). We therefore restrict our review to studies published from 2000 to the spring of 2013 (when the review was conducted). We used the same search tool as for the review of health effects from pesticides, i.e. EBSCOhost, and we restricted the search to published peer-reviewed articles in scientific journals. We used the search criteria "pesticide" and "willingness" in either the title or the abstract. We do not restrict our review to only studies eliciting WTP related to human health risk. Instead we include all

relevant studies eliciting individual WTP to reduce or eliminate pesticides use. We found 32 studies that met our requirements and were studies eliciting monetary values to reduce pesticides usage.

The studies are presented in alphabetical order in Table 4 in the appendix. The studies are classified: (i) according to origin, i.e. the country in which the study was conducted, (ii) what type of actor, which has been defined as either farmer or consumer, (iii) which study type, where the study is defined as either RP or SP, but also the actual technique is specified, and (iv) what type of good WTP is elicited for. Regarding type of actor, some studies did not specify that the sample represented consumers, but instead the general public was the target. We have coded this group as consumers. Regarding the type of good we have grouped the studies into health (H), environment (E), organic (O), and miscellaneous (M). Miscellaneous could, e.g., refer to "non-GM" (genetically modified) goods. The table reveals that WTP reflects more than one type of good in several studies. The final column of the table describes how WTP was defined in the study, e.g. as a price premium for a pesticide free product compared with a conventional product.

The review shows that it is still in the US where most studies are being conducted. There has been a growing interest in Europe, though; we found 11 studies conducted primarily in the UK and Italy compared with one study conducted in the UK before 2000 (Florax et al., 2005; Travisi et al., 2006). The review also reveals that most studies are conducted with the aim of eliciting consumers' preferences. Only 6 of the 32 studies focused on farmers. Moreover, there seems to be more interest in farmers' preferences in the developing world; 5 out of the 7 studies conducted in Turkey, Ghana, Nepal, Nicaragua, Pakistan, Thailand, and the Philippines are on farmers' preferences compared to only one study in the US, Canada, and western Europe. Further, a huge majority of the studies were conducted using SP compared to RP techniques or experiments, 25 out of 32, with two studies using both SP and RP techniques or experiments. Finally, three types of "goods" dominate, a reduced pesticide use that reduces health risk, health risk and environmental effects, and organic produce.

The almost non-existing interest in farmers' preferences in the developed countries could reflect the view that the farmers in these countries are well protected by available safety equipment and also by legislation. It could also be argued that they are well informed, and therefore make well-informed decision on their optimal risk exposure without any need for additional government intervention. Indeed, the only study conducted in these countries used RP data to examine farmers' preferences for reduced pesticide use with a positive impact on both health and the environment (Sydorovych and Michele, 2008). The studies on farmers in the developing world focused on the health effects (Atreya et al., 2012; Garming and Waibel, 2009; Khan, 2009; Palis et al., 2006), with one exception that elicited WTP for a more efficient pesticide (Al-Hassan et al., 2010). Focusing on the studies eliciting WTP for health, Garming and Waibel (2009) and Khan (2009) employed the contingent valuation method (CVM) which is an SP method that directly ask respondents about their WTP, either as an open-ended question where respondents state their maximum WTP, or in a referendum format where respondents are asked to agree or not to pay a specified bid.[6] Both studies asked respondents whether they were prepared to pay a premium for a pesticide that was safer to use, either compared

[6] This description is highly simplified. For a more comprehensive description of the design of CVM surveys see, e.g., Bateman et al. (2002).

to a "standard pesticide" or the one that they currently used. Palis et al. (2006) instead created an experiment where farmers and laborers in the agricultural sector were offered protective equipment that they could buy, whereas Atreya et al. (2012) employed both the CVM and information on the farmers' reported expenditures on different safety measures.

The studies focusing on consumers' preferences in most cases asked respondents if they were prepared to pay a price premium for either a specified product, such as tomatoes or a loaf of bread, or a "basket of products". All but four studies (in one case a lab experiment (Marette et al., 2012)) were based on SP data and often framed as either organic or "non-GM", i.e. it was not specified that the good would reduce health risks and/or have a positive impact on the environment (see, e.g., Balcombe et al., 2007; Canavari and Nayga, 2009; Loureiro et al., 2002). Hence, in those studies respondents' WTP reflect their preferences for organic and non-GM food. This is also the case for the one RP study that was conducted which elicited parents' WTP for organic food for their babies (Maguire et al., 2004). Using RP data it can be difficult to disentangle the different attributes of the goods that influence buyers' WTP, i.e. it is hard to know if consumers of organic food buy it for the health benefit, because it is more environment friendly, the taste, or something else. For instance in the experiment in Nielsen (2013) subjects were asked to choose between fries from conventional or GM potatoes, where the latter were described as "environmentally friendlier". In a follow-up survey many subjects reported, though, that their decision about the GM fries was influenced by health concerns. The SP approach offers here an advantage compared to the RP approach, since it is possible to specify what the positive effects are for reduced pesticide use and then to derive specific monetary values for each effect. Many of the CVM studies also framed the scenario such that respondents were informed that they would either benefit from better health or environment from reduced pesticide use (e.g., Akgüngör et al., 2001; Hammitt and Haninger, 2010). Other SP studies employed the choice experiment (CE) technique, which is based on a multiattribute scenario which enables the analyst to derive monetary values for the chosen attribute. Hence, by having respondents choosing between products with different levels of the attribute, their WTP for each attribute can be estimated. For instance, Balcombe et al. (2009) and Travisi and Nijkamp (2008) used the CE technique to derive monetary values for different types of pesticides and for both health and environmental attributes, respectively.

The description of the WTP in the final column of Table 4 shows that, in general, the WTP is either defined as a price premium of a specified product or basket of products, or that no WTP was specified (NA). The objective of many studies (e.g., Canavari and Nayga, 2009; Govindasamy et al., 2001) was not to estimate monetary values for policy use (BCA, taxation, etc.), but to examine whether consumers' preferences are heterogeneous and whether respondents are willing to pay more for produce: (1) that contain, or are produced with fewer, pesticides, and (2) are organic, non-GM, etc. Thus, "not available" (NA) defines studies in which the respondents were not provided any reference price such that a monetary value could be derived or when authors did not reported any monetary values as in the experiments by Palis et al. (2006) and Nielsen (2013).

3.3 Concluding remarks on preference elicitation to reduce pesticide risks

The review reveals heterogeneity in goods used, and therefore in monetary values estimated. The design of the surveys suggests that the main objective of these studies is not to derive monetary values

that reflect individual preferences that could be used for policy purposes. Instead, the two main objectives seem to be to examine whether: (i) individuals have a positive WTP for produce with less pesticide, with positive benefits for the health and the environment, or for organic or non-GM produce, and (ii) preferences are heterogeneous in the population. The evidence is strong that WTP is positive, both among consumers and farmers. There is also some evidence that preferences vary with individual characteristics, such as gender, age, income, and experience with pesticides.

Whereas the analysis of the effects from pesticides reported in section 2 mainly focused on specific pesticides and health effects, the WTP literature on pesticides have mainly elicited individuals' preferences based on non-specified pesticide use with general health effects. Moreover, whereas the health studies focused on people with direct exposure, consumers' preferences were of main interest in the WTP studies. These differences between the health and WTP literature are expected. The former is interested in the direct effect from specific pesticides on well-defined, or in some studies general, health effects to examine which pesticides are dangerous to human health. This is also of interest to analysts estimating WTP, but it can be hard to identity the effect from single pesticides using market data (RP) and to communicate the health effects from single pesticides in SP studies. Instead most of the WTP studies describe a general health impairment from pesticide exposure, usually related to consuming either "pesticide safe/free" food (e.g., Akgüngör et al., 2001; Cranfield and Magnusson, 2003; Posri et al., 2007), or organic food (e.g., Canavari et al., 2005; Haghiri and McNamara, 2007).

The research on WTP to reduce health risks from pesticide use contrasts health risk evaluation in many other fields such as workplace safety (Viscusi and Aldy, 2003) and traffic safety (Andersson and Treich, 2011). In those studies the risk reduction is defined as a change in the probability of an adverse health effect, injury or fatality, which enables the analyst to normalize the derived WTP to a "standardized value", such as the value of a statistical life (VSL) for a reduction in fatality risk (Jones-Lee, 1976, Cropper et al., 2011). The only study following this approach was Hammitt and Haninger (2010) which estimated the VSL. Two other studies, Mourato et al. (2000) and Travisi and Nijkamp (2008), followed a similar approach and valued cases of illness due to pesticide exposure. They both estimated WTP for a non-fatal outcome with the illness specified so that all respondents had the same information regarding the health impairment. The advantage of using a standardized value is that it can be compared between studies, and that it is better suited for benefits transfers.

The reason most WTP studies have focused on consumers' preferences instead of those individuals with direct exposure is probably because the latter group is relatively small, especially in developed countries. Even if the individual cost from impaired health is substantial for a farmer compared with a consumer, the total social cost from the second group is likely to be substantially higher due the large number of individuals in the second group. From a social perspective it therefore seems reasonable to continue focusing on consumer preferences, even if the preferences of those with direct exposure shall not be neglected.

It is established in the health literature that pesticide exposure has often a negative impact on individual health, even if it varies with type of pesticide. It is also established from the WTP literature that individuals are prepared to pay a premium for pesticide free or "safe" produce. We believe, though, that it is necessary for the WTP research to be more precise about what the WTP reflects.

When eliciting WTP for organic produce, for instance, the premium paid probably measure taste, and environmental and health concerns, but maybe also other characteristics such as supporting locally grown produce, etc. Moreover, even if we would know which attributes consumers pay for, we would not know their shares of the WTP. Thus, to obtain monetary values that can be used for benefits transfer, i.e. that are not too context specific, it is necessary that the health outcomes are precise and/or that WTP is elicited for given amounts of specific pesticides. Hammitt and Haninger (2010) excluded the use of organic produce when eliciting their WTP for a fatality risk to minimize the risk of respondents also weighing in environmental and other characteristics when answering the WTP question and thereby obtaining a confounded measure of WTP. Chalak et al. (2008) in their choice sets specified the change of use from different types of pesticides. This approach does not directly elicit respondents' WTP for specific health outcomes, but it allows the analysts to elicit preferences for specific types of pesticides and based on the evidence from the health literature the health effects due to changes in pesticide levels can be estimated by the analyst.

4 Pesticides and policy

4.1 Benefit-cost analysis

In most countries, pesticides sales and use must be approved by public authorities. In the US, the EPA is responsible for pesticide regulation under the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) and the Food Quality Protection Act (FQPA) (see for instance Berwald et al. (2006) for a presentation of pesticide regulation and policy use of economic analysis in the US). In principle, the EPA regulates pesticides to ensure that these products do not pose adverse effects to humans or the environment, whereas FIFRA explicitly advocates a balancing criterion in the sense that pesticides use is permitted absent "unreasonable risks" to human life or the environment. This balancing criterion seems consistent with the use of BCA, which has been a legal obligation in the US since the early 1980s (Sunstein, 2002).[7] But in practice EPA through FIFRA essentially seeks to reduce exposures to carcinogenic pesticides below an individual risk level of 1 in 1 million, with some tolerance for higher levels. Similarly EPA through FQPA admits tolerances for carcinogenic pesticide residues in food such that there is a "reasonable certainty that no harm will result from aggregate exposure to the pesticide". A similar approach is adopted in Europe where for instance pesticide residues on crops are monitored through a maximum residue level (MRL) (EFSA, 2013).

Therefore, as in many other areas of risk regulation, the "de minimis risk" approach is the predominant approach influencing pesticide regulation (Adler, 2005). Essentially, this approach means that policy makers are instructed by their authorities to ensure that the probability of some bad outcome is below a low probability threshold. An advantage of this approach is that it seems simple, "fair", and can be easily translated into law. This approach might also be judged convenient politically since it often suggests that safety is ensured below the critical threshold. Obviously, this is often an

[7] We must add that BCA has been required except when prohibited by law or interpreted that way by administrators. For instance, the well-known and extremely controversial Delaney Clause precluded the use of BCA considerations for any cancer-causing additive. Pesticides were removed from the Delaney clause in 1996.

illusion of safety since many pollutants do not have thresholds below which they have no effects. Moreover, as we discussed above, the threshold might be too ambitious and impossible to reach, and thus not implemented in practice. However, perhaps the most fundamental reason for why the de minimis risk approach is problematic is an economic one. Indeed, under a de minimis risk approach, the cost of regulation is not counted, and thus this cost cannot be compared to the benefit of the risk reduction target. To illustrate the inefficiency, consider that for some pesticide it is extremely costly to reach the threshold, while for another pesticide it is not so costly, and it might even not be very costly to reduce the risk further. Moreover, imagine that the first pesticide only concerns a few people while the second concerns millions of people. So targeting a uniform probability threshold of risk exposure across pesticides and populations seems particularly inefficient in general.

These observations relate to long-standing concerns about risk assessment practices. A major concern applies to the related "individual risk" approach. This approach typically computes a threshold probability using a hypothetical individual model with unrealistic assumptions like high exposure, high ingestion rates, low body rate etc. Besides, ad hoc "safety factors" are often employed when some causal relationships are uncertain. Moreover, when some parameter is unknown, extreme percentiles of the probability distribution are often used. This implies that risk assessment practices may induce a conservatism bias. For instance, Belzer (1991) estimated that the de minimis risk estimated by the EPA for dioxin was 5,000 times greater than its expected value, and that of perchloroethylene was 35,000 times greater than its expected value. As a consequence it has been suggested that the de minimis approach may lead us to go much too far in terms of regulation. To illustrate, we mention here the spectacular figure of the BCA about the regulation of atrazine/alachlor drinking water standards which revealed a cost per life saved of US$92,000 million. This is the most inefficient policy of all policies reported in Sunstein (2002) that might well be explained by a conservatism bias.

Cropper et al. (1992) develop an econometric study of the overall EPA pesticide cancelation policy between 1975 and 1989. Their results offer some good news for economists. Indeed, they show that the probability of cancelation of pesticides is increasing in environmental and health risk and decreasing in economic benefits of pesticide use. However, the study also shows that EPA uses an implicit VSL for pesticide applicators of US$35 million while the implicit VSL for consumers is only US$60,000. Interestingly, EPA recommends elsewhere in BCA guidelines to policy makers to use VSL in the range of US$1 to US$10 million. Therefore, the estimates of Cropper et al. (1992) show inconsistency across EPA practices. Note that this inconsistency can be explained by EPA targeting higher risks. Indeed, the risk of exposure to a pesticide applicator has been estimated to be about 15 times higher than that of a consumer. This observation is consistent with the de minimis risk approach discussed above, in which regulators target a maximal risk but do not compare the relative benefits and costs of changing the risks. Along similar lines, Van Houtven and Cropper (1996) find evidence that EPA is biased toward minimizing risk for target vulnerable populations, like farm workers.

