A novel, effective formulation for bed bug control

Due to its double active ingredient composition (natural pyrethrum and S-methoprene), Biopren® BFS bed bug and flea killer spray not only flushes out and knocks down hiding insects, but its S-methoprene insect growth regulator active ingredient also disrupts the life cycle of insects by preventing them to develop into adults.

The product is able to control even those pests that are resistant to synthetic pyrethroid insecticides.

BIOPREN BFS-A is distributed in the UK by Agropharm Ltd. [HSE 9533]

Use insecticides safely. Always read the label and product information before use. Biopren® is a registered trademark of Babolna Bio.
Bed bugs and Insecticide resistance

Implications for pest managers

Resistance among bed bugs to the pyrethroids, carbamates and organophosphates is well documented. Frontline pest professionals relying solely on insecticides, especially pyrethroids, can expect failures so what can be done to gain control?

The resurgence

Over the last fifteen years, bed bugs have undergone a dramatic global resurgence. In the United Kingdom, calls to councils concerning bed bugs increased during 2000-2006 by an average of 28.5% per annum. In Australia, a survey of professional pest managers in 2006 revealed that infestations increased by 4,500% since the start of the new millennium. Similar dramatic rises have been seen all around the world.

Bed bug infestations have now occurred almost everywhere that humans frequent, such as cinema complexes, clothing stores, public transportation and vehicles, hospital wards and doctors’ waiting rooms, school classrooms, the home, as well as being a major pest in the hotel and lodging industries.

Various factors have been postulated as being responsible, but it is widely acknowledge that insecticide resistance is the key trigger for the pests’ comeback.

Insecticide resistance

‘Resistance’ is defined by the Insecticide Resistance Action Committee (www.irac-online.org) as: ‘a heritable change in the sensitivity of a pest population that is reflected in the repeated failure of a product to achieve the expected level of control when used according to the label recommendation.’

The first case of failure was reported in 1947 from a naval barracks in Pearl Harbour, Hawaii, and resistance to DDT was confirmed. By the 1960s, bed bugs resistant to DDT were found worldwide.

The development of resistance in one group of insecticide can also confer ‘cross resistance’ to other insecticides that share similar mode of actions. Both DDT and the pyrethroids target the sodium channels of nerve cells, thus it was not surprising that resistance was subsequently observed with the pyrethroids.

First modern reports in 2006

In the UK, the first modern reports of insecticide resistance in bed bugs were in 2006 by Clive Boase and colleagues, where they found that a dose of insecticide that produced 99% mortality in a susceptible strain, failed to kill two field strains.

In Australia, resistance profiling was undertaken comparing a modern field strain of C. lectularius from Sydney with an old susceptible strain (from Bayer, Monheim). The lethal dose to kill the Sydney bugs was around 430,000 times greater for deltamethrin and an extraordinary 1.4 million times greater with permethrin.

Resistance to the carbamates and...
organophosphates has also been well documented.

See panel opposite for a run down of resistance mechanisms.

Implications for control
So what does all this insecticide resistance mean to the pest manager?

Simply, that you can expect failures when controlling bed bug infestations if you rely solely on insecticides, particularly the pyrethroids.

However, insecticide formulation does complicate this issue even further. For example, every pyrethroid based aerosol that we have tested will kill bed bugs very quickly when sprayed directly at the insect; why this is, we are unsure. Perhaps the carriers overcome the cuticular resistance, thereby providing better penetration, and the addition of a synergist like piperonyl butoxide (PBO) can overcome P450 resistance. Information such as this may lead to the development of other more effective formulations in the future.

Ineffective as residuals
Yet, when aerosols are applied as a residual, they do almost nothing to control bed bugs and this is true of most pyrethroids; they are simply ineffective as residual products. Thus for example, the use of mattresses and encasements impregnated with older pyrethroids such as permethrin simply makes no sense, and may even contribute to further resistance development.

The pest manager must also be wary of insecticidal manufacturers’ claims with efficacy data in product advertisements. Was the insecticide tested on a resistant or susceptible strain? If the strain was resistant, didn’t P450, or cuticular resistance? These are important questions and manufacturers must begin to report on the resistance status of the bed bugs used in their tests.

Even some of the newer insecticides appear to only offer limited benefits. The neonicotinoids provide excellent control when sprayed directly at bed bugs but offer poor residual control. Again, P450 may be contributing to cross resistance with this group. While with the insect growth regulators (IGRs), there are ethical aspects that must be considered; for an IGR to work, your client must be bitten!

