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Incidence and treatment of European foulbrood and sacbrood disease of honey bees (*Apis mellifera* linn.) in kangra valley: A review

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Abstract
Honey bees and honey comb bees are very valuable for wild flowering plants and economically important crop due to their role as pollinators. However, these insects confront many diseases threats (viruses, parasites, bacteria and fungi and large pesticide concentration in the environment. European foulbrood and Sac brood diseases is the most significant and wild spread diseases effect the wellbeing and survival of western honey bees (*Apis mellifera*) and estern honey bees (*Apis Cerana*). As social insects honey bees are particularly valuable to rapid transmission of this ectoparasite within and across colonies managed honey bees play a vital role in pollinating crops that rely on animal pollination, contributing to 35% of global food production. Their importance extends beyond agriculture, as they also pollinate diverse wild flowers, maintaining biodiversity. The sac brood disease has killed 95% of *Apis cerana* colonies in India, while sac brood, disease has affected only 15% of *Apis mellifera* colonies. The symptoms are sometimes confused with those of the bacterial disease European foulbrood in *Apis cerana.* European foulbrood is a bacterial diseases caused by *Melissococcus plutonius.* It affects honey bee (*Apis mellifera).*European foulbrood and sac brood disease is the most significant and wild spread diseases effect the wellbeing and survival of European honey bees. Honey bees are very valuable for wild flowering plants and economically important crops due to their role as pollinators. However, these insects confront many disease threats (viruses, parasites, bacteria and fungi and large pesticide concentrations in the environment. My aim is provide information about EFB and sac brood and its seasonal incidence. I hope it will work and will help in protecting the honey bees from EFB and sac brood.

Keywords: Honey bees, pollinators, European foulbrood, sac brood disease, *Apis mellifera*, *apis* cerana, seasonal incidence

Introduction
Honey bees are among the most fascinating and beneficial creatures in the animal kingdom. As holometabolous insects, they exhibit eusociality, demonstrating a high level of social organization, communication, and defense mechanisms. Beekeeping, or apiculture, has been practiced in India since ancient times, with references to honey bees in religious texts such as the Vedas, Ramayana, and Quran. Despite its economic importance, many regions still rely on traditional methods of beekeeping. Apiculture is vital for agriculture, contributing to the pollination of about 80 cash crops and producing valuable products like honey, pollen, royal jelly, beeswax, and propolis (Johannesmeier and Mostert, 2001) [13]. The Chambal region of Madhya Pradesh, with its rich flora from crops like mustard, coriander, sesame, and neem, is ideal for beekeeping, drawing farmers and beekeepers from neighboring states. India hosts four predominant honey bee species: Rock bee (*Apis dorsata*), little bee (*Apis florea*), Indian bee (*Apis cerana indica*), and European bee (*Apis mellifera*). While the former two remain wild, the latter two are domesticated and reared in wooden hives. However, honey bees face numerous challenges, including diseases, pests, and environmental factors, leading to colony losses and reduced honey production (Genersch, 2010) [10]. European foulbrood, caused by *Melissococcus plutonius*, primarily affects young larvae, causing them to decay and die in twisted positions (Bailey, 1961) [26]. Sac brood, a viral disease, leads to larval death and is most common in the spring when colonies grow rapidly (Bailey, 1969; Chandel *et al.*, 1999) [5, 26].