We now report the results from Pimentel (2005) who provides the only full-fledged BCA of pesticide use that we are aware of. He estimates that the direct benefit and cost for US farmers of using pesticides are respectively US$40 and US$10 billion. Moreover there is an indirect cost for farmers in terms of loss of natural enemies (US$0.5 billion), pesticide resistance (US$1.5 billion) and crop losses (US$1.4 billion). Pimentel (2005) also estimates that indirect environmental losses, mostly

through bird losses, amount to slightly more than US$2 billion. Moreover, he estimates the cost for monitoring and cleaning pesticide-polluted groundwater to about another US$2 billion. Finally, he estimates the health costs, including costs of hospitalization due to poisoning, of outpatient-treating poisonings, pesticide cancers and fatalities (with a VSL of US$3.7 million), to about US$1.3 billion. These results suggest that the use of pesticides is largely beneficial to the society. Nevertheless Pimentel (2005) adds that some costs are ignored or not accurately measured in his BCA, and thus a "complete and long-term [BCA] of pesticide use would reduce the perceived profitability of pesticides". We must also notice that an important limitation in this study is that it assesses the cost and benefit of full elimination of pesticides. In fact, realistic policy options concern the reduction of pesticide use, not its full elimination. Yet, evaluating these policy options require more precise information, typically in the form of dose-response relationships.

Following the brief presentation of the BCA of Pimentel (2005), we highlight some conceptual difficulties to apply BCA to pesticides use and regulation. We only discuss here the case for regulating pesticides for protecting the health risks of farmers, and of consumers, which is the main focus of our review. Indeed, we note that farmers have the choice on whether to purchase and use special equipment to protect them from pesticides. They can also choose crops that are more or less intensive in pesticides use. They can choose not to use pesticides at all, that is, to produce organic food. At the limit, they can even choose another occupation. Clearly, for farmers, there is a cost of using pesticides in terms of the input cost in production and the possible acute and chronic health risks. But there is also benefit of using pesticide which improves productivity and provides a self-insurance against pest uncertainty. But these are essentially private costs and private benefits. Indeed the analysis of Pimentel (2005) indicates that the use of pesticides is strongly profitable to farmers (i.e., a cursory analysis using his data indicates that a US$1 dollar investment in pesticide control returns about US$3 including monetized health costs). It is no surprise that the main opponent to the major revision of workers' protection standard for agricultural pesticides issued by EPA was the Department of Agriculture (True, 1997). It is not clear that there are solid arguments for government intervention in pesticides control on the ground of protecting farmers' health alone. This observation is in turn consistent with the high implicit VSL of US$35 million for pesticide applicators estimated by Cropper et al. (1992), which suggests that regulation went too far.

What about the case for regulating pesticides for protecting the health of consumers? It may well be that the society gain in terms of cheaper food is much higher than the monetized health cost of pesticides. Moreover, we must remember that consumers have the option of limiting pesticide exposure by purchasing particular, often more expensive, products (e.g., organic produces, bottled water). Therefore, one must recognize that consumers have the opportunity to make most of their money-health trade off privately. Again, this suggests that there is no strong argument for public intervention. Instead, public intervention prevents the freedom of choice, and the heterogeneity of consumers' tastes and risk preferences from being expressed on the market.

Therefore, we argue the rationale for government intervention in the regulation of pesticides should be better articulated. Obviously, a thorough discussion of government intervention should also address the possible externalities of the health effects of pesticides through the collective health

system, as well as the possible role of the government through the development of research programs and of information campaigns.

4.2 Risk perception

Despite decades of research about pesticides, there is still high uncertainty about health effects of pesticides. It is well known that the toxicological studies based on animals require strong extrapolative assumptions. Epidemiological studies that we reviewed in section 2 are often sporadic and they often reveal disagreement; indeed we have seen that it happens most often that some studies find a significant effect while other studies do not find any effect. In addition, pesticides involve complex cumulative and synergetic effects that are seldom accounted for. Also, we recall that pesticide regulation is a high stake and politically sensitive issue, and there is often concern about possible conflict of interests faced by scientists leading the studies. As a result of these observations, we can hardly expect lay people like farmers or consumers, to be well informed and confident in their beliefs about the health effects due to pesticides.

To illustrate possible problems of information about health risks due to pesticides, we briefly summarize the controversies about the regulation of Alar, a pesticide long used when growing apples in the US. We use the summary provided by Kuran and Sunstein (1999). In 1989, preliminary toxicological results showed that rodents exposed to Alar have a higher than normal incidence of tumors. On the basis of this study the Natural Resources Defense Council (NRDC) made a series of pessimistic allegations, and a television show (i.e., 60 Minutes) publicized those allegations. This instigated public outcry, including actress Meryl Streep, who founded an activist group called “Mothers and Others for Pesticides Limits”. The EPA then reviewed the allegations concluding that the risk was largely exaggerated by NRDC. Nevertheless, many people treated apples as highly toxic substances, and the demand for apples plummeted. The damage to the apple industry was enormous; in the Washington State alone it was about US$125 million in losses in half a year. Moreover, EPA eventually developed further risk assessment studies and concluded that the risk was even lower than initially estimated in its first study, and about lower by a factor 60 compared to the NRDC allegations. A 1991 Science editorial stated that “a clearly dubious report about possible carcinogenicity by a special interest group was hyped by a news organization without the simplest checks on its reliability or documentation”. Finally, a United Nation report concluded that Alar is not dangerous to people.

We believe that this story is symptomatic of some problems that may arise under high uncertainty conditions. The public lacks information about pesticide risks. The risks are small, and small risks are usually overestimated. Chemical risks are also overestimated compared to natural risks. Moreover, food is a special good, which may trigger special emotion and may explain over-reaction to arrival of information. Availability heuristics also play a role when the risk is made salient and popular. Besides, scientific complexity and trust issues in experts and public authorities may make it difficult to inform the public, and mitigate warning efforts. With an important gap of knowledge between experts and the public, what should the regulator do?

This question is considered by Portney’s (1992) dilemma of Happyville, an imaginary society where people believe that the drinking water is contaminated while all experts agree it is harmless. Moreover, any attempt to inform people that the water is safe has had no effect. People remain

anxious and they urge the Director of the Environmental Protection agency of Happyville to invest in a water cleanup technology. What should the Director do? The answer may be framed as a choice between populism and paternalism. A populist Director would invest a cleanup technology on the ground that worried people would "feel" protected. This is consistent with the usual welfarist approach. But a paternalistic Director computing the benefit using experts' beliefs would not invest in a cleanup technology on the ground that there is an opportunity cost of preventing a phantom risk; in other words, one could save more lives by preventing other risks. The choice is difficult. We must add, moreover, that this choice may be even more subtle because it must account for the citizens' behavioral responses to the policy. Indeed, Salanie and Treich (2009) showed that it may well be optimal for a paternalist Director to invest as well (i.e., like the populist) in the cleanup technology. The reason is an "encouragement effect" that leads citizens to increase their risk exposure (which is judged too low from the paternalistic Director viewpoint). Going back to the Alar example, this "encouragement effect" means that a paternalistic regulator may want to over invest in safety under pessimistic risk beliefs in order to mitigate the decrease in the demand of apples. This may well provide a new justification for pesticide regulation. But how to treat this paternalistic justification within BCA is a difficult question. Indeed, this justification for public intervention is not consistent with standard preference-based methods in economics.

5 Conclusions

We have provided two literature reviews on recent scientific findings on health effects and on preference valuation of health risks related to pesticides use, and a review and discussion of policies related to pesticides and difficulties of evaluating them. Central to our reviews have been the critical discussions of the different findings.

Our health literature review showed that most research focused on individuals with direct exposure to pesticides, e.g. farmers. Since research on people with indirect exposure was harder to find, and since most individuals belong to this group, more research should be conducted analyzing the potential health effects of pesticides on consumers and/or people with non-direct contact to pesticides. Our review also shows that for many of the health outcomes results are ambiguous; some studies find evidence of pesticides exposure leading to health impairments, whereas other do not. Further, a weakness in the literature on pesticides exposure and health effects is that many studies are based on the same AHS data set. Since context and geographical conditions may influence results, future studies on other data sources will provide important information about whether the findings based on the AHS can be confirmed or rejected.

In our review of the literature on individuals' preferences to reduce pesticides risks we found a large heterogeneity in the goods used and monetary values estimated. Based on our review we conclude that the main objective of the studies eliciting individual preferences, with some few exceptions, has not been to estimate WTP that can be used for policy purpose, e.g. in a BCA, but to examine whether individuals have a positive WTP for produce with less pesticides, or for organic or non-GM produce. This suggests that these studies focus largely on a marketing/brand/sales perspective rather than public health perspective. We also found that a difference between the health and WTP literature was that whereas the former focused on individuals with direct exposure to pesticides, the

latter focused on those with non-direct exposure, such as consumers. On the one hand this focus arguably makes sense from a general welfare analysis perspective, since the number of individuals exposed among consumers and the public is substantially larger than direct exposed individuals. On the other hand, the evidence of health effects from pesticides among those who are not directly exposed, e.g. from pesticide residue in food, is sometimes weak or non-existing, which suggests individual WTP is based on uninformed risk levels.

Finally, in the previous section, we have briefly reviewed pesticides policies. We have indicated that existing policies are often based on a "de minimis risk" approach, and as a result they may not be consistent with economic analysis. Moreover, the rationale for pesticide regulation must be clarified. Indeed, benefits and costs related to pesticides use on farmers or on consumers essentially have a private nature, so that the very existence of market failures is not clear. We have also discussed the issue of risk perceptions. Empirical evidence indeed suggests that consumers have different perceptions about health risks from those held by our best scientific experts or by our policy makers. Yet, how to treat this divergence of risk perceptions within BCA is not clear.

Our final observation is that our reviews, which involve highly specialized research in different disciplines, also illustrate that interactions across disciplines are limited. This observation is exemplified by the different scope of the studies in our reviews. Indeed, studies in health are typically concerned with pesticide-specific effects on farmers while studies in economics are typically concerned with non-specified pesticide effects on consumers. This relates to Zilberman and Millock (1997)'s observation that pesticide policies would improve if economic literacy among natural scientists and policymakers would increase. Interestingly, Zilberman and Millock (1997) also observe that pesticide regulation at EPA are typically separated into two units, one for risk assessment and the other for economics (and we note that a similar separation holds for the regulation of chemicals in the European Union). Arguably, this institutional organization of the policy evaluation process does not favor interdisciplinary collaboration. We thus conclude by recalling that we typically view BCA as way to incorporate knowledge from a variety of natural and health sciences, and to determine trade-offs with economic costs across various policy options. Our paper should thus be seen as a modest step toward such an interdisciplinary research effort that may help to address the immense challenge posed by pesticides regulation.

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Appendix

Table 1: Direct exposure

Reference	Main conclusions	Data	Quantitative Results
CANCER			
Alavanja et al. (2003)	Significant prostate cancer risk associated to pesticide exposure. Interaction of family history with pesticide exposure is observed.	Agricultural Health Study: 55,332 male pesticide applicators from Iowa and North Carolina.	Prostate cancer standardized incidence ratio of 1.14 (95%CI: 1.05-1.24).
Alavanja et al. (2004)	Non-significant lung cancer risk is associated to pesticide exposure in general (due in large part to low cigarette smoking prevalence). Two herbicides (metolachlor and pendimethalin) and two insecticides (chlorpyrifos and diazinon) show significant exposure response for lung cancer.	Agricultural Health Study: 57,284 male pesticide applicators and 32,333 spouses of applicators from Iowa and North Carolina.	Lung cancer standardized incidence ratio of 0.44 (95%CI: 0.39-0.49). Significant exposure-response trends: Metolachlor P(trend)=0.0002 Pendimethalin: P(trend)=0.003 Chlorpyrifos: P(trend)=0.03 Diazinon: P(trend)=0.04
Andreotti et al. (2009)	Organochlorines are not associated with an excess risk of pancreatic cancer, while herbicides might be associated with it.	Agricultural Health Study: 93 incident cases, and 82,503 cancer-free controls.	Significant odd ratios for: Pendimethalin: 3.00 (95%CI: 1.3-7.2) EPTC: 2.56 (95%CI: 1.1-5.4)
Andreotti et al. (2010)	Obesity and pesticide use are associated with increased risk of cancer. The results suggest that certain pesticides may modify the effects of obesity on the risks of colon and lung cancer.	Agricultural Health Study: 39,628 men and 28,319 women from Iowa and North Carolina.	Hazard ratios: BMI (body mass index) and colon cancer: 1.05; BMI and lung cancer: 0.92; BMI and colon cancer for users of carbofuran:1.10; BMI and lung cancer for users of carbofuran: 1.01 (not significant)
Baris et al. (2004)	There is a modest increase in risk of multiple myeloma due to pesticide exposure. Certain animal viruses may be involved in the increasing risk.	Data collected: 573 myeloma cases and 2131 control cases, and a job-exposure matrix developed to calculate the exposure levels.	Significant odds ratios for sheep farm workers (1.7), pharmacists, dieticians and therapists (6.1), service occupations (1.3), but not for farmers.
Barry et al. (2012)	The findings suggest a significant role of the nucleotide excision repair in the increased risk of prostate cancer risk due to pesticide exposure.	Agricultural Health Study: 776 prostate cancer cases and 1444 male controls. Focus on white pesticide applicators.	Significant odds ratio for prostate cancer risk with high fonofos use (2.98), and high carbofuran use (2.01) for specific nucleotide excision repairs.
Beane et al. (2005)	Routine application of diazinon (using lifetime exposure days) shows increased risks with significant tests for trend for lung cancer and leukemia.	Agricultural Health Study: 23,106 male applicators in Iowa and North Carolina.	Rate ratios: Lung Cancer: 2.41 (95% CI: 1.31-4.43) Leukemia: 3.36 (95% CI: 1.08-10.49)
Beane et al. (2011)	No consistent evidence to associate atrazine use and any cancer. For some cancers, results are based on relatively small numbers, which reduces the robustness of the results.	Agricultural Health Study: 57,310 pesticide applicators; from which 36,357 users of atrazine, with 3,146 cancer cases (in Iowa and North Carolina).	95% CI of Relative Risks (RRs) for all type of cancers, by pesticide exposure quartiles: Q1 (reference) ; Q2: [0.93 - 1.13]; Q3: [0.90 - 1.10] ; Q4 [0.96 - 1.18].
Datta et al. (2006)	The use of organic arsenicals as pesticides in mineral soils may not be a safe practice from a human health risk perspective.	Static incubation study to estimate soil speciation and in-vitro bioavailability of arsenic as a function of soil properties.	Excess Cancer Risk (ECR) with Tobosa soil (after 12 months using bioavailability values) Concentration of 45: 0.23 ECR Concentration of 225: 1.59 ECR Concentration of 450: 3.78 ECR
De Brito et al. (2005)	There is an almost two times higher probability of cancer development among rural workers, with a significant relative risk for agriculture workers.	Data collected: 68 cancer cases and 60 controls, obtained from the Amaral Carvalho Hospital, at the city of Jau from 2000 to 2002.	Relative risk ratio for agriculture workers (versus other occupations) of 1.6 (CI are not reported).

