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The rise and demise of control options for fruit fly in Australia

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ABSTRACT

For several decades, control of fruit fly pests in Australia has depended to a large degree on dimethoate, a broad-spectrum organophosphate insecticide that has been widely used on many different crops as well as on ornamental plants. However, many dimethoate use patterns were withdrawn in 2011 following a review by regulatory authorities. This manuscript places dimethoate in context as the latest in a long line of pre-harvest pesticides such as the arsenics, tartar emetic, sodium fluosilicate, nicotine sulphate, the organochlorines, and organophosphates, that have been lost from the fruit fly control toolkit over the past century. The succession of postharvest treatments such as fungants, dips and sprays is also examined. Dimethoate and fenthion have offered relatively easy and cost effective pest control solutions since the 1960s, but in the absence of equivalent alternatives it is now necessary to develop "systems approaches" based on multiple control strategies and risk assessment. Such approaches represent a fundamental shift in pest management strategy, and will require improved understanding of fruit fly biology to prevent infestation in the field, combined with postharvest surveillance and non-chemical treatments. Some options are briefly discussed.

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1. Introduction

Tephritid fruit flies are important pests of fruit production in most regions of the globe. In eastern Australia, Queensland fruit fly (Qfly) *Bactrocera tryoni* (Froggatt) (Diptera: Tephritidae) is the most important fruit fly pest (Dominiak and Daniels, 2012: Plant Health Australia, 2008) and is currently known to infest more than 100 native and introduced hosts (Hancock et al., 2000; Oliver, 2007). The total average export value for Australia's top 25 commodities that are fruit fly hosts has been estimated at \$432 million. Additionally, more than \$1 billion worth of products traded domestically are vulnerable to this pest. As a result, Qfly poses a major threat to national and international market access for horticultural commodities produced in eastern Australia (Plant Health Australia, 2008).

However, records indicate that Qfly was not always an important pest. Although Qfly susceptible fruit was grown in Sydney following European settlement in 1788, no larvae were reported in fruit before 1819 (Drew, 1989). It was not until 1852 that fruit fly became known as a pest to New South Wales (NSW) fruit growers, while the first major outbreak of fruit fly in commercial fruit occurred in 1884 (Froggatt, 1897).

It is likely that Qfly was originally restricted to native subtropical and tropical forests and mangroves of eastern and northern Australia. Its natural range may not have extended south farther than Gladstone, in mid Queensland. However, once Qfly moved from native hosts into orchard fruit, unrestricted transport of infested produce from Queensland inadvertently distributed it nationally (Gurney, 1925; Lea, 1899; May, 1963; Dominiak and Daniels, 2012). Early settlers and fruit producers were unprepared for fruit fly and only a few basic control measures were available (May, 1963). Over time, there has been a long succession of preferred and permitted pesticides for fruit fly control. This paper reviews the main pesticides used in Australia for fruit fly control over the last century and places the current review of dimethoate and fenthion in context as the latest of many to fall by the wayside as single kill-step solutions.

2. Repellents

While repellents are not pesticides, they can serve a similarly protective role and were particularly important in the early days of fruit fly control in the absence of pesticides. Products such as tallow, coal-tar, wood-tar, creosote, carbolic acid and vinegar, were considered to be repellent to insects in general and possibly effective



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against Qfly (Benson, 1895; French, 1898, 1907). Other products tested included carbolacene (a liquid disinfectant soap) (Jarvis, 1925), sodium fluosilicate (Allman, 1942) and nicotine sulphate (Veitch, 1934). Bordeaux mixture (copper + lime) sprayed before harvest was also claimed to reduce infestation (Gurney, 1925). Veitch (1934) reported promising results using sprayable oil. Recent results support this observation, with mineral oils shown to reduce oviposition by Qfly on tomato fruit (Liu et al., 2002; Nguyen et al., 2007). Spray oils appear to be one of the more promising areas for future research, and research is continuing in this area. Gibberellic acid use has resulted in large reduction in stinging in *Anastrepha ludens* (Loew) (Birke et al., 2011).

3. Bait sprays

Insecticides are generally contact poisons that transfer to the insect when it contacts a treated substrate, or require ingestion to exert their toxic effects. Either way, to be effective, insects need to come into contact with the insecticide. One way to economically increase the chances of insects coming into contact with insecticides is the use of persistent attractive baits. Great reliance has been placed on bait sprays, which also have the benefit of restricting insecticide distribution to a few points in the environment, away from produce. These bait sprays generally consist of a toxicant plus food source (protein and/or sugar) designed to attract flies; young flies require protein to mature (Perez-Staples et al., 2007) while sugar provides energy for longevity or movement (Perez-Staples et al., 2008; Prabhu et al., 2009). They may be applied to a bait station or sprayed on foliage or applied as spot sprays.

