Journal of Environment and Earth Science www.iiste.org
ISSN 2224-3216 (Paper) ISSN 2225-0948 (Online)
Vol.8, No.6, 2018

Review on Effect of Pesticide on Environment, Honey Bees and Hence on Humans

Gizaw Mohammed[1] AregaTafere*[1] Maradona Birhanu[2] Chemere Ayenew[2]
1.Wolaita Sodo University School of Veterinary Medicine
2.Alage Agricultural Technical Vocational and Education Training College

Summary
Pesticides are used to kill the pests and insects which attack on crops and harm them. Different kinds of pesticides have been used for crop protection for centuries. Pesticides benefit the crops; however, they also impose a serious negative impact on the environment. Excessive use of pesticides may lead to the destruction of biodiversity. Many birds, aquatic organisms and animals are under the threat of harmful pesticides for their survival. Pesticides are a concern for sustainability of environment and global stability. Despite their importance for human being, honey bees die with alarming speed. In recent years, in different parts of the world, due to pollution, pesticides and neglect there was registered an unprecedented rate of disappearance of honey bees. Einstein's theory, the fact that once the bees cease to exist, humanity has only four years to extinction, seems now truer than ever. Thus, the issue has gained a tone of maximum urgency; the bee crisis can entirely shatter the world food security, already affected by the economic crisis. There are plenty of factors that could cause honey bee population decline: disease, parasites, climatic factors (high temperature, drought) or decrease in the diversity of honey flora. It may sometimes happen that the beekeeper himself causes the poisoning of his honey bees, use inappropriate products which should protect the honey bees. It is therefore possible to imagine a multi-factorial explanation of problems encountered by honey bees and to underestimate the key role of pesticides. Considering these, Pesticides residues in vegetables, fruits, pulses, grains and water can cause numerous health complications like cancer, genetic defects and impotency.
Keywords: Pesticides, pollination, Honey bees, honey.

1. Introduction

Many radical changes took place in the 1950, shortly after the Second World War, when tractors replaced horses, chemical fertilizers replaced manure, the applying of pesticides using the aircraft became common, and farmers became aware of the cost of production. It was a time when humanity was not aware of the danger that pesticides brought, being satisfied to have eliminated agricultural diseases and pests. Thus, year by year, the usage of pesticides grew considerably. Perhaps the only ones who have suffered from the beginning due to agricultural modernization were honey bees and beekeepers respectively (Maini et al. 2010).

Animal pollination, mainly performed by bees, is an important ecosystem service with almost 90 percent of flowering plants and 75 percent of the world's most common crops benefiting from animal flower visitation (Klein et al. 2007). Habitat loss and fragmentation, pesticides, pathogens, climate change, invasive species, intense management of managed bees, and decreased interest in beekeeping have all been suggested as threats to bees and pollination services, but the relative importance of these drivers remains uncertain (Ollerton et al. 2011).

Intense development of agriculture and animal production has caused exposure to substances with which bees have never before come into contact. The increasing demand for food has forced farmers to use more mineral fertilizers and pesticides to generate higher yields (Sattaris et al. 2016). The residues of these substances in the form of contaminants are then transferred into grains, vegetables, and fruit (Lozowicka et al. 2014). They have also been discovered in herbs such as mint (Mentha) or lemon balm (Melissa officinal's) (Lozowicka *et al.* 2014). Recent research is uncovering diverse sub-lethal effects of pesticides on bees. Insecticides and fungicides can alter insect and spider enzyme activity, development, ovipositor behavior, offspring sex ratios, mobility, navigation and orientation, feeding behavior, learning and immune function (Van Englesdrop *et al*. 2010). Reduced immune functioning is of particular interest because of recent disease-related declines of bees including honey bees (Van Englesdrop *et al.* 2012, Cameron *et al*. 2011).

Pesticide and toxin exposure increases susceptibility to and mortality from diseases including the gut parasite *Nosema* spp. (Pettis *et al.* 2012, Vidau *et al.* 2011). These increases may be linked to insecticide-induced alterations to immune system pathways, which have been found for several insects, including honey bees, (Chaimane *et al*. 2012). Neonicotinoids and fipronil are relatively new, widely used, systemic compounds designed as plant protection products to kill insects which cause damage to crops. They are also used in veterinary medicine to control parasites such as fleas, ticks and worms on domesticated animals and as pesticides to control non-agricultural pests. Neonicotinoid insecticides and fipronil are presently used on a very large scale (Simon-Delso *et al.* 2014), are highly persistent in soils, tend to accumulate in soils and sediments, have a high runoff and leaching potential to surface and groundwater and have been detected frequently in the global environment (Bonmatin *et al*. 2014). Effects of exposure to the large-scale pollution with these neurotoxic chemicals on non-target insects and possibly other invertebrates can be expected as identified for other insecticides. However, for the majority of insect and other invertebrate species that are likely to be exposed to neonicotinoids and fipronil in agricultural or (semi)natural ecosystems, no or very little information is available about the impact of these pesticides on their biology.

While public attention has recently focused on the threat to honey bees and bumble bees from neonicotinoid (neonic) pesticides, there is growing evidence that another species may be a risk from these pervasive chemicals- humans. Many scientists now say that exposure to neonics may pose a risk to human health. Laboratory tests with cell cultures and rodents led the European Food Safety Authority (EFSA) to categorize two neonics - imidacloprid and acetamiprid as possibly impairing the developing human nervous system (Jennifer Sass, 2014).

Pesticides vary in their effects on bees. Contact pesticides are usually sprayed on plants and can kill bees when they crawl over sprayed surfaces of plants or other areas around it. Systemic pesticides, on the other hand, are usually incorporated into the soil or onto seeds and move up into the stem, leaves, nectar, and pollen of plants (Washington State Agriculture, 2006).

Of contact pesticides, dust and wet table powder pesticides tend to be more hazardous to bees than solutions or emulsifiable concentrates. When a bee comes in contact with pesticides while foraging, the bee may die immediately without returning to the hive. In this case, the queen_bee, brood, and nurse bees are not contaminated and the colony survives. Alternatively, the bee may come into contact with an insecticide and transport it back to the colony in contaminated pollen or nectar or on its body, potentially causing widespread colony death (Jennifer Sass, 2014) .