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Reference	Main conclusions	Data	Quantitative Results
De Roos et al. (2005)	Glyphosate exposure is not associated with cancer incidence overall or with most of the cancer subtypes studied.	Agricultural Health Study: 57,311 licensed pesticide applicators in Iowa and North Carolina.	Rate ratios adjusted for age: All cancers: 1.0 (95%CI: 0.9-1.1) Melanoma: 1.8 (95% CI: 1.0-3.4)
Dennis et al. (2010)	There is an association between several pesticides and melanoma, which provides support for considering agricultural chemicals as another source of melanoma risk.	Agricultural Health Study: 52,394 private pesticide applicators, 4,916 licensed commercial applicators, and 32,347 spouses of applicators in Iowa and North Carolina.	95% CI of odds ratios: Maneb (>63days): 1.2 - 4.9 Parathion (>56days):1.3 - 4.4 Carbaryl (>56days): 1.1 - 2.5
Greenburg et al. (2008)	The results of this study do not provide evidence of an increased risk for the development of cancer due to captan exposure	Agricultural Health Study: 48,986 private and commercial pesticide applicators in Iowa and North Carolina.	No significant rate ratios between highest tertile of captan exposure and development of all cancers: RR = 0.89 (95% CI: 0.71 - 1.13)
Hardell et al. (2002)	Increased risk of non-Hodgkin lymphoma and hairy cell leukemia are found following an univariate analysis associated with exposure to herbicides, insecticides, fungicides, and impregnating agents.	Data collected by questionnaires: pooled analysis with 515 cases and 1141 controls.	Significant odds ratios for: herbicides (1.75), insecticides (1.43), fungicides (3.11), impregnating agents (1.48).
Hou et al. (2004)	Results suggest an association between pendimethalin exposure and incidence of rectum and lung cancer.	Agricultural Health Study: 9,089 pendimethalin-exposed cases and 26,836 controls, enrolled between 1993 and 1997.	Significant increased RRs for pendimethalin-exposed cases of: 3.5 (for rectum cancer); 5.2 (for lung cancer and upper half of highest tertile exposure days).
Hughes et al. (2011)	Significant association between prostate cancer and fonofos exposure on men with family history of prostate cancer, suggesting a role of BER genetic variation in pesticide-associated prostate cancer risk.	Agricultural Health Study: 776 prostate cancer cases, and 1,444 controls (in Iowa and North Carolina).	95% CI of odds ratios of prostate cancer risk and: rs1983132 (low use): 0.91 - 3.01 rs1983132 (high use): 1.78 - 5.92
Kang et al. (2008)	Trifluralin exposure is not associated with cancer incidence overall however, there is an excess of colon cancer in the exposure category of higher half of highest tertile (might be a chance finding)	Agricultural Health Study: 50,127 private and commercial pesticide applicators in Iowa and North Carolina.	Rate ratios (referent = non-exposed): All cancers: 0.99 (95% CI: 0.83-1.17) Colon cancer: 1.76 (95% CI: 1.05-2.95)
Karunanayake et al. (2012)	Results suggest an increased risk of Hodgkin lymphoma associated with specific pesticides.	Data collected: Mailed questionnaire followed by a telephone interview with 316 Hodgkin lymphoma cases and 1506 controls in 6 regions of Canada	Increased risk for Hodgkin lymphoma associated to exposure to insecticide chlorpyrifos (1.19, 95% CI: 1.03,1.37), and other factors, such as family history of cancer and previous diagnosis of shingles.
Kokouva et al. (2011)	Lymphohaematopoietic cancers (LHC) are associated with pesticide exposures. Smoking or eating during pesticide application are modifying factors that increase the risk.	Data collected: 354 confirmed LHC cases (diagnosed from 2004 to 2006) and 455 controls, located at Larissa, Greece.	Significant odds ratios of pesticide exposure for: total LHC cases (1.46), myelodysplastic syndrome (1.87), and leukaemia (2.14).
Koutros et al. (2009)	Significant trends in risk with increasing lifetime exposure to imazethapyr are found for bladder cancer and colon cancer.	Agricultural Health Study: 49,398 licensed pesticide applicators in the US.	Rate ratios: Bladder Cancer: 2.37 (95% CI: 1.20-4.68) Colon Cancer: 1.78 (95% CI: 1.08-2.93)

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Reference	Main conclusions	Data	Quantitative Results
Lee et al. (2004)	There exists an association between alachlor application and incidence of lymphohematopoietic cancers.	Agricultural Health Study: 49,980 pesticide applicators in Iowa and North Carolina.	Rate ratios (using intensity-weighted exposure-days): All lymphohematopoietic cancers: 2.42 (95% CI: 1.0-5.89) P(trend)= 0.03
Lee et al. (2004a)	The results suggest an association between chlorpyrifos use and incidence of lung cancer.	Agricultural Health Study: 54,383 pesticide applicators in Iowa and North Carolina.	Rate ratios (adjusting for other pesticide exposures and demographic factors): Lung cancer: 2.18 (95% CI: 1.31-3.64)
Lee et al. (2006)	No significant association is found between pesticide exposures and mortality of lung cancer in the population of Leningrad	Data collected: 540 lung cancer cases and 582 controls obtained from the registers of people dying in the hospitals of Leningrad between 1993 and 1998.	No significant odds ratios between pesticide exposure and lung cancer mortality: 1.06 (95% CI: 0.82 - 1.36).
Lee et al. (2007b)	There exists an association between exposure to certain pesticides and incidence of colorectal cancer among pesticide applicators.	Agricultural Health Study: 56,813 pesticide applicators in Iowa and North Carolina.	Odds ratios: Chlorpyrifos for rectal cancer: 2.7 (95% CI: 1.2-6.4) Aldicarb with colon cancer: 4.1 (95% CI:1.3-12.8)
Lynch et al. (2006)	There is no clear and consistent association between cyanazine exposure and any of the cancer analyzed.	Agricultural Health Study: 57,311 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios: All cancers: 0.80 - 1.24 Prostate cancer: 0.87 - 1.70 All lymphohematopoietic cancers: 0.50 - 1.72
Lynch et al. (2009)	Statistically significant increased risks and exposure-response trends are observed for all lymphohematopoietic cancers and non-Hodgkin lymphoma, as well as increased risk for prostate cancer.	Agricultural Health Study: 19,655 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios: Prostate cancer: 1.27 - 3.44 NHL cancer:1.29 - 9.21 All lymphohematopoietic cancers: 1.18 - 4.37
Mathur et al. (2002)	Organochlorine pesticides taken for analysis are found significantly high in breast cancer patients irrespective of age, diet, and geographic distribution.	Data collected: 135 breast cancer patients and 50 female controls from the Birla Cancer Institute (Jaipur).	Organochlorine pesticide residues(DDT) in the blood of: controls: 1.034 (0.221); females with breast cancer: 2.254 (0.405)
McDuffie et al. (2001)	Non-Hodgkin Lymphoma is associated with specific pesticides after adjustment for other independent predictors.	Canadian multicenter population-based incident, case (n=517) - control (n=1506) study.	95% CI of odd ratios for different pesticides: Phenoxyherbicides: (1.06,1.81); Dicamba (1.32,2.68); Carbamate (1.22,3.04); Organophosphorus (1.27,2.36); Fumigant carbon tetrachloride (1.19,5.14)
Mozzachio et al. (2008)	No strong evidence for an association between chlorothalonil and the cancers investigated is found.	Agricultural Health Study: 47,625 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios: All cancers: 0.83 - 1.32; Colon cancer: 0.70 - 3.03; Lung cancer: 0.51 - 1.83; Prostate cancer: 0.52 - 1.21
Pahwa et al. (2011)	Soft-tissue sarcoma is associated only with specific pesticides (Aldrin and Diazinon).	Data collected: Mailed questionnaire followed by a telephone interview with 357 multiple myeloma cases and 1506 controls in 6 regions of Canada	Significant odds ratios of soft-tissue sarcoma for exposure to different pesticides: Aldrin (3.71, 95% CI: 1.00,13.76); Diazinon (3.31, 95% CI: 1.78, 6.23)
Pahwa et al. (2012)	Increased risk of multiple myeloma is associated to the exposure to carbamate pesticides, and fungicide captan.	Data collected: Mailed questionnaire followed by a telephone interview with 342 multiple myeloma cases and 1506 controls in 6 regions of Canada	Significant odds ratios for multiple myeloma associated to exposure to carbamates (1.90) and fungicide captan (2.35).
Potti et al. (2003)	There is evidence associating pesticide exposure to the early development of prostate adenocarcinoma, potentially aggressive.	Data collected: 56 young males with adenocarcinoma diagnosed between 1991 and 2001, with complete records in North Dakota and Western Minnesota.	From the subjects with adenocarcinoma, 66.1% have significant exposure to pesticides. The mean survival of the subgroup with pesticide exposure is 11.3 months, while for those without exposure is 20.1 months.

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Reference	Main conclusions	Data	Quantitative Results
Ruder et al. (2004)	No positive association of farm pesticide exposure and glioma is found in this study.	Data collected: 457 glioma cases and 648 controls. Adult men residents of Iowa, Michigan, Minnesota, and Wisconsin.	No significant odds ratios for: insecticides (0.53), fumigants (0.57), and organochlorines (0.66).
Rusiecki et al. (2004)	The analysis does not find any clear association between atrazine exposure and any cancer analyzed.	Agricultural Health Study: 53,943 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios for different cancers: Prostate: (0.63,1.23); Lung (0.93, 3.94); Bladder (0.86,10.81); NHL (0.62,4.16); Multiple myeloma (0.37,7.01)
Rusiecki et al. (2006)	The analysis does not detect strong evidence for an association between metolachlor exposure and any of the cancer sites investigated. Future research should put special attention to prostate and lung cancer.	Agricultural Health Study: 50,193 pesticide applicators in Iowa and North Carolina.	Rate ratios for exposure to metolachlor: Highest-exposure for prostate cancer: 0.59 2nd-highest-exposure for prostate cancer: 0.66
Rusiecki et al. (2009)	The study finds no association between permethrin and most cancers analyzed. For multiple myeloma this result is based on small numbers.	Agricultural Health Study: 49,093 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios for different cancers: Prostate: (0.64,1.18); Lung (0.35, 1.34); Bladder (0.64,2.74); NHL (0.28,1.71); Multiple myeloma (2.76,11.87); Leukemia (0.83,3.64); Melanoma (0.35,1.83).
Samanic et al. (2006)	No clear evidence is found for an association between dicamba exposure and cancer risk. However, the relation with lung and colon cancer should be further studied.	Agricultural Health Study: 41,969 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios for different cancers: All cancers: (0.85,1.23); Colon: (0.78,2.58); Prostate: (0.81,1.46); Lung (0.79, 2.72); Bladder (0.36,1.88); NHL (0.50,2.85); All hematopoietic: (0.74,2.31); Melanoma (0.33,2.13)
Samanic et al. (2008)	Increased risk of meningioma and herbicide exposure is significant for women, but no association is found for men.	Data collected in the U.S.: 462 glioma cases, 195 meningioma cases (diagnosed between 1994 and 1998), and 765 controls.	Significant odds ratio for women using herbicides (2.4, 95% CI: 1.4,4.3), with a significant trend with increasing years of exposure.
Van Bemmel et al. (2008)	S-ethyl-N,N-dipropylthiocarbamate (EPTC) use is associated with colon cancer and leukemia. Due to small numbers results should be interpreted with caution and further investigations are needed.	Agricultural Health Study: 48,378 pesticide applicators in Iowa and North Carolina.	95% CI of the Rate ratios and p-trend: Colon cancer: 1.26 - 3.47 (p-trend = <0.01) Leukemia: 1.16 - 4.84 (p-trend = 0.02)
Weichenthal et al. (2010)	Review of cancer studies related to pesticide use in the AHS. Most of the 32 pesticides examined are not strongly associated with cancer incidence.	28 studies that examined the relationship between pesticide exposures and cancer incidence in the AHS cohort.	In Table 1 the authors resume all the pesticides with significant association with cancer being analyzed using the AHS.
Xu et al. (2010)	The results suggest that OC pesticide use is related with an increase on cancer risk.	National Health and Nutrition Examination Survey (1999-2004). From 1,475 to 1,693 participants, depending on the year.	Adjusted odd ratios for second and third tertiles: beta-HCH: 1.46 and 3.36; trans-nonachlor: 5.84 and 14.1; dieldrin: 1.06 and 2.74.
Zhao et al. (2011)	High-level organochlorine pesticide exposure combined with aflatoxin B1 and Hepatitis B virus may greatly enhance the risk of Hepatocellular carcinoma.	Data collected: Questionnaire database built from 346 Hepatocellular carcinoma cases, and 961 controls in Xiamen (2007-09).	Positive interactions of DDT pesticide residues with HBV, diabetes mellitus, aflatoxin B1.
Zheng et al. (2001)	The results suggest an increased risk of non-Hodgkin lymphoma associated with carbamate pesticide exposure.	Three population-based case-control studies: 985 white male subjects and 2895 controls, from four Midwestern states in the U.S.	Significant odds ratios of NHL associated with Carbamate pesticide use: 1.5 (95% CI: 1.1 - 2.0)
DEPRESSION & NEUROLOGICAL DEFICITS			
Abdel et al. (2008)	Functional cognitive deficits are associated with increased years of exposure to organophosphate pesticides.	Data collected: 50 male children applying pesticides, and 50 controls were recruited in Egypt.	Pesticide applicators performed significantly worse in 10 tasks (out of 10) measuring neurobehavioral performance (p-value < 0.04).