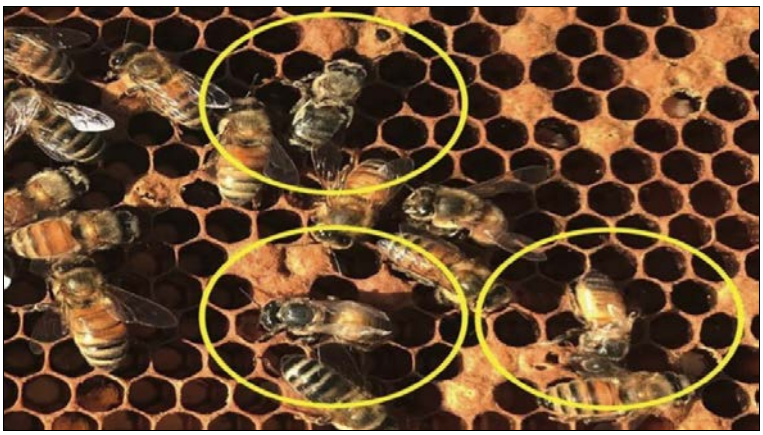
The first records of European foulbrood and sac brood in India were from Maharashtra (Diwan *et al.*, 1971) [8] and Thailand (1981), respectively. Sac brood was later observed in India with a 2.52-2.92% brood mortality in *A. mellifera* colonies (Chandel *et al.*, 1999) [5]. Beekeepers face significant challenges from diseases, pests, and predators, impairing colony health and productivity. The study focuses on the seasonal incidence of such threats to *Apis mellifera* in the Kangra Valley, Himachal Pradesh, during the active bee season (mid-March to mid-May). It aims to identify these challenges, evaluate their impact, and recommend measures to improve colony health and ensure sustainable apiculture practices.

Pathogen transmission in bee colonies occurs through two main routes

1. Vertical transmission-Infected queens pass pathogens to their offspring.
2. Horizontal transmission-Infected worker bees transmit pathogen to other bees transmit pathogens to other bees through physical contact or food exchange known as trophallaxis (Chen *et.al*, 2006) [23].

There is increasing evidence that honey bee pathogen can infect other insect species and plants (Pattemare *et al.*, 2014) thus passing a destabilizing threat to entire local and regional ecosystem. The global bee colony health crisis is escalating, prompting scientist to recommend various control preventive measures including-synthetic and natural chemicals, such as essential oils, Insecticide, Reduced bee density, Improved hive hygienic.

The global bee colony health crisis is escalating, prompting scientists to recommend various control preventive measure including-synthetic and natural chemicals, such as essential oils, In



Diseases of honey bees

European foulbrood: In *Apis mellifera*, European foulbrood (EFB) disease has long been reported. It was first identified in the United Kingdom (Cheshire and Cheyne, 1865) [6] and the United States of America (White, 1907) [24]. The disease has since been reported worldwide wherever *A. mellifera* exists, including Canada (Katznelson *et al.*, 1952) [14], Switzerland, France, England, USA (Morgenthaler, 1995) [16], Argentina (Camegli, 1962) [7], Nepal, and Thailand (Thapa *et al.*, 2000). In India, the disease was first reported in *A. cerana* during 1970 (Diwan *et al.*, 1971) [8] and in *A. mellifera* in 1996 (Anonynous, 1998; Virakarans, 1998) [2]. It reappeared in *A. cerana* after about three decades in Himachal Pradesh during 2002 (Rana *et al.*, 2004) [27] European foulbrood disease is caused by *Melissococcus plutonius*. This bacterium, the etiological agent of EFB in honey bees, is widespread and economically significant in regions such as North and South America, Europe, Japan, Australia, India, China, and South Africa (Jamaludin *et al.*, 2002) [12]. *M. plutonius* is a gram-positive bacterium residing in the larval gut, where it competes with larvae for food. When food is abundant, both larvae and bacteria survive. However, under food scarcity, the bacteria consume available nutrients, causing larval starvation and death (Akor and Sell, 2006). Bailey (1960) [25] reported that outbreaks of EFB are linked to stressful conditions such as food or water scarcity. Factors like genetics, weather, and geography may also play roles. Most larvae die within a brief period, typically around midsummer (Bailey, 1961) [26]. Outbreaks are often followed by sporadic recovery a few weeks later (Bailey, 1960) [25]. Severely infected colonies moved from endemic areas to disease-free zones can recover spontaneously and appear healthy (Bailey and Tascher, 1968). European foulbrood-infected larvae initially appear yellowish compared to the plump, glistening healthy larvae and are slightly displaced within the comb cells. Dead larvae emit a vinegar-like odor (White, 1920; Bailey, 1960, 1983) [25, 28] and exhibit a watery to granular consistency, eventually forming scales upon drying. These scales appear twisted, soft in texture, and are easily removable from cells (Camegli, 1962; Morse, 1980) [7, 29]. Field diagnosis of EFB relies on visual inspection of brood combs and identifying diseased larvae. Symptoms typically appear in unsealed brood 3-6 days old. The infected larvae are displaced in cells, flaccid, and brown, showing various stages of decomposition (Bailey and Ball, 1951). The bacteria generally kill larvae during the coiled stage (4-5 days old), before prepupal and pupal stages. In *A. cerana*, symptoms resemble those of Thai sacbrood disease and mild infestations. In *A. mellifera*, the disease affected 4.3-7.4% of colonies in Colorado (Moffett, 1952) [15], 7.0% in Italy (Galavarini, 1956), and 26.0% in Hungary (Buza and Kovacs, 1969) [30]. In *A. cerana*, EFB affected 25-30% of colonies in Maharashtra during 1970 (Diwan *et al.*, 1971) [8] and 50% in Himachal Pradesh during 2002 (Rana *et al.*, 2003). Fagds *et al.*, (2001) reported that fungal diseases during winter months weakened honey bee colonies, contributing to the development of EFB. They noted that alternating temperature fluctuations in spring hinder optimal hive microclimates, leaving peripheral brood vulnerable to infections. These conditions predispose larvae to *M. plutonius* infections. Somerville (2001) suggested that EFB outbreaks often stem from seasonal changes and stress-related factors, such as nutritional deficiencies and hive disturbances,