Actual damage to bee populations is a function of toxicity and exposure of the compound, in combination with the mode of application. A systemic pesticide, which is incorporated into the soil or coated on seeds, may kill soil-dwelling insects, such as grubs or mole crickets as well as other insects, including bees, that are exposed to the leaves, fruits, pollen, and nectar of the treated plants (Henry *et al*. 2012; Clinch *et al.*1973).

Based on the above background, this review paper is prepared with the objective to:

- Summarize the role of honey bees in pollination and keeping of ecosystems
- Highlight the impact of pesticide on honey bees
- Delineate the major impacts of pesticide on human through honey bee products

2. Importance of Honey Bees in Pollination

Pollination serves as the backbone of com-plex ecological systems (Heithaus, 1974) and is essential to agricultural production (Bommarco *et al*. 2013). To boost pollination and fruit production, European honey bees (Apis mellifera Linnaeus) have been introduced to agricultural systems world (Moritz *et al*. 2005). Following these introductions, A. melliferahas repeatedly naturalized and spread, now occurring in natural areas in most parts of the glob (Moritz *et al*. 2005).

The importance of bees lies not only in the production of honey, wax, propolis, royal jelly and venom, but also in the role that they have in the pollination of entomophilous crops. It was found that the growth in agricultural production, achieved by the pollinating bees, is 10 -15 times higher than the production of honey, wax, etc. (Bura, M. *et al.*, 2005). Pollination of agricultural entomophilous crops by bees is an important agro-technical measure that contributes to a natural, clean and without any additional investment growth in the production of seeds, fruit and vegetables (Bura, M. *et al.*, 2003).The honey bee is the most important commercial pollinating insect, and its importance is increasing because of the reduction in the number of wild honey bees and wild pollinators. However, the contribution of wild pollinators, such as bumblebees and orchard bees, is significant. All pollinating bees should be protected (James E. Tew, 2004).

Honey bees have a key role in agriculture and in environmental preservation. Beekeeping is a fundamental agricultural activity, not only for providing hive products as honey, pollen, wax, royal jelly etc., but also for assuring the pollination of a large number of crops. The major part of cultivated plants, in fact, needs insect pollination in order to be fecundated. It has been calculated, that 35% of the world food sources derive from insect pollinated crops Moreover, honey bees together with other pollinators provide the pollination of the spontaneous and wild vegetation, thus playing a major role in landscape and natural resource preservation and domestic honey bees strongly contributed to that (Klein *et al*., 2007).

3. Route of Exposure of Honey Bees to Pesticide

The effects of pesticide exposures on honey bee colony health have been a major concern for several years. The most direct effect that can be seen from pesticides is poisoning. These pesticide exposures are often reduced as the result of label restrictions that protect non-target insects from the unintentional exposures to pesticides. However, there are occasional honey bee deaths that occur from the improper use of pesticides and the direct exposure of these pesticides to honey bee colonies (vanEngelsdorp and Meixner, 2010).

3.1. Exposure via Direct Contact

Aerial spray contamination is one of the most common ways of exposure of bees to plant protection products. Honey bees can be directly contaminated while flying in a field during a spray treatment; even though mostly all regulations forbid pesticide use during crops flowering and with unfavorable weather conditions, this way of exposure cannot be excluded. Moreover, the grass covering in the field and the spontaneous vegetation in close proximity may result attractive for foragers, so that contamination can occur even when the spray treatment is performed out of the flowering period of the main crop. Considering that spray treatments are influenced by wind drift, the vegetation surrounding the treated field may be contaminated as well, representing additional source of residual contamination for bees. When using systemic products, they may also penetrate through the plant foliar tissue and reach the phloem, so that the residual contamination would be spread into the whole plant (Comité Scientifique et Technique, 2003; Greatti *et al*., 2003).

3.2. Exposure via Indirect Contact

Field bees are the bees most likely to be exposed to environmental toxins because they forage outside the hive later in life. They may encounter a variety of risks when leaving the hive: predators, accidents, weather, and exposure to toxins in the environment. While foraging, honey bees collect pollen from flower anthers and nectar from plant blossoms and extrafloral nectaries. Foraging honey bees may fly 2–5 miles (3.2–8 km) from their colony in any direction. Despite this large possible foraging range, honey bees do tend to forage on the richest, most nutrient-dense and abundant food sources closest to the hive. Honey bees typically forage during daylight hours when temperatures are above 55°F (12.8°C), and they reduce their activity at dawn, dusk, and during inclement weather. They forage for nectar and pollen and even collect water from sources close to their colonies. Different hives in the same location can forage on different food sources (Hooven, 2013).

As a result, hive exposure to environmental toxins can vary by hive, depending on the forage source and the pesticide residue it may contain. It is during these foraging hours and at these points of contact (foliage, pollen, nectar, water, and propolis) that bees are most likely to be exposed to pesticides and other environmental toxins present in their foraging area. Consequently, foliar surfaces, soils, and exposed pollen and/or nectar that have received direct spray, dusting, or other direct application or drift of pesticides provide an immediate route of exposure to field or foraging bees. Systemic pesticides, which may be applied by soil treatment, seed treatment, direct injection, or foliar applications, are translocated within the treated plants and may be expressed in the nectar, pollen, or various plant tissues (Hooven, 2013).

3.3 Exposure via Ingestion

The systemic characteristic of several plant protection products provides the translocation of the active ingredient through the phloem towards all the plant tissues; as a consequence, pollinators and among them, honey bees, are likely to be exposed to these products by feeding nectar and pollen. In particular, the pesticide presence in pollen has been proven to be a consequence of field treatments both for aerial spray and seed treatments. In the first case, for example, a field case study reported the presence of insecticides diazinon and thiacloprid and fungicide difenoconazole in pollen loads 10 days after the treatment in an apple orchard (Skerl *et al.*, 2009).

3.4 Combined Exposure to Multiple Pesticides

Honey bees can be exposed in the field to combination of pesticides both whenever an area is involved in consecutive treatments with different products, and when a mixture of products is used for a single treatment. Depending on the type of the treatment, the ways of exposure could also be different. The in-hive contamination occurs via pollen, wax and nectar and, as highlighted by several researches (Chauzat et al., 2006; Mullin et al., 2010), honey bees can come in contact with a large number of active ingredients, among which, synergies are possible.