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Reference	Main conclusions	Data	Quantitative Results
Baldi et al. (2001)	Low-level exposure to pesticides in occupational conditions has an effect on long-term cognitive performance.	Data collected: 917 Bordeaux vineyard workers (528 directly exposed to pesticides; 173 indirectly exposed; 216 never exposed).	Workers with direct exposure: significant lower performance (than no exposed workers) in 6 out of 9 tests. Workers with indirect exposure: significant lower performance (than no exposed) in 5 out of 9 tests.
Beseler et al. (2008)	Both acute high-intensity and cumulative pesticide exposure may contribute to depression in pesticide applicators. Depression is also associated with chronic pesticide exposure in the absence of a physician-diagnosed poisoning.	Agricultural Health Study: 534 cases and 17,051 controls among private pesticide applicators in Iowa and North Carolina.	95% CI of the Odds ratios: Pesticide poisoning: 1.74 - 3.79; Intermediate cumulative exposure: 0.87 - 1.31; High cumulative exposure: 0.87 - 1.42; HPEE: 1.33 - 2.05; High cumulative exposure among applicators without history of acute poisoning: 1.16 - 2.04
Bosma et al. (2000)	Exposure to pesticides by arable farmers and gardeners is associated with increased risks of mild cognitive dysfunction (MCD).	Maastricht Aging Study: 830 individuals included with 3-year follow-up examination (1996-98)	Significant increase in risk for MCD due to pesticide exposure: for self-reported exposure (OR=4.94); for job-related exposure (OR=1.47) (cross-sectional results).
Corder et al. (2006)	Inherited variation in mitochondrial complex genes and pesticide exposure together influence the risk for Parkinson's disease.	Data collected: 306 cases and 321 controls from an Australian case-control sample.	Groups with "early onset and regular exposure", and "early onset and limited exposure" presented higher risk for Parkinson's disease (OR: 2.7 and 4.0).
Eckerman et al. (2007)	The effect of pesticide exposure on neurobehavioral performance appeared to be especially strong for youngest population (10-11 year old).	Data collected: 38 farm children and 28 children from a city, both in the state of Rio de Janeiro, Brazil, and with ages between 10 and 18 years.	For 10-11 year old participants, 5 out of 16 behavioral measures show a strong negative association, while for 12-13 year old participants only 1, and none for participants older than 13 year old.
Fong et al. (2007)	Susceptible variants of MsSOD and NQO1 genes may have an interaction with occupational pesticide exposure to increase the risk of Parkinson's disease (PD).	Data collected: 153 patients with idiopathic PD and 155 healthy control subjects from southwestern region of Taiwan (2001-03).	Risk of PD is associated with pesticide exposure: unadjusted OR=1.69; adjusted OR=1.68. Polymorphisms of MnSOD and NQO1 genes are associated to pesticide exposure (OR=2.49 and OR=2.42, respectively).
Hancock et al. (2008)	Results show positive associations of pesticide exposure with Parkinson's disease (PD). Frequency, duration, and cumulative exposure are also significant factors.	Data collected: 319 cases and 296 controls, recruited from 2000 to 2006 at the Duke University Medical Center.	Significant increase in risk for PD for individuals applying: herbicides (OR=1.59); pesticides other than herbicides (OR=1.61); insecticides (OR=1.83).
Hong et al. (2009)	Evidence suggests that exposure to pesticide spraying is associated with the development of depression in farmers.	Data collected: 82 farmers living in the Western region of South Korea	The scores of depression tests are associated with the frequency of spraying over a year ($p<0.05$), number of years farming ($p<0.01$), and history of intoxication ($p<0.01$).
Kamel et al. (2005)	Results suggest that self-reported neurologic symptoms are associated with cumulative exposure to moderate levels of fumigants and organophosphate and organochlorine insecticides.	Agricultural Health Study: 18,782 private pesticide applicators in Iowa and North Carolina.	Significant odd ratios for organophosphates (chlorines): Insomnia: 1.70 (1.56); Depression: 2.09 (1.68); Absentmindedness: 2.13 (1.75); Changes in smell or taste: 1.83 (2.12); Difficulty speaking: 1.94 (1.97)
Kamel et al. (2007)	Incident Parkinson disease is associated with cumulative days of pesticide use at enrollment. Exposure to certain pesticides may increase Parkinson disease risk.	Agricultural Health Study: 83 prevalent cases, 78 incident cases, 79,557 prevalent controls, and 55,931 incident controls among pesticide applicators in Iowa and North Carolina.	95% CI of odds ratios: Incident Parkinson: 1.2 - 4.5; Prevalent Parkinson: 0.4 - 1.5
Lee et al. (2007)	There is a possible association between chlorpyrifos use and external causes of death. Although these results are based on small numbers, may reflect a link between chlorpyrifos and depression or other neurobehavioral symptoms.	Agricultural Health Study: 55,071 pesticide applicators in Iowa and North Carolina.	95% CI of Rate ratios related to lifetime chlorpyrifos exposure-days: Suicide: 1.03 - 5.48 Non-motor-vehicle accidents: 1.36 - 3.52
Park et al. (2012)	No significant results, however the authors find some association between occupational pesticide exposure and peripheral neurophysiologic abnormality.	Data collected: Nerve conduction studies on a cross-sectional study group of 31 male farmers who periodically applied pesticides.	All nerve conduction studies (on the median, ulnar, posterior tibial, peroneal, and sural nerves) remained within laboratory normal limits.

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Reference	Main conclusions	Data	Quantitative Results
Peiris-John et al. (2002)	Long-term low-level exposure to organophosphate pesticides is associated with sensory and motor impairment.	Data collected: 30 farmers who regularly spray organophosphate pesticides and 30 fishermen (controls).	Significant differences between farmers and controls found in: sensory conduction velocity (P=0.04); motor conduction velocity (P=0.04).
Roldan-Tapia et al. (2005)	There is a significant association of long-term exposure to carbamates and organophosphates and worse performance in neuropsychological functions.	Data collected in southeastern Spain from March and April 1998: 26 in the control group and 40 in the exposed group.	Effect of cumulative exposure in worsening: perceptive function performance (OR=6.93); visuomotor praxis (OR=5.00); integrative task performance (OR=4.12)
Roldan-Tapia et al. (2006)	Acutely poisoned long-term workers and chronically high exposed workers exhibit similar disturbances in perception and visuomotor processing.	Data collected in southeastern Spain: 26 in the control group, 40 in the non-poisoned but exposed group, and 24 in the acutely poisoned group	Tests where acutely poisoned group performed worse than the control group: Picture completion, rey-auditory verbal learning test, logical memory, rey-osterrieth figure test, and benton visual form test.
Rothlein et al. (2006)	Evidence regarding a lower neurobehavioral performance of Hispanic immigrant farmworkers is found, compared to nonagricultural Hispanic immigrant population.	Data collected: 96 farmworkers recruited by community members of the Migrant Head Start program in Hood River, between 20 and 52 year old, and originally from Mexico.	Mean scores of different tests with significant differences between agricultural and nonagricultural subjects: Digit span backward (3.86 vs 4.53); Finger tapping [females] (81.68 vs 90.41).
Sathiakumar et al. (2004)	The results are consistent with the absence of an association between triallate and measures of neurological function.	Data collected: 349 subjects working at a plant. Selection of 50 with the highest triallate scores and 50 with no-exposure.	Mean sural nerve peak amplitude comparison: High-exposed: 11.7 vs No-exposed: 15.2 (p=0.03) After adjusting for other potential risk: High-exposed: 12.5 vs No-exposed: 14.5 (p=0.25)
Schultz et al. (2013)	Cognitive ability declines faster in people with occupational pesticide exposure compared to their unexposed counterparts.	Data collected: 18 subjects with occupational pesticide exposure and 35 controls. All of them white English native speakers.	Results of Mini-Mental Status Exam showed a significant negative correlation with age only for subjects with occupational exposure (r= -0.45, p<0.04)
Stallones(2006)	Results suggest an association between pesticide exposed occupations and suicides. However, this association is significant only for women.	Data collected: 4,991 suicide deaths and 107,692 other deaths in Colorado between 1990 and 1999.	95% CI of odds ratios of suicide associated to occupational pesticide exposure: men (0.97, 1.34); women (1.01, 3.88).
Starks et al. (2012)	Evidence linking high pesticide exposure events (HPEE) and adverse central nervous system outcomes is found.	Agricultural Health Study: 156 male participants (from 2006 to 2008) with a HPEE, and 537 controls completed 9 neurobehavioral tests.	Participants reporting a HPEE were 4.2s slower in a processing test, and 2.5s slower in a motor speed test.
Starks et al. (2012b)	Significant evidence associating long-term organophosphate pesticides and impaired Peripheral Nervous System function.	Agricultural Health Study: 1,807 eligible subjects, with a 39% participation rate (in Iowa and North Carolina).	Ten out of 16 OP pesticides associated with at least one Neurological Physical Examination outcome. 6 OP pesticides were associated with abnormal toe proprioception, with odds ratios ranging from 2.03 to 3.06.
Steenland et al. (2013)	Elderly subjects with past occupational pesticide exposure are associated with a higher risk for dementia and Parkinson's disease (PD).	Data collected: 400 elderly subjects at two government-run clinics in Costa Rica. 361 of them did two different tests, and 144 were examined by a neurologist.	Exposed subjects have significant higher risk of abnormal scores on tremor-at-rest test (OR=2.58), on finger-tapping (OR=2.94), and an increased risk of PD (OR=2.57).
Van Wijngaarden (2003)	Employment in jobs involving pesticide exposure is associated with the risk of death from mental disorders.	Data collected: 7,756 deaths from mental disorders and 330,452 controls. Data comes from US death certificates (1988-1992).	Significant odds ratios of the risk of death due to mental disorders associated with pesticide exposure: 1.46 (all); 2.65 (women); 4.32 (women dying from neurotic disorders).
Zhang et al. (2009)	Potential markers of chronic pesticide exposure is significantly associated with suicidal ideation.	Data collected: survey with 9,811 rural residents in Zhejiang province.	Significant effect of pesticide storage at home with suicidal ideation over the prior 2 years: Unadjusted OR=2.12; Adjusted OR=1.63.

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Reference	Main conclusions	Data	Quantitative Results
DIABETES			
Cox et al. (2007)	Higher serum levels of certain organochlorine pesticides may be associated with increased prevalence of diabetes.	Hispanic Health and Nutrition Examination Survey: 1,303 Mexican Americans between 20 and 74 years of age.	95% CI of crude odds ratios for different pesticides: Hexachlorobenzene (1.2,2.3); Dieldrin (1.8,18.5); trans-Nonachlor (3.2,11.9); Oxychlordane (2.9,15.8); p,p'-DDT (2.0,8.4); p,p'-DDE (2.9,14.7); B-HCH (2.9,15.8)
Montgomery et al. (2008)	Long-term exposure from handling certain pesticides, in particular organochlorine and organophosphate insecticides, may be associated with increased risk of diabetes.	Agricultural Health Study: 33,457 licensed applicators in Iowa and North Carolina.	Statistical significant adjusted odds ratios (at 95%): Chlordane: 1.16; Heptachlor: 1.20; Coumaphos: 1.26; Phorate: 1.22; Terbufos: 1.17; Trichlorfon: 1.85
Saldana et al. (2007)	Activities involving exposure to agricultural pesticides during the first trimester of pregnancy may increase the risk of gestational diabetes mellitus (GDM).	Agricultural Health Study: 11,273 women whose pregnancy occurred within 25 years of enrollment.	95% CI of odds ratios: Agricultural pesticide exposure: 1.5 - 3.3 Residential pesticide exposure: 0.8 - 1.3
RESPIRATORY			
Boers et al. (2008)	No significant association between exposure to ethylenebisdithiocarbamates or other pesticides and asthma is found.	Data collected: From 5 field studies (in the Netherlands, Italy, Finland, and Bulgaria[2]). 248 workers exposed to pesticides and 231 controls.	No significant results for associations between occupational exposure to pesticides and asthma (OR=0.41), chest tightness (OR=0.60), wheeze (OR=0.56), or asthma attack (0.52).
Fieten et al. (2009)	Although the present study could not establish a causal relation, it does suggests association between exposure to pesticides and respiratory symptoms. The major limitation is the small study population and lack of quantitative exposure estimates.	Cross-sectional study among a population of indigenous women in Costa Rica (69 exposed women, and 58 unexposed).	Statistically significant odd ratios for wheeze: Chlorpyrifos: 2.7; Terbufos (smoking): 2.8; Chlorpyrifos (smoking): 3.5; Terbufos (non-smokers): 5.9; Chlorpyrifos (non-smokers): 6.7
Hernandez et al. (2008)	Exposure to certain pesticides is associated to a fall in the diffusing capacity of the lungs and with lower pulmonary volumes.	Data collected: 89 pesticide sprayers and 25 nonspraying control farmers from the south of Spain.	Significant increase in the risk of presenting respiratory symptoms due to the exposure to endosulfan: OR=3.68 (95% CI: 1.16,11.68)
Hoppin et al. (2002)	There is evidence that some pesticides may contribute to respiratory symptoms among farmers, specifically there are some pesticides associated to wheeze.	Agricultural Health Study: 20,468 licensed applicators in Iowa and North Carolina.	Statistically significant odd ratios for wheeze: Alachlor: 1.24; Altrazine: 1.20; Chlorimuron ethyl: 1.14; Paraquat: 1.27; Petroleum oil: 1.28; Permethrin: 1.26
Hoppin et al. (2006)	Organochlorine and carbamate pesticides should be further evaluated as potential risk factors for farmer's lung.	Agricultural Health Study: 50,000 farmers and farmer's spouses in Iowa and North Carolina.	Statistically significant odd ratios for farmer's lung: Handling silage: 1.41; High pesticide exposure events: 1.75; Use of Organochlorine: 1.34; Use of Carbamate: 1.32
Hoppin et al. (2006b)	The results of this study suggest a link between respiratory health and organophopshate insecticides such as chlorimuron-ethyl.	Agricultural Health Study: 2,255 Iowa commercial pesticide applicators.	Significant increasing dose-response trends: Chlorimuron-ethyl: p-trend = 0.012; Chlorpyrifos: p-trend = 0.003; Phorate: p-trend = 0.010.
Hoppin et al. (2007)	The findings of this study suggest that pesticides may contribute to atopic asthma among farm women.	Agricultural Health Study: 25,814 farm women in Iowa and North Carolina.	95% CI of odds ratios for atopic asthma: Use of pesticides: 1.14 - 1.87 Grew up on farm & did not apply pesticides: 0.27 - 0.62