3.1. Arsenic

The first commonly used fruit fly killing agent was copper arsenate, also known as "Paris green". Lead arsenate proved less phytotoxic, and became the most widely used of all the arsenical compounds (Peryea, 1998). As toxicity depends on consumption by the target pest, foliage was sprayed with a combination of lead arsenate and sugar or molasses (Compere, 1907; Quinn, 1907; Tryon, 1912; Jarvis, 1923, 1926; Gurney, 1925). Other arsenic compounds evaluated for Qfly control included potassium arsenate (Compere, 1907; Jarvis, 1923), calcium arsenate, sodium arsenite and arsenious oxide (McCarthy, 1925). Calcium arsenate represented a cheaper option following World War One, when lead became expensive (Peryea, 1998).

Lead arsenate and calcium arsenate killed flies within 4 h, while sodium arsenite killed within 30 min. As sodium and calcium arsenite caused fruit damage these pesticides were applied weekly to boards (10 inches by 24 inches), with one board/tree. Despite application of increasing rates of chemical to these boards, McCarthy (1927) reported only a 70%–80% control with this mixture.

Although lead arsenate use was not recommended in NSW by the 1940s (Anon., 1942, 1943), foliage sprays continued to be recommended in Queensland (May, 1944). Use only declined after the introduction of dichloro-diphenyl-trichloroethane (DDT) in the 1950s (Lagerwerff, 1972). Although arsenic tends to leach out of soil, lead is retained for longer periods. As a consequence, orchard soils may have 25–35 times the normal concentration of lead and/ or arsenic in the top 30 cm (Merry et al., 1983). All arsenic compounds are highly toxic to humans. Potassium and sodium arsenite have been found to be carcinogenic.

3.2. Tartar emetic and sodium fluosilicate

Two new chemicals became available in the 1930s: tartar emetic and sodium fluosilicate. Sodium fluosilicate was cheaper than arsenic compounds, killed insects quickly by contact as well as by ingestion and was less toxic to humans (Marcovitch, 1924). In 1937, sodium fluosilicate became an alternative bait spray (Allman, 1939a; Anon., 1940) and subsequently became the recommended foliage spray (Anon., 1938, 1941a,b; Sproul, 2001).

Tartar emetic (antimony potassium tartrate) was slightly more effective and less phytotoxic than sodium fluosilicate (Allman, 1940, 1941). It contains the metal antimony, which produces symptoms similar to those from arsenic. In 1947, tartar emetic was used to control the first Qfly outbreak in South Australia (SA) (Madge et al., 1997). Tartar emetic combined with sugar soon replaced sodium fluosilicate as the standard foliage spray (May, 1944; Allman and Friend, 1948).

Despite this, neither tartar emetic or sodium fluosilicate were completely effective. Tartar emetic baits provided reasonable control when fly populations were low and no alternative food was available (Hely, 1949), but higher populations necessitated the addition of DDT or parathion (Anon., 1954). Tartar emetic and sodium fluosilicate were replaced by DDT in SA by 1950 (Madge et al., 1997) and were no longer recommended in NSW by the 1960s (Skepper and Sweedman, 1968).

3.3. Nicotine

Nicotine is an alkaloid produced naturally by solanaceous plants as a defence against insect attack. When consumed it acts as a nerve poison, directly affecting clumps of nerve tissue (ganglia) at multiple points in the insects nervous system (Pratt and Babers, 1977). When used as an insecticide, nicotine is usually applied as nicotine sulphate. Wright (1935) claimed that nicotine sulphate mixtures reduced fruit fly infestation. In 1944, nicotine sulphate was used experimentally, and later sugar was added to encourage feeding. Although recommended as an alternative to tartar emetic or sodium fluosilicate, some flies can recover after the initial knockdown (Anon., 1947, 1955). Although approved under some organic systems, nicotine sulphate is extremely toxic and no longer used. The structurally similar chemical imidacloprid is not registered for fruit fly control, but may be toxic to Qfly adults and larvae.

3.4. Organophosphates

Organophosphate compounds are the basis of many different and current pesticides. They inactivate the enzyme acetylcholinesterase, which is essential to normal nerve function. Accumulation of acetylcholine in the nervous system leads to tremors, convulsions, paralysis and finally death (Pratt and Babers, 1977). Organophosphates degrade more quickly in the environment than organochlorines such as DDT. However, they also tend to be more acutely toxic to mammals, including humans.

One of the first organophosphate insecticides developed was parathion. Invented during the 1940s in Germany, parathion was proposed as both a bait ingredient and cover spray for Qfly control by 1948 (Allman, 1948). Parathion is reputed to be one of the most dangerous of all chemicals. It is already banned in most developed countries, with moves to ban it worldwide (Kegley et al., 2010).

Malathion, another organophosphate, has much lower toxicity and so was a useful replacement for parathion in bait sprays, particularly after the development of resistance to DDT (Anon., 1960). Baits containing protein and malathion have been recommended for controlling adult Qfly since 1962 (Anon., 1962; O'Loughlin, 1964). By the mid 1960s, such baits were being applied at a rate of 20 \times 100 ml squirts/acre to control town Qfly populations across many areas of southern and western NSW (Skepper and Sweedman, 1968; Anon., 1968). Malathion and protein baits continue to be used to the current day (Anon., 1996; Madge et al., Download English Version:

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