particularly during early spring. Rassenova and Parvanov (2005) observed that EFB typically occurs in spring and early summer, with its spread facilitated by bees removing sick or dead larvae. Abrol and Ball (2006) [1] conducted a survey in apiaries across Jammu in 2003-2004 to monitor diseases. Their findings revealed that 10-15% of colonies suffered from EFB. Symptoms included sudden colony weakening, irregular brood patterns, and larvae appearing twisted with greyish-white gut contents visible through the body wall. The disease peaked during the dearth period. Infected larvae, less than 18 hours old, often died while in the coiled stage. Dead larvae were initially soft and watery, later becoming pasty and rubbery upon drying. Infected larvae in the initial coiled stage (3-4 days old) were often found lying along the cell walls. Their color transitioned from bright white to yellowish-brown and eventually dark brown or black. Infected brood cells exhibited perforated, sunken cappings. In some cases, prepupal tongues protruded outwards. Adult bees showed abnormalities, lethargy, and often died shortly after emergence.

Fig 1.1: European foulbrood disease

Symptoms

This disease can be recognized by several distinctive symptoms. Infected larvae display unusual color changes as the condition advances starting off as pearly white and eventually turning yellow, brown, or even dark brown (Govan VA, Allsopp M, and Davison S.) Affected larvae become sunken and eventually disintegrate within the cells, leading to their decomposition. Twisted or contorted larvae are also observed, which serve as distinctive symptoms of European Foulbrood (EFB) (Govan VA, Allsopp M, Davison S. and Djordjevic SP, Noone PA, Smith LA, Hornitzky MA, Bailey L. Infected honey bee colonies often exhibit a distinctive and unpleasant odor. (Genersch E.) The presence of European Foulbrood is further corroborated by distinct disruption in brood development, characterized by: (Forsgren E. European foulbrood in honey bees)

- Irregular brood pattern
- Spotty or patchy brood distribution
- Disrupted brood cell formation

These residual signs serve as diagnostic indicator of European foulbrood infection, providing valuable insights for beekeeper and researchers. (Genersch E.) European foulbrood has debilitating effects on honeybee colonies, culminating in: (Djordjevic SP, Noone PA, Smith LA, Hornitzky MA, Bailey L.

Colony debilitation: Reduced population size and diminished colony strength.