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Reference	Main conclusions	Data	Quantitative Results
Hoppin et al. (2007b)	This study provide preliminary evidence that pesticide use may increase chronic bronchitis prevalence.	Agricultural Health Study: 20,908 private pesticide applicators in Iowa and North Carolina.	95% CI of odds ratios for chronic bronchitis: Heptachlor: 1.19 - 1.89 High exposure event: 1.51 - 2.25 Pesticide use in off-farm jobs: 1.04 - 1.88
Hoppin et al. (2009)	This study contribute with the literature suggesting that pesticides may increase asthma risk among farmers.	Agricultural Health Study: 19,704 male farmers in Iowa and North Carolina.	Significant odds ratios for allergic asthma (>2): Coumaphos: 2.34; Heptachlor: 2.01; Parathion: 2.05; Carbon tetrachloride/disulfide: 2.15; Ethylene dibromide: 2.07
Mekonnen et al. (2002)	Farm workers in various job categories are subject to reduced pulmonary function and present respiratory symptoms that could lead to chronic respiratory health problems.	Data collected: 103 sprayers, 15 supervisors, 14 technicians, 19 pest assessors, and 80 controls at Ethiopia.	Prevalence of respiratory symptoms among non-smoker farm workers: Breathlessness: Sprayers 16.5%; Supervisors 26.7%; Technicians 35.7%; Pest assessors 10.5%; Controls 16.3%
Salameh et al. (2006)	The results suggest that occupational exposure to pesticides may explain chronic respiratory symptoms and asthma	Data collected: 186 case and 186 control subjects coming from 10 medical centers in all Lebanese regions.	Significant association between any pesticide exposure and asthma (OR=2.11), occupational exposure and asthma (OR=4.98), and regional exposure and asthma (OR=3.51).
Slager et al. (2009)	Exposure to pesticides may increase the risk of rhinitis.	Agricultural Health Study: 2,245 Iowa commercial pesticide applicators.	Significant odds ratios for rhinitis: 2,4-D: 1.34 ; Glyphosate: 1.32 ; Petroleum oil: 1.74 ; Diazinon: 1.84; Fungicide benomyl: 2.35
Sutoluk et al. (2011)	Chronic environmental organophosphorus exposure causes a decrease in the serum chlorinesterase levels in farm workers, but has no effect on pulmonary functions	Data collected: 50 male seasonal farm workers and 50 male non-farm workers located in Cukurova region, Turkey (2007).	Mean serum cholinesterase enzyme levels are significantly lower for the farm group (7095.5) than for the non-farm group (9716.4).
WOMEN SPECIFIC DISORDERS			
Akkina et al. (2004)	High exposure to organochlorine pesticides is associated with a decrease in the mean age at menopause.	Hispanic Health and Nutrition Examination Survey: 219 menopausal women (1982-1984).	Significant reduction on the years to menopause for women with high exposure to pesticides: between 3.4 and 5.7 years earlier depending on the pesticide.
Farr et al. (2004)	The findings of this study suggest that use of certain hormonally active pesticides may affect menstrual cycles.	Agricultural Health Study: 3,103 women living on farms in Iowa and North Carolina.	Significant odds ratios. Use of pesticides on missed period: 1.6; Carbamate use on long menstrual cycle: 2.1; Crop insecticide on missed period: 1.6; Use of hormonally active pesticides had a 60-100% increased odds of experiencing long cycles, missed periods, and intermenstrual bleeding compared with no use of pesticides
Farr et al. (2006)	Pesticide use may be associated with a larger age at menopause.	Agricultural Health Study: 8,038 women living and working on farms in Iowa and North Carolina.	Increased median time to menopause when using pesticides: 3 months. Increased median time to menopause when using hormonally active pesticides: 5 months.
Lauria et al. (2006)	Pregnancies with delayed conception are associated to pesticide exposure, but the results are not significant when adjusting for confounding variables.	Data collected: 713 women active in 34 greenhouse flower growing enterprises in 1998-2000.	Non-significant hazard ratio for reduced fertility among the group exposed to pesticides (HR=0.96. 95% CI: 0.81,1.13).
Saldana et al. (2009)	Exposure to pesticides during pregnancy may increase the risk of hypertensive disorders of pregnancy.	Agricultural Health Study: 11,274 wives of farmers from Iowa and North Carolina (1993-1997).	First-trimester agricultural activities with potential exposure to pesticides are significantly associated with: pregnancy-induced hypertension (aOR=1.60) and preeclampsia (aOR=2.07).

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Reference	Main conclusions	Data	Quantitative Results
GENERAL HEALTH, MULTIPLE DISEASES AND OTHERS			
Azmi et al. (2006)	Exposure to multiple pesticides for prolonged period affects the normal functioning of different organ systems and possibly has clinical effects such as hepatitis, dyspnea and burning sensation in urine.	Blood samples from 83 farm workers from 14 different fruit and vegetable farm stations located in the surroundings of Gadap (Pakistan).	t-statistics (P-values) for differences in enzyme (GOT, GPT, ALP) levels unexposed vs exposed: GPT: -2.57 (0.62); GOT: -3.20 (0.033); ALP: -7.3 (0.0019)
Baranska et al. (2008)	The study identify individuals within the group of pesticide exposed workers with a genetic polymorphism who show a lower antibody response.	Data collected: 238 workers occupationally exposed to pesticides, and 198 controls, in four European countries.	Significantly lower mean anti-HB antibody levels for exposed subjects (1.72) than for controls (3.08), with IL1alpha2.2 polymorphic gene expression.
Beard et al. (2003)	The findings of this study suggest an association between adverse health effects (asthma, diabetes, pancreatic cancer, leukemia, lower neuropsychologic functioning scores, and diabetes) and exposure to pesticides.	Comparison of mortality off 1,999 outdoor staff with pesticide exposure, 1,984 outdoor workers not exposed, and Australian population average levels.	95% CI of standardized mortality ratios: asthma: 1.39 - 7.10; diabetes: 1.16 - 8.32; pancreatic cancer: 1.09 - 15.40; leukemia (incidence ratio for modern chemicals): 1.54 - 284.41
Beard et al. (2011)	There is no association between prior pesticide use and suicide in applicators and their spouses.	Agricultural Health Study: 110 suicides between 1993 and 2009, among 81,998 cohort members (in Iowa and North Carolina).	95% CI of Hazard ratios based on the days per year personally mixed or applied pesticides (none = reference): less than 20 (0.42,2.28) ; more than 20 (0.36,2.20)
Crawford et al. (2008)	Control for exposure to noise and other neurotoxicants is limited, however the results extend previous reports suggesting that organophosphate exposure increases the risk of hearing loss.	Agricultural Health Study: 14,299 white male pesticide applicators in Iowa and North Carolina.	Significant odds ratios for hearing loss: All insecticides: 1.19; Organophosphate insecticides: 1.17; High pesticide exposure events: 1.38; Hospitalization: 1.81; Diagnosed pesticide poisoning: 1.75
Dayton et al. (2010)	Pesticides may contribute to Myocardial Infarction (MI) risk among farm women.	Agricultural Health Study: 22,425 farm women in Iowa and North Carolina.	Significant odds ratios for non-fatal MI: Chlorpyrifos: 2.1; Coumaphos: 3.2; Carbofuran: 2.5; Metalaxyl: 2.4; Pendimethalin: 2.5; Trifluralin: 1.8
Del Prado-Lu (2007)	The findings of this study are further proof of the hematoxic effects of pesticide exposure. There exists a detrimental effect of pesticide exposure on RBC cholinesterase levels.	Data collected: 102 randomly selected cutflower farmers in La trinidad, Benguet.	P-values for significant variables explaining abnormal RBC cholinesterase levels: Age: p=0.02; selling pesticide containers: p=0.008; number of years of using pesticides: p=0.022; use of contaminated cloth: p=0.033; illness due to pesticides: p=0.005; improper mixing of pesticides: p=0.041; sex: p=0.002
Fleming et al. (2003)	Farmers and pesticide applicators have a greater risk of accidental mortality than the rest of the workers. They also present an increased risk of hematopoietic and nervous system cancers.	National Health Interview Surveys (NHIS): 9,471 farmers and 438,228 controls in US	Significant rate ratios for different causes of death: All causes: 1.3; Heart diseases: 1.4; Nervous system cancer: 2.4; Hematopoietic cancer: 2.2
Goldner et al. (2010)	The collected data support a role of organochlorines, in addition to fungicides, in the etiology of thyroid disease among female spouses enrolled in the AHS.	Agricultural Health Study: 16,529 spouses of pesticide applicators of Iowa and North Carolina.	Significant adjusted odds ratios for hypothyroidism: Organochlorine insecticides: 1.2 Fungicides: 1.4 Significant adjusted odds ratios for hyperthyroidism: Maneb/mancozeb: 2.3
Gomez-Marin et al. (2004)	The present results indicate that at any point in time, compared to other US workers, farmers are in general healthier, whereas, despite the small numbers, pesticide applicators have a similar or poorer health	National Health Interview Surveys (NHIS): 9,576 farmers and 453,219 controls in US	Significant odds ratios (and 95% CI) for chronic disability (doctor visit): Farmers (vs other workers): 0.82 (0.76-0.88) Pesticide applicators (vs other workers): 1.21 (0.80-1.83)

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Reference	Main conclusions	Data	Quantitative Results
Hernandez et al. (2003)	Chronic exposure to pesticides might be associated with serum paraoxonase (PON1) decrease activity.	Data collected: 102 individuals with long term low dose exposure to pesticides in a plastic greenhouse setting.	Significant differences between sprayers (14.1%) and non sprayers (5.1%) were observed in the PON1-909 G/C polymorphism (p=0.045)
Howard et al. (2010)	Single-nucleotide polymorphisms (SNPs) in the BCHE gene is associated with pesticide exposure.	Data collected: From the Community ärticipatory Approach to Measuring Farmworker Pesticide exposure: 287 farmworkers recruited.	Association of SNPs in BCHE with cholinesterase activity (FDR adjusted p-values): rs2668207 (p=0.00098) rs2048493 (p=0.00068)
Kesavachandran et al. (2009)	The findings of this study provide some evidence of clinical manifestations because of multiple exposures to pesticides and poor safety culture at work place.	Data collected: detailed information was recolleted through questionnaries from 20 subjects and controls in Uttar Pradesh, India.	Significant rate ratios for shopkeepers exposed to pesticides (p <0.05): Cardiovascular problems: 3.36; Genito-urinary problems: 8.64; Nervous system related problems: 2.88; Skin problems: 4.32.
Khan et al. (2009)	Unsafe practices among small and medium sized pesticides industrial workers cause significant increase in pesticide exposure, oxidative stress, and derangement of hepatic and renal function.	Data collected: Plasma cholinesterase (PChE) levels from 238 exposed workers (54 controls) in Pakistan, estimated by Ellmann's method.	Significant (p<0.05) differences in biochemical markers between size-exposed groups and control: Hemoglobin: small (13.98); control (14.92) Alanine aminotransferase: small (34); medium (31); control (25) Asparate aminotransferase: small (25); medium (27); control (24)
Kirrane et al. (2005)	The findings suggest that exposure to some fungicides and other pesticides may increase the risk of retinal degeneration.	Agricultural Health Study: 31,173 farmers' wives in Iowa and North Carolina.	Significant adjusted odds ratios and 95% CI for retinal degeneration: Fungicides (general): 1.9 (1.2 - 3.1)
Lacasaña et al. (2010)	Results suggest that the exposure to organophosphate pesticides may affect the serum hormone levels, acting as endocrine disruptors in humans.	Data collected: 136 male subjects from State of Mexico and Morelos, occupationally exposed to organophosphate pesticides.	Significant increase in TSH and T4 hormones in serum associated with increased exposure of pesticides (dimethylphosphate, p-trend < 0.001), as well as a decrease in T3 serum levels (p-trend=0.053).
Lacasaña et al. (2010b)	The results suggest an interaction between organophosphate pesticide exposure and thyroid function which is stronger for individuals with lower paraoxonase-1 enzyme (PON1) activity.	Data collected: during two periods (July-Oct. 2004 and Dec. 2004 - May 2005) for 84 workers.	Significant variation (in %) on the thyroid stimulating hormone for each increment in one log unit of the dialkylphosphate metabolite in the urine for: PON1(192)QR polymorphism (23%); PON1(192)RR polymorphism (59%).
Landgren et al. (2009)	The prevalence of monoclonal gammopathy of undetermined significance (MGUS) among pesticide applicators was twice that in a population-based sample of men from Minnesota. Specific pesticides are causatively linked to myelomagenesis.	Agricultural Health Study: stratified random sample of 678 pesticide applicators in Iowa and North Carolina.	Significant odds ratios for MGUS by pesticide: Dieldrin: 5.6 Carbon-tetrachloride / disulfide mix: 3.9 Chlorthalonil: 2.4
Martin et al. (2002)	Black farmers present lower exposure to pesticides than white farmers as well as fewer pesticide-related symptoms. Black farmers may also have other work practices that affect exposure and risk.	Agricultural Health Study: self-administered questionnaires by 891 black and 11,909 white farmers.	Significant odds ratios (black vs white) for exposition to different pesticides: Herbicides: 0.49; Insecticides: 0.28; Fumigants: 0.40; Fungicides: 0.82
Melkonian et al. (2011)	Evidence found points to the significance of synergisms between effects of arsenic exposure and pesticide/fertilizer use on incident skin lesions.	Health Effects of Arsenic Longitudinal Study: 5,042 men from Araihazar, Bangladesh recruited from 2000 to 2002.	Significant synergistic effects between fertilizer use and water arsenic: Relative excess risk = 0.06 (95% CI: 0.01,0.12).
Mills et al. (2009)	There is no evidence of a dose response with any pesticide measure with respect to myocardial infarction (MI). However some pesticides seems to be related to MI.	Agricultural Health Study: 54,609 pesticide applicators in Iowa and North Carolina.	Significant (p<0.05) hazard ratios for MI by pesticide: For MI mortality: Ethylene dibromide: 1.54; Maneb: 1.34; Ziram: 2.40 For MI incidence: Aldrin: 1.20; DDT: 1.24; 2,4,5-T: 1.21