Knowledge on the combined toxicity of multiple chemicals in wild bees is still limited. However, such scientific evidence in honeybees is increasingly available, mostly from laboratory studies dealing with the acute toxic effects (LD_{50}) of compounds commonly found in bee matrices (i.e. pollen, honey and wax) namely insecticides (e.g. organophosphates, neonicotinoids), fungicides, antibiotics and acaricides (e.g. varroacides). The mechanisms by which combined exposure to chemicals may exert their toxicity include dose addition, response addition and interactions. Interactions include synergy, for which an increase in toxicity occurs from the resulting interactions between the chemicals, and antagonism, for which a decrease in mixture toxicity is observed (ANSES, 2015; Johnson, 2015; Quignot et al., 2015; Spurgeon et al., 2016; Thompson, 2012).

4. Risk Associate with Pesticide Use

4.1. Pesticide Risk to Environment

Risks associated with pesticide use have surpassed their beneficial effects. Pesticides have drastic effects on non-target species and affect animal and plant biodiversity, aquatic as well as terrestrial food webs and ecosystems. According to Majewski and Capel (1995), about 80–90 % of the applied pesticides can volatilize within a few days of application (Majewski and Capel 1995). It is quite common and likely to take place while using sprayers. The volatilized pesticides evaporate into the air and subsequently may cause harm to non-target organism. A very good example of this is the use of herbicides, which volatilize off the treated plants and the vapors are sufficient to cause severe damage to other plants (Straathoff, 1986). Uncontrolled use of pesticides has resulted in reduction of several terrestrial and aquatic animal and plant species. They have also threatened the survival of some rare species such as the bald eagle, peregrine falcon and osprey (Helfrich *et al*., 2009). Additionally, air, water and soil bodies have also being contaminated with these chemicals to toxic levels. Among all the categories of pesticides, insecticides are considered to be most toxic whereas fungicides and herbicides are second and third on the toxicity list. Pesticides enter the natural ecosystems by two different means depending upon their solubility. Water soluble pesticides get dissolve in water and enter ground water, streams, rivers and lakes hence causing harm to untargeted species. On the other hand, fat soluble pesticides enter the bodies of animals by a process known as "bioamplification" . They get absorbed in the fatty tissues of animals hence resulting in persistence of pesticide in food chains for extended periods of time (WHO, 2010).

4.2. Risk of Toxic Honey

Honey produced from flowers of certain plants can cause honey intoxication and various symptoms such as dizziness, weakness, sweating, nausea, vomiting, hypotension, shock, and arrhythmia and death might be encountered. Basically, some substances are toxic to humans but are not toxic to bees (Koca and Koca, 2007). Nectar of certain plants produced toxic and sometimes fatal honey. Fermented honey produces ethanol which is toxic (Okuyan *et al*., 2007). Indeed, there are many reports discussing toxic honey, but few observational studies in scientific literature have been reported so far (Ozhan *et al*., 2004).

5. The Impact of Pesticides on Honey Bees

Honey bees' poisoning with pesticide may be: acute and chronic. In case of acute poisoning the toxic reactions are violent and occur unexpectedly, honey bees die from contact or ingestion of pesticides. Chronic poisoning are those in which toxicity is absorbed in small and repeated doses, reactions occur slowly, but continuously, they may involve the death of the entire family (Charrière *et al*., 1999).

5.1. The Acute Effect

The acute toxicity of pesticides on bees, which could be by contact or ingestion, is usually quantified by LD_{50}. Acute toxicity of pesticides causes a range of effects on bees, which can include agitation, vomiting, wing paralysis, arching of the abdomen similar to sting reflex, and uncoordinated movement. Some pesticides, including Neonicotinoids, are more toxic to bees and cause acute symptoms with lower doses compared to older classes of insecticides (indicated in table 1). Acute toxicity may depend on the mode of exposure, for instance, many pesticides cause toxic effects by contact while Neonicotinoids are more toxic when consumed orally. The acute toxicity, although more lethal, is less common than sub-lethal toxicity or cumulative effects (Washington state, 2006).

Table 1. Acute effect concentrations of neonicotinoids for workers of the honey bee (*A. mellifera*) by oral and contact exposure as determined in different laboratory studies

Neonicotinoids	Exposure	LD_{50} (μg bee^{-1})	References
Acetamiprid	Contact: individual bee (acute; no info on concentration range)	24 h: 7.07	Iwasa et al. (2004)
Acetamiprid	Contact + oral: individual bee (acute; no information on concentration range)	48 h: 14.5 (oral) + 8.09 (contact)	Decourtye and Devillers (2010)
Clothianidin	Contact: individual bee (acute; no information on concentration range)	24 h: 0.022	Iwasa et al. (2004)
Clothianidin	Contact + oral: individual bee (acute; no information on concentration range)	48 h: 0.044 (contact) +0.003 (oral)	Decourtye and Devillers (2010)
	Contact to dry residue: 75–1.5 mg l^{-1} (2 days exposure) Oral: 75 mg l^{-1} to 7.5 μg l^{-1} (acute; 3 days)	No LD_{50} determined but 100% loss at 15 mg l^{-1} after 48 h 48 h: 0.003 μg l^{-1}	Laurino et al. (2011)
Dinotefuran	Oral: individual bee (acute; no information on concentration range)	48 h: 0.023	Decourtye and Devillers (2010)
Nitenpyram	Contact: individual bees (acute; no information on concentration range)	24 h: 0.138	Iwasa et al. (2004)
Thiacloprid	Contact + oral: individual bees (acute; no information on concentration range)	48 h: 38.8 (contact) + 17.3 (oral)	Decourtye and Devillers (2010)
Thiamethoxam	Contact: individual bees (acute; no information on concentration range)	24 h: 0.03	Iwasa et al. (2004)
Thiamethoxam	Contact + oral: individual bees (acute; no information on concentration range)	48 h: 0.024 (contact) + 0.005 (oral)	Decourtye and Devillers (2010)

5.2. The Sub lethal and Chronic Effect

Sub lethal doses or concentrations are defined as the quantities of substance that do not entail significant mortality effect. Nevertheless, the study of chronic effects of sublethal doses on mortality has well demonstrated that the duration of exposure may strongly influence the mortality effect. For example, the ingestion of imidacloprid sublethal concentrations for 10 days or 40 days, might lead to a high mortality, ranging from 50 to 100 % (Suchail *et al*., 2001; Dechaume Moncharmont *et al*., 2003).