Economic impact: Decreased honey production and reduced pollination services.

European foulbrood infection can escalate into catastrophic events, triggering: (Govan VA, Allsopp M, Davison S.) European foulbrood can be differentiated from American foulbrood through a simple yet distinctive diagnostic test:

Lack of *ropiess*: Unlike European foulbrood infected brood does not exhibit rope -like structure or strings when subjected to a diagnostic stretch test.

This test involves gently stretching the brood with a toothpick or matchstick to observe the presence of *ropiess.* (Forsgren E. European foulbrood in honeybees)

Control

European foulbrood diseases:

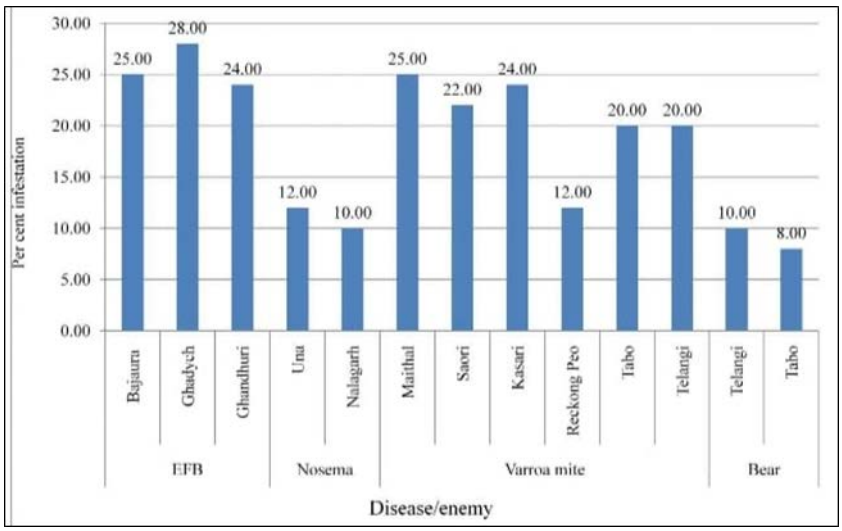
Controlling European foulbrood disease in honey bee colonies is crucial for maintain colony health and productivity. Various method havebeen developed to manage to impact of this disease. High levels of hygienic behaviour can detect and remove the larvae, reducing the disease (Spivak M, Reuter G.S). Providing honeybees with a well-balanced and nutrient rich diet place a critical role in bolstring their immune system and mitigating the risk of European foulbrood infection (Alaux C, Ducloz F, Crauser D, Le Conte Y.) Effective hive ventilation is essential in creating an environment (Spivak M, Reuter G.S) Regular monitoring of colonies for European foulbrood infection enables beekeeper to detect European foulbrood early, Implement containment measures (Van Engelsdorp D, Otis GW, Spivak M.) continued research,monitoring, collaboration among managing European foulbrood (Genersch E, van Engelsdorp D, Evans J D, Saegerman C, Mullin C, Haubruge E, Nguyen BK. *et al.*)

Sacbrood

Sac brood disease of honeybees occurs worldwide and was first identified in the United States of America by White (1917) [21]. The disease is prevalent in America, Australia, Europe, Egypt, and India (Griffin, 1953; Fyg, 1962; Bailey *et al.*, 1964; Ter and Furgala, 1965a, b; Chandel *et al.*, 1999) [5, 31, 32]. In India, the last recorded incidence of sac brood disease was in 1996 in the Kangra region of Himachal Pradesh (Chandel *et al.*, 1999) [5]. The persistence of sac brood virus (SBV) from year to year is facilitated by adult bees in which