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Reference	Main conclusions	Data	Quantitative Results
Murphy et al. (2004)	Small scale West Sumatran female farmers apply pesticides without personal protection in a highly unsafe manner. They found striking the number of products that are handled on a weekly basis, their human health hazard level and the fact that these are handled during continuous growing seasons.	Data collected from 161 women sprayer (cases), and 353 rice farmers (controls), in Indonesia.	Significant relative risk for different symptoms: Burning nose (53.2); Sore throat (18.5); Muscle cramps (10.6); Nausea (9.4); Constipation (8.3); Eye burning (7.3); Excessive salivation (6.7); Dizzy (5.2); Blurred vision (4.4); Stomack pain (2.7); Numbness (2.6); Dry throat (1.7); Shortness of breath (1.5)
Ogut et al. (2011)	The results suggest an association between chronic exposure to pesticides and oxidative stress.	Data collected: 94 pesticide-formulating workers, and 45 controls.	Chronic exposure to pesticides is significantly associated with increased activities of catalase (17.40 vs 29.22), of superoxide dismutase (39.31 vs 74.39), and others, reflecting oxidative stress.
Orton et al. (2011)	All pesticides with previous evidence of androgen receptor antagonism (14) are confirmed as antiandrogenic, along with 9 previously untested pesticides.	37 pesticides tested for invitro androgen receptor antagonism.	ERR - Environmental Relevance Ratios (Top 5): Procymidone (202.5); Fenitrothion (112.2); Vinclozolin (79.8); Dimethomorph (45.6); Fludiowonil (31.2).
Padungtod et al. (2000)	Exposure to specific organophosphate pesticides might have a moderate adverse effect on semen quality	Data collected: 32 male workers occupationally exposed to pesticides and 43 controls, from two nearby factories in China.	Significant reduction of sperm concentration (exposed: 35.9*10^6; non-exposed: 62.8*10^6), and percentage of motility (exposed: 57%; non-exposed: 61%).
Perry et al. (2011)	An association between dimethylphosphate pesticide exposure and low performance of sperm (in concentration and motility) is found.	Data collected: 94 cases and 95 controls, recruited from July 2003 to February 2005 in Anhui Province.	Significant relative odds of case status for dimethylphosphate exposure: aOR=1.30. Any other pesticide was non-significant.
Recio et al. (2001)	Organophosphorous pesticides (OP) might affect sperm chromosome segregation and increase the risk for genetic syndromes.	Data collected: multicolor fluorescence in situ hybridization on samples from 9 men before and during pesticide spraying season.	Significant associations between diethylphosphate and sex null (betha=0.00022, p=0.0001). Smaller, but still significant associations were found for other OP.
Recio et al. (2005)	The results suggest that organophosphorous pesticides (OP) might disrupt the hypothalamic-pituitary endocrine function, and the most affected hormones are the follicle-stimulating hormone (FSH), and the luteinizing (LH).	Data collected: 64 healthy men from Villa Juarez, Durango, in Mexico, provided the complete set of samples to carry on the analysis.	Significant effect of dimethylthiophosphate on LH levels (betha=-0.0002, p=0.008), and on FSH levels (betha=-0.002, p=0.0003).
Sanchez-Pena et al. (2004)	Results suggest an association between organophosphate pesticide exposure and alterations in the human sperm chromatin.	Data collected: 33 agricultural workers from the community of Villa Juarez, Durango, in Mexico.	The results are presented as mean values of the subjects compared with reference values proposed in other studies. 82% of the samples were above reference values proposed in the literature.
Sekiyama et al. (2007)	Precautionary measures have a significant impact on the analysis of number of reported symptoms, therefore the role of formal training and information on the use and handling of pesticides is crucial.	Data collected in 2006 at West Java, Indonesia. 73 farmers from two villages were enrolled in the study.	Coefficients (p-value) for significant variables in the regression analysis of number of reported symptoms: Headgear use: 0.315 (0.005); wet clothing: -0.280 (0.012); smoking while spraying: -0.262 (0.017)
Sprince et al. (2000)	Results confirming associations between respiratory symptoms and conventional vertical silos may be important in future studies aimed at prevention and control of exposures. There is also an association between insecticide application and symptoms of airways disease.	Iowa Farm Family Health and Hazard Surveillance Project: 385 farmer participants.	Significant odds ratios for different symptoms: Phlegm: 1.91; Chest ever wheezy: 3.92; Flu-like symptoms: 2.93
Strong et al. (2004)	Certain self-reported health symptoms in farmworkers may be associated with indicators of exposure to pesticides.	Data collected: 211 households with children between ages of 2 and 6 years in Eastern Washington.	Health symptoms most commonly reported: Headaches: 50% Burning eyes: 39% Pain in muscles, joints, or bones: 35% Rash or itchy skin: 25%
Swan (2006)	The results suggest an association between current-use pesticides and reduced semen quality (concentration and motility).	Data collected: From 493 men from Columbia, New York, Minneapolis, and L.A., 25 with lower sperm quality were selected as cases, and 25 with standard sperm quality as controls.	Men are significantly more likely to be cases if they presented high levels of alachlor (OR=30) and diazinon (OR=16.7)(compared to men with low levels), as well as men with atrazine over the limit of detection (OR=11.3).

Table 2. Indirect exposure

Reference	Main conclusions	Data	Quantitative Results
CANCER			
Brody et al. (2004)	No overall association between pesticide use and breast cancer is found. However, increases in risk of cancer is associated with aerial application of persistent pesticides on cranberry bogs, and less persistent pesticides applied for agriculture.	Data collected: 1,165 women residing in Cape Cod, MA, diagnosed with breast cancer (1988-1995) and 1,006 controls. Geographic information system technology is used to assess the exposure to pesticides.	Just one significant association between pesticide exposure (low) related to agriculture and higher risk of cancer (5-year tumor promotion period assumed): aOR=1.4 (95% CI: 1.1-1.8).
Carreon et al. (2005)	Results show that exposure to pesticides is not associated with an increased risk of intracranial gliomas in women. No significant increase in risk of glioma is observed for carbamate herbicides, similar to what is observed with 12 specific pesticides.	National Institute for Occupational Safety and Health Upper Midwest Health Study: 341 cases and 528 controls.	95% CI of odd ratios for different pesticides: Arsenicals (0.5,1.9); Benzoic acids (0.4,1.5); Carbamates (0.5,1.9); Chloroacetanilides (0.6,2.0); Dinitroanilines (0.4,1.5); Inorganics(0.3,2.1); Organochlorines (0.7,1.8); Organophosphates (0.6,1.5); Phenoxys (0.5,1.5)
Cockburn et al. (2011)	Evidence is found regarding an association between prostate cancer and ambient pesticide exposures in and around homes in intensely agricultural areas.	Data collected: 173 cases and 163 controls from the Central Valley, CA (2005-06).Past exposure is estimated using a geographic information system approach.	Significant associations between higher prostate cancer risk and exposure to: methyl bromide (OR=1.62) and organochlorines (OR=1.64).
Ding et al. (2012)	Pesticide exposure measured through urine tests may be associated with an elevated risk of childhood acute lymphocytic leukemia (ALL).	Data collected: 176 children aged 0-14 years and 180 matched controls (Shanghai, 2010-2011).	Significant increased risk of ALL for the highest quartiles of different metabolites level (compared to lowest quartiles): total metabolites (OR=2.75); cis-DCCA (OR=2.1); trans-DCCA (OR=2.21); 3PBA (OR=1.84).
Engel et al. (2005)	Some evidence of increased risk of breast cancer is found when exposed to 2,4,5-TP, dieldrin, captan, but small numbers of cases are presented, so the results may not be reliable. Risk is modestly elevated among women whose homes are closest to areas of pesticide application.	Agricultural Health Study: 30,454 farmers' wives in Iowa and North Carolina.	95% CI of rate ratios for different pesticides: Any pesticides (0.7,1.1); Dieldrin (1.1,3.3); 2,4,5-TP (1.2,3.2); Captan (1.7,4.3)
Ferreira et al. (2013)	Results suggest that pesticide exposure during pregnancy may be involved in the etiology of acute leukemia in children under 2 years of age.	Data collected: Mothers of 252 cases and 423 controls located in one of 13 Brazilian states, during 1999-2007.	Significant associations of pesticide use during pregnancy and higher risk of: acute lymphoid leukemia (aOR=2.10) and acute myeloid leukemia (aOR=5.01) for 0-11 month-old kids; acute lymphoid leukemia (aOR=1.88) for 12-23 month-old kids.
Flower et al. (2004)	Risk of all childhood cancers combined increases, as well as risk of all lymphomas combined, and Hodgkin's lymphoma. Increased risk of cancer is detected among children whose fathers do not use chemically resistant gloves.	Agricultural Health Study: 17,357 children of Iowa pesticide applicators.	95% CI of standardized incidence ratios: All cancers: 1.03-1.79; All lymphomas: 1.13 - 4.19; Hodgkin's lymphoma: 1.06 - 6.14; 95% CI of odds ratio for fathers not using resistant gloves: 1.05 - 3.76
Ma et al. (2002)	Exposure to household pesticides is associated with an increased risk of childhood leukemia.	Northern California Childhood Leukemia study: 162 patients with leukemia (diagnosis between 1995 and 1999) and 162 matched controls.	Significant effects on increased risk of childhood leukemia and exposure to pesticides: exposure from 1 year before birth to 3 years after (OR=2.8); during year 2 (OR=3.6); 3 months before pregnancy (OR=1.8); during pregnancy (OR=2.1).
Meinert et al. (2000)	Some evidence for an increased leukemia risk for children living on farms is found, as well as association between household pesticides and risk of childhood leukemia or lymphoma.	Data collected: 1,184 children with leukemia, 234 with non-Hodgkin's lymphoma, and 940 with a solid tumor, as well as 2,588 controls in the states of West Germany (1993-97).	Significant associations of: use of pesticides on farms and childhood leukemia (OR=1.5); residential use of insecticides and childhood lymphoma (OR=2.6).

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Reference	Main conclusions	Data	Quantitative Results
Nielsen et al. (2010)	Results suggest an interaction effect between exposure in childhood to organophosphorus insecticides and a reduced ability to detoxify that might lead to childhood brain tumors (CBTs).	Data collected: DNA samples obtained from newborn screening archives for 201 cases and 285 controls, born in California or Washington State between 1978 and 1990.	Significant association between CBT and some genetic polymorphisms among exposed children: PON1(108T) allele (OR=1.8); FMO1(953-6A) allele (OR=2.7). This associations are not significant for not exposed children.
O'Leary et al. (2004)	Exposure to pesticides in the environment is associated with breast cancer risk under specific situations.	Data collected: 105 breast cancer cases (1980-1992) and 210 matched controls. Pesticide exposure is estimated through water measures and distance to hazardous waste sites (HWS) using GIS.	Significant effect on increased breast cancer risk for: living within 1 mile of HWS containing organochlorine pesticides (OR=2.8); women living on previously agricultural land with age over 26 years at first birth (compared with no agricultural land and age under 26 years at first age) OR=6.4.
Pearce et al. (2006)	Results do not support a role for preconception paternal occupational exposures to pesticides or herbicides in the etiology of childhood cancer.	Data collected: 4,032 cases from the Northern Region Young Persons' Malignant Disease Registry (RYPMDR), and 183,083 controls from the same source and from the Cumbrian Births Database.	Significant associations only for male subjects using the RYPMDR controls for pesticide exposure and: Neuroblastoma and ganglione uroblastoma (OR=2.38); Fibrosarcoma and other fibromatous neoplasm (OR=3.89).
Rudant et al. (2007)	The findings go in line with the hypothesis of domestic use of pesticides playing a role in the etiology of childhood hematopoietic malignancies.	ESCALE (Etude sur les cancers de l'enfant): 1,060 incident cases of hematopoietic malignancies included (cases in France, from 2003 to 2004).	Significant effects of insecticide use during pregnancy on: childhood acute leukemia (OR=2.1); non-Hodgkin lymphoma (1.8); Burkitt lymphoma (OR=2.7).
Safi (2002)	Heavy use and misuse of pesticides and other toxic substances in the Gaza environment is suspected to correlate with the growing incidence of Cancer.	Data collected: Pesticide usage in Gaza Governorates was recorded from 1990-1999. 2,277 cases of cancer in males and 2,458 in females were analyzed.	Significant associations of cancer incidence and type of pesticide: insecticides 0.992 ($p<0.001$); fungicides 0.952 ($p<0.001$); herbicides 0.812 ($p=0.004$); nematicides 0.925 ($p<0.001$).
Shim et al. (2009)	Significant associations between parental exposure to pesticides and risk of astrocytoma in offspring is found, except for primitive neuroectodermal tumors.	Data collected: 526 cases and the same number of matched controls, located in one of four U.S. Atlantic Coast states.	Significant effects of parental herbicide exposure from residential use and risk of astrocytoma (OR=1.9), and in the case of both, residential and occupational exposure (OR=1.8).
Ward et al. (2009)	Polychlorinated biphenyl congeners (PCBs) may represent a risk factor for childhood acute lymphocytic leukemia (ALL).	Data collected: 184 cases of ALL (kids 0-7 years old) and 212 matched controls. 35 counties in northern and central California (2000-06). Carpet dust samples collected.	Significant associations of PCBs residues in the carpet dust and higher risk of ALL (OR=1.97). Comparing highest quartile with the lowest (in terms of PCBs residues) OR=2.78.
DEPRESSION & NEUROLOGICAL DEFICITS			
Beseler et al. (2006)	Pesticide poisoning may contribute to risk of depression of spouses of pesticide applicators.	Agricultural Health Study: 29,074 spouses of private pesticide applicators of Iowa and North Carolina.	95% CI of Odds ratios: History of pesticide poisoning: 1.72 - 6.19 Low cumulative pesticide exposure: 0.91 - 1.31 High cumulative pesticide exposure: 0.91 - 1.31
Bouchard et al. (2011)	Prenatal dialkyl phosphate (DAP) concentrations are associated with poorer intellectual development in 7-year-old children.	CHAMACOS study: Birth cohort study among Latino farmworker families from California. Final sample of 329 children.	Effect: 10-fold increase in maternal DAP, DM, and DE concentrations over IQ. Coefficient (95% CI): DAP: -5.6 (-9.0 to -2.2); DM: -4.7 (-7.7 to -1.6); DE: -2.8 (-5.6 to 0.3)