Field exposure of bees to pesticides, especially with relation to neonicotinoids, is most commonly sub-lethal. Sub-lethal effects to honey bees are of major concern and include behavioral disruptions such as disorientation, reduced foraging, impaired memory and learning, and a shift in communication behaviors. Additional sub-lethal effects may include compromised immunity of bees and delayed development (Henry *et al.*, 2012).

Neonicotinoids are especially likely to cause cumulative effects on bees due to their mechanism of function as this pesticide group works by binding to nicotinic acetylcholine receptors in the brains of the insects, and such receptors are particularly abundant in bees. Over-accumulation of acetylcholine results in paralysis and death (Perry *et al*., 2012).

The sub-lethal doses of insecticides affect the movement of honey bees (Parathion), harvesting and transport of nectar (Diazon) flight guidance (Deltamethrin) (Desneux *et al.*, 2007) observed at honey bees changes in the development, survival, fertility and queen capacity of laying eggs, the ability of honey bees to orient over short distances (using visual or olfactory memory,) the ability of honey bees to orient over long distances (orientation dependent on the sun and the associated memory to this capacity), feeding behavior and ability to improve, the intensity of feeding, thermoregulation. Also, plant protection products (PPP) are likely to reduce the life of honey bees (essential parameter for the harvest), their immune capacity and other behaviors necessary to the integrity of the colony and good natural development of honey bees, such as honey bee brood feeding, all behaviors leading to swarming, building combs and the balance between cells of drones and cells of worker honey bees, looking for a new nest during swarming and transferring of information to other initiating honey bees (Colin *et al*., 2004).

5.2.1. Sub lethal Effects on Reproduction

Reproduction is an important process to assure the further existence of the colony. Indeed, a loss of reproduction (brood) might be more detrimental for the colony than the loss of older bees (foragers) (Decourtye and Devillers, 2010). This is further supported by studies on the division of tasks in bee colonies. For example in bumble bees (*B. impatiens*) task division is a dynamic process (weak task specialization) and so workers perform multiple tasks during their lifespan (Jandt and Dornhaus, 2009). Therefore it is not unlikely that foragers are replaced by other bees when enough nurses are present in the hive. A few studies have demonstrated the adverse effects on larval development following exposure to imidacloprid (Tasei *et al*., 2000, 2001; Decourtye *et al*., 2005; Abbott *et al*. 2008; Gregorc and Ellis, 2011). Decourtye *et al*. (2005) reported a delay in the time needed for honey bee larvae to hatch or develop as an adult when fed with food contaminated with imidacloprid at 5 μg kg^{-1}. Similar observations were also made by Abbott *et al*. (2008) for *O. lignaria* when imidacloprid was dosed at 30–300 μg kg^{-1} food. Also for bumble bees (*B. terrestris*) a reduction of the brood (larvae) was seen in micro-colonies orally exposed to contaminated sugar water (10 μg kg^{-1} imidacloprid) + pollen (6 μg kg^{-1} imidacloprid) (Tasei *et al*., 2000)

5.2.2. Sublethal Effect on Behavior

Neurotoxic compounds such as neonicotinoids were also reported to interfere with the orientation process of honey bees. Associative learning between a visual mark and a reward (sugar solution) in a complex maze showed that only 38% of the bees found the food source after oral ingestion of thiamethoxam at 3 ng bee^{-1} compared to 61% in the control group (Decourtye and Devillers, 2010). In another study using marked foragers that were first trained to forage on artificial feeders, (Bortolotti *et al*., 2003) noticed that a 500m distance between the hive and the feeding area resulted in no foragers at the hive/feeding area up to 24 h after treatment when foragers were fed with imidacloprid at 500 and 1,000 μg l^{-1}. The latter authors also found that a lower concentration (100 μg l^{-1} imidacloprid) caused a delay in the returning time (to hive or feeding area) of the foragers. Based on these results it is obvious that neonicotinoids interfere with the foraging capacity of bees (Ramirez-Romero *et al*., 2005 and Yang *et al*., 2008).

5.2.3. Effects on overwintering of bees

During the last years a loss of overwintering bee colonies was noticed. Although identification of the causes of this disappearance is difficult, it was argued that reduced bee health might be initially caused by the chronic exposure to pesticides. So far only two studies have been conducted in this context for neonicotinoids. Using 8 honeybee colonies (Faucon *et al.*, 2005) demonstrated that chronic exposure during the summer season (33 days) to 0.5 and 5.0 μg l^{-1} imidacloprid in saccharose syrup did not affect the overwintering abilities of honey bees. Similarly, spring assessment of colony development (brood, worker biomass and colony health) was not affected in overwintered colonies that had foraged on flowering canola grown from seed treated with clothianidin at 0.4 mg kg^{-1}, representing the highest recommended rate. In conclusion, these studies demonstrated no long-term effects on honeybee colonies of environmentally relevant concentrations (Cutler and Scott-Dupree, 2007).

5.3. Synergistic Effect

Synergistic effects occur when exposure to two or more products has more than an additive effect. When pesticides are combined, this can result in more toxicity than the additive effects of both pesticides. The use of multiple pyrethroids can cause synergistic effects because they are all detoxified through the same pathway (Johnson et al., 2006). Synergistic effects have also been observed between different classes of pesticides, including fungicides and insecticides applied in crops and in hive miticides applied by beekeepers. Several fungicides have been shown to interact synergistically with pyrethroid insecticides, increasing their toxicity for both honey bees and bumblebees. There are no label restrictions on mixing pesticides that could produce adverse synergistic effects on pollinators (Sanchez-Bayo, 2014).

5.4. Residues in bee-collected pollen, bees, and honey and wax

Neonicotinoid residues in plants and plant parts only become of importance for bees once they are exposed. The most relevant measures of exposure are the concentrations in bee-collected plant materials, such as pollen, bee products like bee bread, honey and beeswax, and in the bees themselves. The average imidacloprid residue levels in positive pollen samples ranged between 0.9 and 3.1 μg kg^{-1}, while levels in honey and beeswax were generally lower. Concentrations of 6-chloronicotinic acid were only exceeding the limit of detection in the studies of Chauzat *et al*. (2006, 2009, 2011), with average concentrations of 1.2 (>0.3–9.3) μg kg^{-1} and 1.2 (>0.3–10.2) μg kg^{-1} in pollen and honey, respectively. Other studies reported in general lower frequencies of imidacloprid presence in pollen, honey and beeswax samples. Nguyen *et al*. (2009), who sampled in an area with 13.2% of the maize crop receiving seed dressing, detected imidacloprid in 8.4% of the honey samples, but levels were always below the limit of quantification (0.5 μg kg^{-1}).