the virus multiplies without causing disease. In infected hives, the virus remains viable in diseased brood for only a few weeks, serving as a source of infection (White, 1917) [21]. Under natural conditions, the virus typically disappears rapidly during summer and does not spread, even when combs containing diseased larvae are placed in colonies. This is because diseased brood is promptly detected and removed by adult bees (Bailey *et al.*, 1964; Hitchcock, 1966; Bailey, 1967) [3, 32]. Larvae affected by sac brood virus fail to pupate and accumulate ecdysial fluid beneath their stretched skin, forming a sac-like structure. Infected larvae change color from pearly white to pale yellow and, shortly after death, dry out to form dark brown scales. This disease is most common in spring when colonies are growing rapidly, and large numbers of larvae and young adults are present (Grabensteiner *et al.*, 2001) [11]. Sac brood virus primarily affects the brood of *Apis mellifera*, leading to perforation of sealed brood, pre-pupal death due to incomplete pupation, and fluid accumulation around the body. The color of the brood changes from pearly white to pale yellow, and dead brood dries out into dark brown scales. Sac brood infection primarily appears in spring when brood rearing begins (Rana *et al.*, 2011) [17]. Sac brood disease is caused by the sac brood virus, which infects worker bee larvae. Larvae are believed to contract the virus by consuming contaminated water, pollen, or nectar. Infected larvae die shortly after capping, becoming fluid-filled sacs. Diseased brood is often scattered among healthy brood, with discolored, sunken, or perforated cappings. The virus can remain viable in dead larvae, honey, or pollen for a few weeks (Anonynka, 2013) Studies on the severity of sac brood disease have shown that up to 80% of worker brood was infected with SBV in Britain (Bailey, 1967) [3], 45% in Australia (Renyi *et al.*, 2006), 50% in the USA (Sainswiki *et al.*, 1992), and 30% in the United Kingdom. In India, for the first time, mortality rates of 2.52% to 2.92% in *A. mellifera* brood were recorded in 1998 (Chandel *et al.*, 1995) [5]. The disease is generally observed in spring and early summer worldwide (Chandel *et al.*, 1999; Homitzky and Anderson, 2003) [5], but it was also recorded in November in Himachal Pradesh (Rana, 2003). Sac brood virus kills honeybee brood at the pre-pupal stage (approximately 10 days old), two days after the sealing of the brood. In 2003, sac brood virus was detected at two locations in Himachal Pradesh. At Nauni (Solan district), it was observed during spring and summer (March to May), affecting 0.39% to 5.20% of the brood. At Jachh (Kangra district), the disease was detected from March to June, infecting 1.23% to 2.10% of the brood (Rana and Rana, 2006) [33].

Fig 1.2: Sacbrood disease



Symptoms

The larvae with sac brood failed to pupate. Infected larvae change in color from pearly white to peal yellow, shortly after death and dry out, forming a dark brown scale when the colony is growing most rapidly and large numbers of susceptible larval and young adult are available. (Grabensteiner et. al., 2001) [11]. Sac brood virus affect the brood of Apis mellifera. The color of brood changes from pearly white to pale yellow, dead brood dry out to dark brown scale (Rana *et al.*, 2001). Sac brood disease caused by sac brood virus which affect the worker bee larvae. Infected larval die shortly after capping and become fluid filled sac (Anonymous, 2013). Disease indicated that 80% of worker brood was infected with sac brood virus in Britain (Bailey, 1967) [3], and 49% in Australia (Berenyi *et al.*, 2006) [22]. Upto 90% in USA (Shimanuki *et al.*, 1992) [18] and 30% in United Kingdom. Sac brood virus killed honey bee brood at pre pupal stage (10 days of age). On the second day after the sealing of brood.

Seasonal incidence

The incidence of the European foulbrood disease in *Apis* bee colonies was highest in July (17.50%) when temperature, humidity, and rainfall were high. Sac brood disease was most common in May (6.90%) when temperature was high, but humidity and rainfall were low.

In *Apis Mellifera* colonies, European foulbrood disease was most prevalent in September (37.10%) when temperature was high, and humidity and rainfall were moderate, under stationary conditions. However, under migratory condtions, Sac brood disease incidence was also highest in May under both conditions (6.60% and 5.80%) when temperature was, and humidity and rainfall were moderate.

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