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Reference	Main conclusions	Data	Quantitative Results
Brender et al. (2010)	Neural tube defects (NTD) are associated with preconception residence within 0.25 miles of cultivated fields, and use of pesticides within the home.	Data collected: 184 Mexican American case women and 225 controls interviewed regarding environmental and occupational exposure to pesticides.	Significant associations of NTD affected pregnancies with: living within 0.25 miles of cultivated fields (OR=3.6); exposure to pesticides twice (aOR=2.3) or three times (aOR=2.8).
Eskenazi et al. (2007)	Significant adverse associations of prenatal exposure to pesticides with mental development and pervasive developmental problems at 24 months old are reported.	Data collected: children's performance measures at 6 (n=396), 12 (n=395), and 24 (n=372), and urine samples (mother and child) in Latino families in California.	Significant associations between prenatal dialkliphosphate exposure with risk of pervasive developmental disorder (OR=2.3), and for postnatal exposure (OR=1.7).
Eskenazi et al. (2010)	PON1 is correlated with child neurobehavioral development, bur more research is needed to confirm if it modifies the relation with in utero OP exposure.	CHAMACOS study: Longitudinal birth cohort study among primarily Latino farmworker families at California. Final sample of 371 mothers (with respective children).	Effect of 10x increase in maternal DM DAP levels on child MDI scores, dependent on genes: PON1[-108CC]: -2.2 (p=0.45) PON1[-108CT]: -3.4 (p=0.09) PON1[-108TT]: -5.9 (p=0.03)
Guodong et al. (2012)	No significant associations are found between child urinary levels of organophosphate pesticides (OPs) metabolites and any of the developmental quotients (DQ) scores.	Data collected: 301 children between 23 and 25 months old, recruited from 2 community hospitals in Shanghai from February to October 2008.	Children have relatively higher levels of OP urinary metabolites compared with developed countries reports, but no significant association is found between these and the DQ scores.
Harari et al. (2010)	Prenatal exposure to pesticides can cause lasting adverse effects on brain development in children, even for levels not producing adverse health outcomes in the mother.	Data collected: 84 children attending a local public school in northern Ecuador, with 35 exposed to pesticides via maternal occupational exposure, and 23 with indirect exposure from paternal work.	Prenatal exposures to pesticides as predictor of adverse effects [95% CI] at 6-8 years old: Visual-performance functions: -0.7 [-1.3 to -.01] Blood pressure (Systolic): 3.3 [0.5 to 6.1]
Parrón et al. (2011)	Environmental exposure to pesticides may affect the human health by increasing the incidence of neurological disorders for the entire population living in the area exposed.	Data collected: 17,429 cases of neurological disorders collected from hospital records in the south of Spain (1998-2005).	Population living in areas with high pesticide use has an increased risk for: Alzheimer's disease (aOR=1.65); suicide attempts (aOR=1.76); polyneuropathies (males aOR=1.68).
Rastogi et al. (2010)	Some neurologic self-reported symptoms, such as headache, watering in eyes, and burning sensation in eyes or face, are the clinical manifestations most attributed to organophosphate pesticide exposure (OP).	Data collected: neurologic signs and symptoms from 225 rural children that belong to agricultural families, and 50 children as suitable reference group, in India.	Symptoms associated to OP exposure: maximum prevalence of salivation (18.22% of the cases), lacrimation (17.33%s), diarrhea (9.33%), excessive sweating (13.78%), tremors (9.3%), mydriasis (8.4%).
Rauh et al. (2011)	There exists significant effect of prenatal CPF exposure on deficits in Working Memory Index and Full-Scale IQ at 7 years of age.	Data collected: 265 children participants in a prospective study of air pollution, at Columbia Center for Children's Environmental Health.	For each sd increase in CPF exposure (4.61 pg/g) there is an IQ decline of 1.4%, and also on Working Memory by 2.8%.
Yesavage et al. (2004)	No increasing prevalence of Parkinsonism attributable to wind drift of pesticides could be found.	Data collected: VA clinical pharmacy datasets for prescription of antiparkinsonian drugs in the coastal San Francisco Bay (1997-2001), and total number of patients receiving prescription of any other kind.	There is a significant correlation between Parkinsonism and pesticide use (r=0.53), but the prevalence of the disease is not higher in the zones downwind of the prevailing winds.
DIABETES			
Everett et al. (2010)	Evidence supporting an increased risk of diabetes among gross population is strongest for heptachlor epoxide and p,p'-DDT, intermediate for oxychlordane, and least for beta-hexachlorocyclohexane, p,p'-DDE, and trans-nonachlor.	National Health and Nutrition Examination Survey, 1999-2004.	95% CI of odds ratios: beta-hexachlorocyclohexane (0.76,1.63); p,p'-DDE (0.98,2.55); p,p'-DDT (1.03,2.32); oxychlordane (0.88,1.88); trans-nonachlor (0.88,1.90); heptachlor epoxide (1.04,2.01)

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Reference	Main conclusions	Data	Quantitative Results
Son et al. (2010)	Low-dose background exposure to OC pesticides is strongly associated with prevalent type 2 diabetes in Koreans. Asians may be more susceptible to adverse effects of OC pesticides than other races.	Data collected: From participants in a community-based health survey in Korea: 40 randomly selected diabetic patients; and 40 controls.	Odds ratios in the 3rd tertile of OC pesticide (95% CI) range: Heptachlorepoxide: 3.1 (0.8-12.1) Oxychlordane: 26.0 (1.3-517.4)
RESPIRATORY			
Balluz et al. (2000)	The health complaints reported by the employees at a health center in Georgia with possible pesticide exposure were precipitated by both environmental and psychological factors. The perception of odors, inadequate air flow, and length of employment are significantly associated with the employees' health complaints.	Data collected in a health center in Georgia: Environmental monitoring for standard indoor-air environmental quality parameters, 117 questionnaires answered by workers of this center, 85 serum samples, and 90 urine samples.	Significant odds ratios of psychological predictors for: Sinusitis: Knowing someone with similar symptoms: 9.1; Work hard very often: 9.8. Eye irritation: Knowing someone with similar symptoms: 9.2 Headache: Knowing someone with similar symptoms: 6.4; Work hard very often: 4.1
WOMEN SPECIFIC DISORDERS			
Arbuckle et al. (2001)	Evidence suggesting an association between pesticide exposure and spontaneous abortions is found. Timing of exposure is important characterizing the increase in risk.	Ontario Farm Family Health Study: 2,110 women providing information on 3,936 pregnancies, including 395 spontaneous abortions.	Significant associations of early abortions with preconception exposure to: phenoxy acetic acid herbicides (OR=1.5); triazines (OR=1.4); any herbicide (OR=1.4). For late abortions, preconception exposure to: glyphosate (OR=1.7); thiocarbamates (OR=1.8); other pesticides (OR=1.5).
Garry et al. (2002)	The results suggest that miscarriage risk increase during spring is associated with specific herbicides applied by their partners, and throughout the year in spouses of applicators using fungicides.	Data collected: 802 spouses of male pesticide applicators located in the 5-county Red River Valley area, Minnesota.	Increased miscarriage risk in the spring is significantly associated with use (by the male partner) of: sulfonylurea (OR=2.1); imidizolinone (OR=2.6); herbicide Cheyenne (OR=2.9). And during the whole year with the use of fungicides: organotin (OR=1.55); EBDC (OR=1.77).
GENERAL HEALTH, MULTIPLE DISEASES AND OTHERS			
Acosta et al. (2009)	The results suggest a relationship between prenatal exposure to pesticides and placental maturity, and may affect the nutrient transport from mothers to the fetus.	Data collected: analysis of the placentas of exposed women (n=9), and non-exposed women (n=31 preterm).	Pesticide exposure significantly associated with higher placental maturity index (PMI) (betha=7.38). Birth weight and gestational age significantly correlated with PMI (r=0.54 and r=0.44, respectively).
Ahamed et al. (2006)	Results do not support any association between exposure to organochlorines and risk of childhood aplastic anaemia.	Data collected: 17 cases of childhood aplastic anaemia and 17 controls, between 3 and 12 years old, enrolled in Lucknow, India (2003-2004).	The only significant difference is found on alpha-hexachlorocyclohexane concentrations between cases (6.84) and controls (5.49).
Aminov et al. (2013)	Polychlorinated biphenyls (PCBs) and organochlorine pesticides are associated with elevations in serum lipids, cholesterol and triglycerides.	Data collected: serum samples obtained from 575 residents of Aniston, and were analyzed for pesticide residues.	Significant associations (Model 1) between pesticides exposure and: total lipids (betha=0.07); total cholesterol (betha=0.04); triglycerides (betha=0.15).
Andersen et al. (2012)	The results indicate an interaction between prenatal pesticide exposure and the PON1 gene, such that children with the R-allele develop adverse cardiovascular risk profiles.	Data collected: PON1-genotype determined for 88 pesticide exposed (prenatal) cases, and 53 controls, in Denmark (1996-2000).	Significant higher means of high exposed (prenatal) group (compared to unexposed) for: abdominal circumference (65.9cm); sum of four skin folds (52mm); body fat percentages (22.3%).
Berkowitz et al. (2004)	Data analysis suggest that chlorpyrifos may have a detrimental effect on fetal neurodevelopment among mothers who exhibit low PON1 activity, due to small head sizes of the offspring.	Data collected: 404 births between May 1998 and May 2002 at Mount Sinai Hospital in New York City.	Significant effect on head circumference of the offspring for mothers with low PON1 activity with TCPy (pesticide exposure) above the level of detection: mean = 33.3 (p=0.014).

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Reference	Main conclusions	Data	Quantitative Results
Boucher et al. (2013)	Prenatal exposure to chlordecone is associated with specific impairments in fine motor function in boys.	Data collected: study conducted in Guadaloupe, measuring exposure to chlordecone at birth (n=141), and at 3 months postpartum (n=75). Development assess by the Ages and Stages Questionnaire.	Significant differences on fine motor scores according to the chlordecone concentration levels at birth: undetected 101.5, low 103.0, high 94.8. (p=0.029 obtained from analyses of variance ANOVAs).
Burns et al. (2012)	Serum OCP concentrations on 8-9 year-old kids are associated with reduced BMI during peripubertal period.	Data collected: Cohort of 350 boys enrolled in the Russian Children's Study between 2003 and 2005 at 8-9 years of age, with serum OCPs measure at study entry.	Effect of OCPs concentration [95% CI] on BMI z-scores: HCB: -0.84 (-1.23, -0.46) beta-HCH: -1.32 (-1.70,-0.95) p,p'-DDE: -1.37 (-1.75,-0.98).
Cecchi et al. (2012)	The increase in cortisol (CT) levels in the maternal compartment may lead to impaired newborn health later in life. Results suggest an endocrine disruption during pregnancy due to environmental OPs exposure.	Data collected: 97 healthy pregnant women, between 15 and 36, entering prenatal care at the Allen Public Hospital, in Argentina, from Nov 2007 to Aug 2008.	Significant increase of CT levels during the first trimester of pregnancy: 55% (p-value <0.01).
Chevrier et al. (2008)	The exposure to polychlorinated biphenyls (PCBs) or hexachlorobenzene may affect thyroid function during pregnancy.	CHAMACOS study: serum samples from 334 pregnant women living in the Salinas Valley California (1999-2000).	Significant adjusted associations between free thyroxine and: PCBs (betha= -0.12); hexachlorobenzene (betha= -0.08).
Dabrowsky et al. (2003)	Maternal exposure to pesticides may contribute to a slight reduction in the duration of pregnancy and to a slower pace of fetal development (this last effect is of borderline 95% significance).	Data collected: 117 women delivering infants with low birth weight and 377 with average birth weights, in the region of Lodz, Poland (1998-2001).	Significant effect of pesticide exposure in 1st or 2nd trimester (compared to non-exposed) on: pregnancy duration (betha=-0.53 weeks, p=0.052); birth weight at given pregnancy duration (betha=-103g, p=0.067).
Fenster et al. (2006)	No adverse associations between maternal serum organochlorine levels and birth weight or crown-heel length is found. Some evidence regarding decreases length of gestation with lipid-adjusted hexachlorobenzene is found.	CHAMACOS study: birth cohort of 385 low-income Latinas living in the Salinas Valley community, California.	Only lipid-adjusted hexachlorobenzene levels were found significant (but small) for decreased length of gestation (betha= - 0.47 weeks). No other association is significant.
Fowler et al. (2007)	Exposure to concentrations of environmental chemicals could affect the fetal human Leydig cell, potentially leading to subtle dysregulation of reproductive development and adult fecundity.	Data collected: Human fetal tests were collected during the 2nd trimester. Testis explants were cultured for 24hours in the presence and absence of LH and dieldrin.	Significant effects of exposure to dieldrin on: reduced LH-induced testosterone secretion (p<0.05); steroid acute regulatory protein (p<0.05).
Freire et al. (2011)	Early exposure to certain environmental chemicals associated with endocrine-disruption may interfere with neonatal thyroid hormone status.	Data collected: 17 organochlorine pesticides analyzed in 220 placentas from a male birth cohort in Southern Spain.	Significant effect on the odds of thyroid-stimulating hormone cord blood levels over 5mU/L for exposure to: endrin (higher odds, OR=2.05); endosulfansulfate (lower odds, OR=0.36).
Harley et al. (2011)	Infants with specific PON1 genotypes and enzyme activity levels (suggesting susceptibility to effects of pesticides) have decreased fetal growth and length of gestation.	CHAMACOS study: 470 pregnant women from the community of Salinas Valley, California, whose urine samples were analyzed twice during pregnancy.	PON1-180TT genotype with significant associations (compared to genotype 108CC) between pesticide exposure and: shorter gestational age (betha= - 0.5 weeks); smaller head circumference (betha= - 0.4 cm).
Hjollund et al. (2004)	There is no increased risk of spontaneous abortion in invitro fertilized (IVF) treated women attributable to paternal exposure to pesticides and growth retardants.	Data collected: 128 cases of pesticide exposed partners and 2925 controls in Denmark.	The proportion of spontaneous abortions by type of exposure are: 19.7% (herbicides), 19.7%(fungicides), 21.3% (pesticides), 22.2% (growth retardants), 28.4% (controls). No statistical difference is found.
Jurewicz et al. (2005)	Infants of mothers performing heavy work inside greenhouse during pregnancy have lower mean birth weight than infants of mothers working out of greenhouse.	Data collected: 1,122 pregnancies between 1977 and 2001 (386 of greenhouse workers, 276 of non-greenhouse workers, and 460 of women who became pregnant during maternity leave).	Mean birth weight of greenhouse workers' infants is 177gr lower than that of those of mothers working out of greenhouses (p=0.05).