6. Impact of Pesticide on Human

History has revealed that humans had used bee products such as honey for thousands of years in all societies worldwide. Honey has been mentioned in the Talmud, the old and new testaments of the Bible, and the Holy Quran (1400 years ago). There is a large chapter (SORA) in Holey Quran named Bee (Al Nahl) and part of it says " And thy LORD taught the bee to build its cells in hills, on trees and in men's habitations, then to eat of all the produce of the earth and find with skill the spacious paths of its LORD, there issues from within their bodies a drink of varying colors, wherein is healing for men, verily in this is a sign for those who give thought." The Muslim prophet Mohammed even recommended the use of honey for the treatment of diarrhea (Green Nood, 1995). Honey has been used to treat coughs and sore throats, infected leg ulcers, earaches, measles, eye diseases, and gastric ulcers (Ankra-Badu, 1992; Obi *et al.*, 1994; P.J.Imperato and Traore, 1969). Bee products are natural food products; they are rich in minerals, antioxidants, and simple sugars. Honey is known to be rich in both enzymatic and non enzymatic antioxidants. Honey can also prevent deteriorative oxidation reactions in foods, such as the browning of fruit and vegetables and lipid oxidation in meat, as well as inhibit the growth of food borne pathogens and microorganisms that cause food spoilage(Chan *et al*., 2000; Mundo *et al*., 2004).

Others and we have found that honey has potential therapeutic properties in infections, wound healing, and cancer (Al-Waili and Saloom, 1999; Al-Waili *et al*., 2011; Othman, 2010; Parmar *et al*., 2009). Therefore, in recent years, bee products have received renewed interest as an essential natural resource that can be employed in new therapies free from side effects that are often encountered with the use of synthetic chemical medicines. However, the market competition on these products imposes extra conditions that can only be ensured by complying with quality assurance and certification protocols (Kelly *et al*., 2005).

Bee products, including honey are pollinated via different sources of contamination. Environmental contaminants include pesticides, heavy metals, bacteria, and radioactive materials. Pesticide residues have been shown to cause genetic mutations and cellular degradation. In addition to the public health problems, the presence of pesticides in bee products decreases its quality. According to European Union regulations, honey as a natural product must be free of chemicals. Poisoning of bee pollinators is a serious adverse effect of insecticide use, which leads to a decrease in the insect population, reduction of honey yields, destruction of plant communities, presence of insecticide residues in food, and ultimately to a significant loss of a beekeepers' income. Basically, the main purposes for monitoring bee products are consumer health protection, international commercial competition, and better quality (Directive, 1974).

6.1. Health Impact of Pesticides on Human

Systemic introduction of pesticides into nectar and pollen may have direct consequences for honey bee health and ultimately lead to pesticide contamination of honey-containing food. The effects of pesticides on human health are harmful based on the toxicity of the chemical and the length and magnitude of exposure. Aberrantly, farm workers and their families have the greatest exposure to agricultural pesticides. Children are most susceptible and sensitive to pesticides due to their small size and underdevelopment. Importantly, the chemicals have the ability to bio accumulate and biomagnify and can bio concentrate in the body over time (Lorenz, 2009).

Effect of exposure to pesticides ranges from mild skin irritation to birth defects, tumors, genetic changes, blood and nerve disorders, endocrine disruption, and even coma or death. Some pesticides, including Aldrin, chlordane, DDT, dihedron, endrin, heptachlor, hexachlorobenzene, mirex, and toxaphene, are considered persistent organic pollutants (POPs) (Ritter *et al*., 2007). POPs may compromise endocrine, reproductive, and immune systems. Many diseases such as cancer; neurobehavioral disorder, infertility, and mutagenic effects might result from chronic exposure. Therefore, some POPs have been banned while others continue to be used (Centers for Disease Control and Prevention, 2007, Lim *et al*., 2010).

7. Conclusion and Recommendation

Pesticides are substances used to eliminate unwanted pests. Unfortunately, honey bees are insects and are greatly affected by pesticides. Exposure of honey bees to pesticides is a continuous challenge because each year there are new pesticides coming out with new formula, one only being potentially devastating for bees. Pollination of agricultural crops by bees contributes to a natural, clean and without any additional investment growth in the production of seeds; fruit and vegetables. There are several ways honey bees can be killed by insecticides. One is direct contact of the insecticide and a honey bee during this there is death and bees do not return to the hive. In this case the queen, brood and nurse bees are not contaminated and the colony survives. The second more deadly way is when the bee comes in contact with an insecticide and transports it back to the colony, either as contaminated pollen or nectar or on its body. The main symptom of honey bee pesticide kill is large numbers of dead bees in front of the hives. Another symptom is a sudden loss of the colony's field force. After a honey bee pesticide loss the colony may suffer additionally from brood diseases and chilled brood. Generally pesticide have a great impact both on honey bees and human and this needs attention.

Based on the above conclusions, the following recommendations are forwarded:

- There should be a plan agreed between the sower and the bee keeper to minimize the use of pesticides, if bees are used to pollinate the crop
- Awareness of bee hive locations before pesticide are applied should be consider
- Determine what types of chemical products are the most appropriate tools for ecologically based pest

management
- Use insecticides only when needed and avoiding it when the crops are in a bloom
- Explore the most promising opportunities to increase benefits and reduce honey bee health and environmental risks of pesticide use.
- In order to protect honey bees from harmful pesticides, beekeepers, growers, government officials, and applicators should have a good deal of co-operation with each other

8. Reference

Abbott V.A., Nadeau J.L., Higo H.A., Winston M.L.(2008). Lethal and sub lethal effects of I midacloprid on *Osmia lignaria* and clothianidin on *Megachile rotundata* (Hy menoptera: Megachilidae*) J Econ Entomol*.;101:784–796.