This all means that you need to follow a programme of integrated pest management to be successful at bed bug control. It is important to firstly utilise non-chemical means of control to reduce the overall number of bed bugs in the infestation prior to any insecticide treatment.

This could include the disposal of infested items, physical removal via vacuuming, and the use of extremes in temperatures such as steam and/or dry heat.

For residual application, there really is no better product than the silicates such as Diatomaceous Earth (DE). DE has the advantage of having a physical action rather than physiological; it works by absorbing the waxes in the cuticle making the insect susceptible to dehydration, and while it is slow to work, it will kill the bed bugs in time. The other advantage of having a physical action is that resistance is unlikely to develop.

Most importantly, before selecting and adding a product to your bed bug control programme, always check to see if it is recommended in one of the industry codes of practices.

References


Resistance mechanisms

There are several ways that insect populations can become resistant and they can exhibit more than one mechanism simultaneously. These mechanisms include; behavioural resistance, reduced cuticular penetration, increased metabolic detoxification and target site insensitivity.

1 Behavioural resistance
Behavioural resistance is the ability of insects to avoid an insecticide through behavioural changes. The best known example is bait aversion in cockroaches. Arguably, bed bugs may have developed behavioural resistance. In a recent study, it was noted that bed bugs avoided resting on the deltamethrin treated filter paper, thereby reducing potential insecticidal contact. However, this behaviour could be simple avoidance due to the ‘excito-repellency’ nature of pyrethroids; further research is required to confirm if behavioural resistance is occurring.

2 Reduced penetration (cuticular resistance)
Various proteins have been identified from the cuticle (‘skin’) of bed bugs that reduces the penetration of insecticides into the insect. Additionally, the cuticle itself is extremely waxy; bed bugs are one of the most desiccant resistant insects on earth, and this waxy layer offers further protection by inhibiting insecticides adhering to or penetrating the cuticle.

3 Increased detoxification (metabolic resistance)
Metabolic resistance is basically the breakdown of a toxin (i.e. insecticide) into a less toxic form. Recent studies have indicated that a range of enzymes, notably ‘cytochrome P450 monooxygenases’ (P450) and ‘esterases’, are responsible for resistance to the pyrethroids and other insecticide groups. These enzymes are common in modern bed bug strains.

4 Target site insensitivity
This refers to modifications in the insect that prevents the insecticide from binding to the site where it normally targets, rendering it ineffective. There are various types of target insensitivity (only those relevant to bed bugs are mentioned).

4.1 Nerve insensitivity (including knockdown resistance)
Pyrethroid insecticides affect the insect nervous system by stimulating nerve cells to produce repetitive discharges, which eventually causes death. An important resistant mechanism against pyrethroids is called ‘knockdown resistance’ (abbreviated ‘kdr’), which results from various mutations that prevent the insecticide from acting on the nerve. The two most commonly reported kdr mutations in bed bugs are ‘V419L’ and ‘L925I’, so named after the amino acid changes on the gene. It appears that these mutations are common in bed bugs around the world.

4.2 Altered acetylcholinesterase
Acetylcholinesterase is an enzyme that controls nerve function by preventing nerves from firing. Many insecticides target this enzyme by switching it off; uncontrolled nerve triggering results in insect death. Altered acetylcholinesterase is associated with resistance to the organophosphates and carbamates, and several studies have identified resistance in bed bugs to these groups. Perhaps bed bugs may have evolved a modified acetylcholinesterase to combat these insecticides.

Thus there are multiple forms of insecticide resistance in bed bugs acting in several layers, all to combat your efforts at controlling their populations.

Therefore when insecticides are applied, such as pyrethroids, bed bugs can hide in tiny cracks and crevices that have missed exposure to avoid the insecticides (potential behavioural resistance).

Once the bed bugs come in contact with insecticides, cuticular proteins reduce the rate of insecticide penetration (reduced penetration). If the insecticides enter the organism, bed bugs will enhance metabolic detoxification to decrease the effect of the insecticide (increased detoxification). Finally, if the insecticides reach the nerve cells, kdr mutations will provide another line of defence against the insecticide (target site insensitivity). These are the mechanisms that we presently are aware of and it is likely that other forms will be discovered in the future.