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Reference	Main conclusions	Data	Quantitative Results
Lee et al. (2002)	Noncancer risks are uniformly higher for children due to a proportionately greater inhalation rate-to-body weight ratio. Potential health effects include neurologic and respiratory effects, as well as cancer.	Estimation of health risks using ambient air data provided by the California Air Resources Board and the California Department of Pesticide Regulation.	Hazard quotients (HQ) estimated for non-cancer effects, 15 different pesticides, and different type of exposure. For chronic exposure, significant HQs for Methylbromide (95th percentile HQ=2.0), and 1,3-Dichloropropene (95th percentile HQ=2.0).
Levario-Carrillo et al. (2004)	Prenatal exposure to pesticides is associated to the presence of intrauterine growth retardation (IUGR).	Data collected: 79 newborns from singleton pregnancies with IUGR and 292 controls, in Chihuahua, Mexico.	Significant effect of pesticide exposure on IUGR: aOR=2.3 (95% CI: 1.0 , 5.3).
Marks et al. (2007)	In utero and postnatal dialkyl phosphate (DAP) exposure is associated adversely with attention, and this is stronger at 5 years old than at 3.5, and among boys.	CHAMACOS study: 331 children assessed at ages 3.5 years and 323 at 5 years. Different tests applied and questionnaires answered by the mother.	Prenatal DAP is associated significantly with maternal report of attention problems at age 5 years (betha=0.7), and associated with scores on the Conners' Kiddie Continuous Performance Test ADHD Confidence Index > 70th percentile (OR=5.1).
Murphy et al. (2007)	The results of this study indicate that long-term health risks to golfers from inhalation of 15 pesticides appear to be minimal in the Northeastern U.S.	Data collected: assessment of inhalation health risk for 15 pesticides typically applied to golf courses in Boston, Philadelphia, and Rochester.	For all pesticides and locations, the hazard quotient (HQ) and the incremental lifetime cancer risk (ICR) are well below critical values (1.0 and 10^-6 respectively). The largest values found are, for HQ (5.8x10^-3), and for ICR (3.7x10^-8).
Nagayama et al. (2007)	Greater exposure to dioxins, polychlorinated biphenyls (PCBs), and organochlorine pesticides influences the immune system of Japanese infants.	Data collected: peripheral blood samples analyzed from 101 Japanese infants of approximately 10 months of age.	Significant effects of: HCH on HLA-DR+ (OR=0.33); HCE on CD8+ (OR=2.34); Dioxins on CD4+/CD8+ (OR=1.71); Chlordane on CD3+ (OR=1.70).
Petit et al. (2010)	Agricultural activities in the municipality of residence (such as cultivation of pea, potato, and wheat crops) may have a negative effect on cranial growth	Data collected: 3,421 pregnant women recruited from the region of Brittany (2002-06). Area devoted to agricultural activities was obtained from the national agricultural census (2000).	Risk of infants with small head circumference is significantly higher for mothers living in municipalities where peas (OR=2.2) were grown. For potatoes (OR=1.5, 95%CI: 0.9-2.4) a potential association is also found.
Quiros-Alcala et al. (2011)	No consistent associations is found between maternal and child organophosphate pesticide exposure on children autonomic nervous system (ANS) up to age 5 years.	Data collected: number of measures of the ANS varies between 96 and 273, depending on the age of subject and type of measure. These measures were collected using the CHAMACOS study.	Only significant effect of pesticide exposure on resting ANS measures is found for 6 month old children for dimethyls (betha=-0.24) and diethyls (betha=-0.27), but not at older ages.
Rauch et al. (2012)	Prenatal urinary dialkyl phosphate (DAP) concentrations are associated with shortened gestation and reduced birth weight, with some heterogeneity depending on race and PON1 genotypes.	HOME study: 306 pregnant women attending seven prenatal clinics in the Cincinnati metropolitan area (2003-2006), providing urine samples at weeks 16 and 26.	10-fold increase in DAP concentrations are associated with a decrease in gestational age (betha= -0.5 weeks; 95% CI: -0.8,-0.1) and birth weight (betha= -151gr; 95% CI: -287,-16). Results are different between white and black newborns, and different PON1 genotypes.
Ronda et al. (2005)	Risk of fetal death in the offspring of agricultural workers exposed to pesticides around the time of conception is higher than in the offspring of nonagricultural workers in mothers who were housewives (do not work outside).	Data collected: 587,360 stillbirths and live births obtained from the Stillbirth and Birth National Register of Spain (1995-99).	Significant adjusted relative risk of fetal death in agricultural workers is 1.68 (95% CI: 1.03,2.73) for mothers not working outside (housewives), compared with their nonagricultural counterpart.

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Reference	Main conclusions	Data	Quantitative Results
Sathyanarayana et al. (2010)	Overall, no associations between birth weight and pesticide-related activities during early pregnancy are observed, except for one pesticide. However the lack of data on temporal specificity of individual pesticide exposures limits the results of this study.	Agricultural Health Study: 2,246 farm women in Iowa and North Carolina.	Statistically significant pesticides on birth weight: Carbaryl: -82gr (95% CI: -132 , -31)
Wang et al. (2012)	High pesticide levels might adversely affect duration of gestation although this association is not present among boys. Maternal urine pesticide levels in Shanghai are much higher than those reported in developed countries.	Data collected: 187 healthy pregnant women between September 2006 and January 2007, in Shanghai, China.	Effect of log unit increase in diethylphosphate, on the gestational duration in girls: -1.79 weeks (95%CI: -2.82 to -0.76).
Weselak et al. (2007)	There is suggestive evidence that allergies and hay fever are more common in offspring exposed to certain pesticides during pregnancy, but this results should be confirmed.	Ontario Farm Family Health Study: 104 cough cases, 173 asthma cases, 341 allergy cases, and 2787 controls.	Significant effect of prenatal exposure and allergies for: any pesticide (aOR=1.58), fungicides (aOR=1.69), insecticides (aOR=1.48), herbicides (aOR=1.56), phenoxy (aOR=1.43), organophosphates (aOR=1.55), and 2,4-D (aOR=1.66).
Weselak et al. (2008)	The results suggest that pre-conception exposure to cyanawine or dicamba is associated with an increase in risk of birth defects in male offspring.	Ontario Farm Family Health Study: 3,412 pregnancies analyzed (108 pregnancies ending in one or more birth defect).	Significant effect of pre-conception exposure on increased risk of birth defects in male offspring for: cyanazine (OR=4.99); diacamba (OR=2.42).
Wickerham et al. (2012)	Evidence suggests that exposure to fungicides may adversely impact fetal growth such as exposure to mixtures of multiple pesticides, but more research is needed.	Data collected: Subset of pregnant women participating in a study. Women at 36 weeks gestation and healthy were recruited from Fuyang Maternal and Children's hospital. Final sample of 113 pregnant women.	Birth weight decreased by 37.1g (95%CI: -72.5, -1.8) for each detected pesticide. In case of fungicides: -116 g (95%CI: -212, -19.2) Vinclozoling: -174g (95%CI: -312, -36.3) Acetochlor: -165g (95%CI:-325, -5.7).
Young et al. (2005)	Evidence suggests a detrimental association between prenatal organophosphate pesticides (OP) exposure and abnormal reflexes, particularly in infants assessed after 3 days of life.	CHAMACOS study: 381 infants under 2 months of age from the Salinas Valley community, California.	Increasing prenatal urinary metabolite levels are associated with the proportion of infants with more than three abnormal reflexes for: dialkylphosphates (OR=4.9); dimethyls (OR=3.2); diethyls (OR=3.4) (in kids over 3 days old.
Zhu et al. (2006)	A little effect of occupational exposures to pesticides on pregnancy outcomes among gardeners or farmers in Denmark is found.	Data collected: National Birth Cohort in Denmark, collecting information on 226 pregnancies of gardeners and 214 of farmers (1997-2003). 62,164 other workers were used as controls.	The only significant increased risk found is a very preterm birth for gardeners (aOR=2.6). But any other association is insignificant.

Table 3. Consumers

Reference	Main conclusions	Data	Quantitative Results
CANCER			
Buczynska et al. (2005)	Results indicate: low cancer risk for people drinking water contaminated with p,p'-DDT and p,p'-DDD ; low non-cancer risk related to MCPA, MCPP and methoxychlor exposure. At one dump site, exposure to 2,4'-D implies possible hematopoietic, nephrotoxic and hepatotoxic effects, and reproductive disorders. Possible health risk are calculated from daily intake in drinking polluted water.	Forty pesticide dump sites in Poland were selected as the largest source of ecological hazard. Two of them, close to residence are and drinking water intakes were selected as potentially most hazardous to health. Water samples were collected and analyzed.	Relative cancer risk for different pesticides: p,p'-DDT: R = 1.83 * 10^(-8) p,p'-DDD: R = 1.24 * 10^(-8) Hazard index for noncarcinogenic pesticides: 2,4'-D: HQ = 3
Li et al. (2008)	Fish with pesticide residuals would not pose a health risk to humans with a consumption of 7.4 to 8.6 g/person day, however the hazardous ratio of the 95th percentile for PCBs (Polychlorinated biphenyls) in fish from Gaobeidian Lake exceeds 1 so daily exposure to PCBs has an increased cancer risk.	Concentration levels of pesticides in some edible fish from Huairou Reservoir and Gaobeidian Lake in Beijing, China.	95th percentile measured concentrations and Hazard Rates (HR): DDTs (Gaobeidian Lake): 141.8, HR = 0.73 DDTs (Huairou Reservoir): 117, HR = 0.61 PCBs (Gaobeidian Lake): 33.0, HR = 1.01
Moon et al. (2009)	Hazard ratios of non-cancer risk of all of the Ocs (Organochlorines) are less than one, while the lifetime cancer risks of PCBs and DDTs are all greater than unity.	Measurement of organochlorines' levels (Ocs) in 26 species of seafood commonly consumed by the Korean population.	Hazard ratios are presented only graphically, and the values are not presented in the paper. Graphically is clear that the hazard ratios for cancer risk is larger than 1 for PCBs among all ages.
Pandit et al. (2002)	Most organochlorine compounds in milk and milk products are not present at high enough levels to cause cancer, with the exception of α-HCH, whose cancer risk estimate exceeds the US EPA guidance value.	Data collected: Coming from 520 samples of milk and milk products of different brands available in the local market selected randomly to determine concentration levels of persistent organochlorine pesticides.	Cancer potencies (mg kg-1 per d)^-1 for organochlorines identified in milk and milk products: α-HCH: 6.3; β-HCH: 1.8; γ-HCH: 1.3; DDE: 0.34; DDT: 0.34
GENERAL HEALTH, MULTIPLE DISEASES AND OTHERS			
Bhanti et al. (2007)	Pesticide residue concentrations in vegetables of different season shows that the winter vegetables are the most contaminated, followed by summer and rainy vegetables. Even if concentration levels are well below the established tolerances, continuous consumption of such vegetables can accumulate in the receptor's body and may prove to be fatal for human population in the long term.	Data collected: Residuals concentration of selected organophosphorous pesticides in vegetables grown in different seasons.	Hazard indexes as per maximum daily intakes of pesticides residue through vegetables (>1 means hazard risk): Methyl parathion: Summer (1.864); Rainy (1.984); Winter (2.868); Chlorpyriphos: Summer (0.313); Rainy (0.256); Winter (0.294); Malathion: Summer (0.165); Rainy (0.128); Winter (0.176)

Table 4. WTP studies

Reference	Country	Group	SP/RP	WTP reflects	WTP defined as
Akgüngör et al. (2001)	Turkey	Consumers	SP: CVM	H	Price premium per kg tomatoes in which pesticide residue do not cause health problems
Al-Hassan et al. (2010)	Ghana	Farmers	SP: CVM	M(P)	Percentage increase in yield or reduced pest control
Atreya et al. (2012)	Nepal	Farmers	SP: CVM + RP: DE	H – E	Price premium in relation to household's pesticide expenditures
Balcombe et al. (2007)	UK	Consumers	SP: CVM	O	Weekly price premium for a basket of food items produced using a non-pesticide technology
Balcombe et al. (2009)	UK	Consumers	SP: CE	E	Price premium on bread from complete ban or reduction in specified pesticides
Bernard and Bernard (2010)	USA	Consumers	Experiment	M(GM)	Price premium for organic potatoes and sweet corn, where organic were divided into "no pesticides" and "non-GM"
Canavari et al. (2005)	Italy	Consumers	SP: CVM	H – E – O	Yearly contribution for the abolition of the use of pesticides and a price premium for organically grown apples
Canavari and Nayga (2009)	Italy	Consumers	SP: CVM	M(GM)	NA (Asked respondents if they were willing to buy GM goods with either less pesticides (input) or nutritionally enhanced (output))
Chalak et al. (2008)	UK	Consumers	SP: CE	H – E	Price premium for bread and weakly basket of fruit and vegetables
Cranfield and Magnusson (2003)	Canada	Consumers	SP: CVM	H – E	Price premium of favorite food free of pesticide
Garming and Waibel (2009)	Nicaragua	Farmers	SP: CVM	H	Price premium per year in relation to "standard pesticide"
Gifford and Bernard (2008)	USA	Consumers	SP: CVM	O	Price premium for a variety of food products presented as "organic", "pesticide-free", and "non-gm"
Glenk et al. (2012)	UK	Consumers	SP: CE	H – E	Price premium on bottle of whisky from ban or reduction in pesticide use

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Reference	Country	Group	SP/RP	WTP reflects	WTP defined as
Govindasamy et al. (2001)	USA	Consumers	SP: CVM	M(IPM)	NA (Respondents were asked if they were willing to pay a 10% premium for IPM over the price of conventional food).
Govindasamy et al. (2005)	USA	Consumers	SP: CVM	O	NA (Asked respondents if they were willing to pay a percentage premium payment).
Haghiri and McNamara (2007)	Canada	Consumers	SP: CVM	H – E – (O)	NA (Respondents were asked if they were willing to pay a 10% premium for organically grown fresh fruit and vegetables).
Haghiri et al. (2009)	Canada	Consumers	SP: CVM	H – E – (O)	NA (Respondents were asked if they were willing to pay a 10% premium for organically grown fresh fruit and vegetables).
Hamilton et al. (2003)	USA	Consumers	SP: CVM + Vote	H – E	NA (Price premium for pesticide-free food (and vote on ban)).
Hammitt and Haninger (2010)	USA	Consumers	SP: CVM	H	WTP to reduce fatal-disease risk (caused by consuming pesticide residues on foods).
Huang et al. (2000)	USA	Consumers	SP: CVM	M(CRF)	NA (Respondents ask if they had a positive WTP for CRF (certified residue-free) produce; if yes asked to choose their relative WTP premium within percentage intervals).
Khan (2009)	Pakistan	Farmers	SP: CVM	H	NA (Price premium of pesticide that was just as effective as their current one, but with no adverse health effects).
Loureiro et al. (2002)	USA	Consumers	SP: CVM	O	Price premium for eco-labeled apples
Maguire et al. (2004)	USA	Consumers	RP: Hedonic	O	Price premium for organic baby food
Marette et al. (2012)	France	Consumers	Experiment + SP: CVM	O	Price (premium) per organic apple or apple with a “few pesticides”.
Mourato et al. (2000)	UK	Consumers	SP: CR	H – E	Per case of human illness and one species of farmland bird.
Nielsen (2013)	Germany	Consumers	Experiment	E – M(GM)	NA (Subjects asked to choose between GM and conventional fries at the same price.)
Palis et al. (2006)	Philippines	Farmers	Experiment	H	NA (Farmers’ and laborers’ were offered to buy protective equipment).

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Reference	Country	Group	SP/RP	WTP reflects	WTP defined as
Posri et al. (2007)	Thailand	Consumers	SP: CVM	H	NA (Respondents were asked if they were willing to pay a premium for "Chinese cabbage" guaranteed to be in compliance with residue limits determined by the government).
Probst et al. (2012)	Benin, Ghana, and Burkina Faso	Consumers	SP: CE	O	Price premium for plate of food served when eating out
Schou et al. (2006)	Denmark	Consumers	SP: CE	E	Price premium of bread then converted to household WTP per year for 1% increase in the survival of partridge chicks and an increase of 1 wild plant/m2.
Sydorovych and Michele (2008)	USA	Farmers	RP	H – E	Per acre per year for health risk reduction and surface water risk reduction.
Travisi and Nijkamp (2008)	Italy	Consumers	SP: CE	H – E	Annual household WTP per unit of bird species, illness, contaminated farmland.

a: SP and RP refer to stated- and revealed preferences with description of techniques used: contingent valuation (CVM), choice experiments (CE), defensive expenditures (DE)

b: H = Health, E = Environment, O = organic, M = Miscellaneous (GM = genetically modified, IPM = integrated pest management). What WTP reflects is not explicitly stated in all studies. In those cases we have interpreted what WTP reflects based on the scenario and/or questions asked.