Al-Waili N.S., and Saloom K.Y., (1999). “Effects of topical honey on post-operative wound infections due to gram positive and gram negative bacteria following caesarean sections and hysterectomies,” *European Journal of Medical Research*, vol. 4, no. 3, pp. 126–130.

Al-Waili N.S., Saloom K.y., and Al-Ghamdi A.A., (2011). “Honey for wound healing, ulcers, and burns; data supporting its use in clinical practice,” *The Scientific World Journal*, vol. 11, pp. 766–787.

Al-Waili N.S., Saloom K.y., Butler G., and Al Ghamdi A.A., (2011). “Honey and microbial infections: a review supporting the use of honey for microbial control,” *Journal of Medicinal Food*, vol. 14, no. 10, pp. 1079–1096.

American bumble bees (2011). Proceedings of the National Academy of Sciences 108: 662–667

Ankra-Badu G.A., (1992). “Sickle cell leg ulcers in Ghana,” *East African Medical Journal,* vol. 69, no. 7, pp. 366–369.

ANSES ((2015). (Agence Nationale de Sécurité Sanitaire Alimentation, Environnement, Travail*) Co-exposition des abeilles aux facteurs de stress. Avis de l'ANSES Saisine n° 2012-SA-0176Rapport d'expertise* (collective 252 pp

Bommarco, R., D. Kleijn, and S.G. Potts. (2013). Ecological intensification: Harnessing eco-system services for food security. *Trends in Ecology and Evolution* 28:230-238.

Bonmatin J.M., Moineau I., Charvet R., Colin M.E., Fleche C., Bengsch E.R. (2005).Behaviour of imidacloprid in fields: toxicity for honey bees. *In: Lichtfourse E, Schwarzbauer J, Robert D (eds) In environmental chemistry: green chemistry and pollutants in ecosystems. Springer,* New York, pp 483–494.

Bortolotti L., Montanari R., Marcelino J., Medrzycki P., Maini S., Porrini C. (2003). Effect of sub-lethal imidacloprid doses on the homing rate and foraging activity of honey bees. *Bull Insectol*.;56:63–67.

Bura, M., Patruica, S., Bura, V. Al., Tehnologie apicola; Ed. Solness, (2005).Timisoara.

Bura, M., Patruica, S., Grozea, I.: Buletinul AGIR, 2003, VIII (4), 79-82.

Cameron S.A., Lozier J.D., Strange J.P., Koch J.B., Cordes N. (2011). Patterns of widespread decline in North

Chaimanee V., Chantawannakul P., Chen Y., Evans J.D., Pettis J.S. (2012). Differential expression of immune genes of adult honey bee (*Apis mellifera*) after inoculated by *Nosema ceranae*. *Journal of Insect Physiology* 58: 1090–1095.

Charrière, J.D. and Imdorf, A. (1999). Protection of honey combs from wax moth damage, *American Bee Journal*, 139(8), 627–630.

Chauzat M.P., Faucon J.P., Martel A.C., Lachaize J., Cougoule N., Aubert M. (2006). A survey of pesticide residues in pollen loads collected by honey bees in France. *Journal of Economic Entomology* 99(2):253-262.

Chen L., Mehta A., Berenbaum M., Zangerl A.R., and Engeseth N.J., (2000). “Honeys from different floral sources as inhibitors of enzymatic browning in fruit and vegetable homogenates*,” Journal of Agricultural and Food Chemistry,* vol. 48, no. 10, pp. 4997–5000,.

Clinch, P. G.; Palmer-Jones, T.; Forster, I. W. (1973). "Effect on honey bees of dicrotophos and methomyl applied as sprays to white clover". *New Zealand Journal of Experimental Agriculture*. 1: 97–99.

Colin, M.E., Bonmatin, J. K. M., Moineau, I., Gaimon, C., Brun, S. and Vermandere, J. P. (2004). A method to quantify and analyse the foraging activity of honey bees : relevance to the sublethal effects induced by systemic insecticides, *Arch. Environn. Contamin. Toxico* l47, 387 – 395.

Comité Scientifique et Technique C. (2003). Imidaclopride utilisé en enrobage de semences (Gaucho) et troubles des abeilles. Rapport du Comité scientifique et Technique de l’étude multifactorielle des troubles des abeilles remis au Ministère de l’Agriculture Paris

Cutler G.C., Scott-Dupree C.D. (2007). Exposure to clothianidin seed-treated canola has no long-term impact on honey bees. *J Econ Entomol*. ; 100:765–772.

Cutler G.C., Scott-Dupree C.D. (2007). Exposure to clothianidin seed-treated canola has no long-term impact on honey bees. *J Econ Entomol*.;100:765–772.

Dechaume Moncharmont F., Decourtye A., Henneguet-Hantier C., Pons O., Pham - Delègue M.H.

(2003). Statistical analysis of honeybee survival after chronic exposure to insecticides. *Environmental Toxicology and Chemistry* 22 (12):3088-3094.

Decourtye A. & Devillers J. (2010). Ecotoxicity of neonicotinoid insecticides to bees. In: Thany SH, editor. Insect nicotinic acetylcholine receptors. 1. *New York: Sprin ger*; pp. 85–95.

Decourtye A., Devillers J., Genecque E., Menach K., Budzinski H., Cluzeau S., Pham-Del è gue M.H.(2005). Comparative sublethal toxicity of nine pesticides on olfacto ry learning performances of the honeybee *Apis mellifera*. *Arch Environ Cont am Toxicol*.;48:242–250.

Desneux, N., Decourtye, A. and Delpuech, J. M. (2007). The sublethal effects of pesticides on beneficial arthropods*, Annual Review of Entomology*, 52, 81-105

Directive, (1974). 74/409/eec: The harmonization of the laws of the member states relating to hone.

Garcia-Chao M, Jesus Agruna M, Flores Calvete G, Sakkas V, Llompart M, Dagnac T.(2010). Validation of an off line solid phase extraction liquid chromatography–tandem mass spectrometry method for the determination of systemic insecticide residues in honey and pollen samples collected in apiaries from NW Spain. *Anal Chim Acta.*;672:107–113

Greatti M, Sabatini AG, Barbattini R, Rossi S, Stravisi A (2003). Risk of environmental contamination by the active ingredient imidacloprid used for corn seed dressing. Preliminary results. Bulletin of Insectology 56 (1):69-72.

Greenwood D. (1995). “Sixty years on: antimicrobial drug resistance comes of age,” *The Lancet*, vol. 346, supplement 1, p. S1,.

Gregorc A, Ellis JD. (2011). Cell death localization in situ in laboratory reared honey bee (*Apis mellifera* L.) larvae treated with pesticides. Pest Biochem Physiol. 99: 2 00 –207.

Heithaus, E.R., (1974). The role of plant-pollina-tor interactions in determining community structure. *Annals of the Missouri Botanical Garden 61*:675-691.

Helfrich LA, Weigmann DL, Hipkins P, Stinson ER (2009): Pesticides and aquatic animals: a guide to reducing impacts on aquatic systems. In*: Virginia Polytechnic Institute and State University.*

Henry, M.; Beguin, M.; Requier, F.; Rollin, O.; Odoux, J. -F.; Aupinel, P.; Aptel, J.;Tchamitchian, S.; Decourtye, A. (2012). "A Common Pesticide Decreases Foraging Success and Survival in Honey Bees". *Science*. 336 (6079): 348–350. doi:10.1126/science.1215039. PMID 22461498.

Henry, M.; Beguin, M.; Requier, F.; Rollin, O.; Odoux, J. -F.; Aupinel, P.;Aptel,J.; Tchamitchian,S.;Decourtye,A.(2012):"ACommonPesticideDecreases Fora ging Success and Survival in HoneyBees". *Science*. 336 (6079): 348–3

Hooven, L., R. Sagili, and E. Johansen.(2013). “How to Reduce Bee Poisoning from Pesticides.” Pacific Northwest Extension Publication, PNW 591, Oregon State University, University of Idaho, Washington State University, 35 pp.

Iwasa T., Motoyama N., Ambrose J.T., Roe M.R. (2004). Mechanism for the differential toxicity of neonicotinoid insecticides in the honey bee, *Apis mellifera*. Crop Prot.;23:371–378.

James E. Tew,(2004). Backyard Beekeeping

Jandt JM, Dornhaus A. (2009). Spatial organization and division of labour in the bum ble bee *Bombus impatiens*. *Anim Behav*.77:641–651.

Jennifer Sass (2014). Neonicotinoid pesticides - bad for bees, and may be bad for people tooSeptember 18, 20

Johnson, R., Wen Z, Schuler M, Brenbaum M. (2006). Mediation of Pyrethro Insecticide Toxicity to Honey Bees (Hymenoptera: Apidae) by Cytochrome P450Monooxygenases. *Journal of Economic Entomology* 99, 4: (1046-1050).23.

Kelly S., Heaton K., and Hoogewerff J., (2005). “Tracing the geographical origin of food: the application of multi-element and multi-isotope analysis*,” Trends in Food Science and Technology*, vol. 16, no. 12, pp. 555–567.

Klein A-M, Vaissière BE, Cane JH, Steffan-Dewenter I, Cunningham SA, Kremen C, Tscharntke T (2007). Importance of pollinators in changing landscapes for world crops. vol 274. First published. doi:10.1098/rspb.2006.3721

Klein AM, Vaissière BE, Cane JH, Steffan-Dewenter I, Cunningham SA, Kremen C, et a (2007). Importance of pollinators in changing landscapes for world crops. Proc R Soc B.;274: 303313. pmid: 17164193

Koca I., and Koca A., (2007). “Poisoning by mad honey: a brief review,” *Food and Chemical Toxicology*, vol. 45, no. 8, pp. 1315–1318.

Laurino D, Porporato M, Patetta A, Manino A. (2011). Toxicity of neonicotinoid insecticides to honey bees laboratory tests. *Bull Insectol*.;64:107–113.

Lim, Y. M. Cho, K. S. Park, and H. K. Lee, (2010). “Persistent organic pollutants, mitochondrial dysfunction, and metabolic syndrome,” *Annals of the New York Academy of Sciences*, vol. 1201, pp. 166–176.

Lorenz E.S., (2009). “Potential health effects of pesticides,” *Ag Communications and Marketing*, pp. 1–8,

Łozowicka B., Abzeitova E., Sagitov A., Kaczynski P.,Toleubayev K., Li A. (2015). Studies of pesticide residues in tomatoes and cucumbers from Kazakhstan and the associated health risks. *Environ. Monit.Assess* .(1876),

Łozowicka B., Jankowska M., Rutkowska E., Hrynko I., Kaczyński P., Micińskij. (2014): The evaluation of a fast and simple pesticide multiresidue 792 Migdał P., et al.method in various herbs by gas chromatography. *J. Nat*. Med. 68, 95).

Maini, S., Medrzycki, P., Porrini, C, (2010). The puzzle of honey bee losses: a *brief review,Bulletin of Insectology*, 63(1), 153-160.

Majewski M, Capel P (1995). Pesticides in the atmosphere: distribution, trends, and governing factors. Pesticides in the hydrologic system, vol 1. *Ann Arbor Press Inc., Boca Raton, FL*, p 118

Monaco JT, Weller SC, Ashton FM (2002). Herbicide registration and environmental impact.

Moritz, R.F., S. Härtel, and P. Neumann. (2005). Global invasions of the western honeybee (Apis mellifera) and the consequences for biodiversity. *Ecoscience* 12:289-301.

Mundo M.A., Padilla-Zakour O.I., and Worobo R.W., (2004). "Growth inhibition of foodborne pathogens and food spoilage organisms by select raw honeys," *International Journal of Food Microbiology*, vol. 97, no. 1, pp. 1–8.

Okuyan E., Uslu A., and Ozan M., (2010). "Cardiac effects of "mad honey": a case series*," Clinical Toxicology*, vol. 48, no. 6, pp. 528–532.

Ollerton J, Winfree R, Tarrant S. (2011). How many flowering plants are pollinated by animals? *Oikos.*;120: 321–325. On Bee Supporting Publications 2012:EN-340 (204 pp.)

Othman N.H., (2011). "Honey and cancer: sustainable inverse relationship particularly for developing nations-a *review," Evidence-Based Complementary and Alternative Medicine*, vol. 2012, Article ID 410406, p. 10, 2012.

Özhan H., Akdemir R., Yazici M., Gündüz H., Duran S., and Uyan C., (2004). "Cardiac emergencies caused by honey ingestion: a single centre experience*," Emergency Medicine Journal,* vol. 21, no. 6, pp. 742–744,.

Perry, Trent; Chan, Janice Q.; Batterham, Phil; Watson, Gerald B.; Geng, Chaoxian; Sparks, Thomas C. (2012). *"Effects of mutations in Drosophila nicotinic acetylcholine receptor subunits on sensitivity to insecticides targeting nicotinic acetylcholine receptors". Pesticide Biochemistry and Physiology. 102: 56–60. doi:10.1016/j.pestbp.2011.10.010*.

Pettis JS, vanEngelsdorp D, Johnson J, Dively G (2012). Pesticide exposure in honey bees results in increased levels of the gut pathogen *Nosema*. *Naturwissenschaften* 58 99: 153–1

Pollinator protection requirements for Section 18 Emergency Exemptions and Section 24(c) special local need registration in Washington State; Registration Services Program Pesticide Management Division Washington State Dept of Agriculture, Dec 2006.

Quignot N., & Audrey G., (2015). Amzal Data Collection on Combined Toxicity of Multiple Chemicals for Animal Health and Ecological Risk Assessment. EFSA Supporting Publication 2015:EN-861 (112 pp.)

R.M. (2015). Johnson Honeybee toxicology Annu. Rev. Entomol. 60 (1) , pp. 415-434, 10.1146/*annurev-ento-*011613-162005CrossRef

Ramirez-Romero R, Chaufaux J, Pham-Delegue MH.(2005). Effects of Cry1Ab protoxin, deltamethrin and imidacloprid on the foraging activity and the learning performances of the honeybee *Apis mellifera*, a comparative approach*. Apidologie*; 36:601–611.

Ritter L., Solomon K.R., Forget J., Stemeroff M., and O'Leary C.,(2007). Persistent organic pollutants: an assessment report on: DDT, Aldrin, Dieldrin, Endrin, Chlordane, Heptachlor, Hexachlorobenzene, Mirex, Toxaphene, Polychlorinated Biphenyls, Dioxins and Furans. *Prepared for The International Programme on Chemical Safety (IPCS), within the framework of the Inter-Organization Programme for the Sound Management of Chemicals* (IOMC).

Sanchez-Bayo, F. and K. Goka. (2014). Pesticide Residues and Bees - A Risk Assessment*. PloS One*, 9(4)

Sattari 7 S.Z., Bouwman A.F., Martinez Rodríguez R.,Beusen A.H.W., Ittersum M.K. (2016). Negative global phosphorus budgets challege sustainableintensification of grasslands*. Nat. Commun*. (10696).

Simon-Delso N, Amaral-Rogers V, Belzunces LP, Bonmatin J-M, Chagnon M, Dow ns C, Furlan L, Gibbons DW, Giorio C, Girolami V, Goulson D, Kreutzweis er DP, Krupke C, Liess M, Long E, McField M, Mineau P, Mitchell EAD, M orrissey CA, Noome DA, Pisa L, Settele J, Stark JD, Tapparo A, Van Dyck H, Van Praagh J, Van der Sluijs JP, Whitehorn PR, Wiemers M (2014): Sys temic insecticides (neonicotinoids and fipronil): trends, uses, mode of action and metabolites*. Environ Sci Pollut Res*

Skerl MIS, Bolta SV, Cesnik HB, Gregorc A (2009). Residues of Pesticides in Honeybee (Apis mellifera carnica) Bee Bread and in Pollen Loads from Tre ated Apple Orchards*. Bull Environ Contam Toxico* l83 (3):374-377. doi:10 . 1 007/s00128-009-9762-0.

Spurgeon D., Hesketh H., Lahive E., Svendsen C., Baas J., Robinson A., Horton A. M.(2016). HeardChronic Oral Lethal and Sub-lethal Toxicities of Different Binary Mixtures of Pesticides and Contaminants in Bees

(*Apis mellifera*, *Osmia bicornis* and *Bombus terrestris*). EFSA Supporting Publication EN-1076

Straathoff H (1986). Investigations on the phytotoxic relevance of volatilization of herbicides. *Mededelingen* 51(2A):433–43

Suchail S, Guez D, Belzunces LP (2001). Discrepancy between acute and chro nic toxicity induced by imidacloprid and its metabolites in Apis mellifera. *Environmental Toxicology and Chemistry* 20(11):2482-2486.

Tasei JN, Lerin J, Ripault G.(2000). Sublethal effects of imidacloprid on bumblebees, *Bombus terrestris* (Hymenoptera: Apidae), during a laboratory feeding test. Pest Manag Sci.; 56:784–788.

Tasei JN, Ripault G, Rivault E.(2001). Hazards of imidacloprid seed coating to *Bombus terrestris* (Hymenoptera: Apidea) when applied to sunflower. *J Econ Entomol* . 94:623–627

Thompson H.M., (2012). Interaction Between Pesticides and Other Factors in Effects

vanEngelsdorp D, Caron D, Hayes J, Underwood R, Henson M, et al. (2012). A natio nal survey of managed honey bee 2010–11 winter colony losses in the USA: results from the Bee Informed Partnership. *Journal of Apicultural Research* 51: 115–124.

vanEngelsdorp D, Speybroeck N, Evans JD, Nguyen BK, Mullin C, et al. (2010). Wei ghing risk factors associated with bee colony collapse disorder by classificati on and regression tree analysis. *Journal of Economic Entomology* 103: 1517–1523.

vanEngelsdorp, D., and Meixner, M. (2010). A historical review of managed honey b ee populations in Europe and the United States and the factors that may affe c t them. *Journal of Invertebrate Pathology,* 103, Supplement(0), S80-S95. Do i: 10.1016/j.jip.2009.06.011

Vidau C, Diogon M, Aufauvre J, Fontbonne R, Vigues B, et al. (2011). Exposure to s ublethal doses of fipronil and thiacloprid highly increases mortality of honey bees previously infected by *Nosema ceranae*. *PLoS ONE* 6: e21550

Washington State Dept of Agriculture, (2006). Pollinator protection requirements for Section 18 Emergency Exemptions and Section 24(c) special local need registration in Washington State; *Registration Services Program Pesticide Management Division.*

WHO (2010). International code of conduct on the distribution and use of pesticides:guidelines for the registration of pesticides.