CottonInfo **Information** *when you need it*

2014-15 End of Season Resistance Monitoring Report

by Sharon Downes

Conventional Insecticide Testing

by Lisa Bird

Best Practice

Hatching and species composition from CSIRO Bt resistance monitoring 2014/15

Across all sampled valleys there were 15964 samples submitted to the program during the 2014-15 season. The majority of samples were eggs, although there were quite a few collections of larvae from chick pea, pigeon pea and mung bean. Of the eggs submitted, 58% successfully hatched. Of the eggs that successfully hatched and the larvae, 43% were *H. armigera*.

Data on the total number of samples collected in 2014-15, and average hatching and species composition are presented in Table 1. The % *H. armigera* values do not include hosts that are known to be dominated by this species (i.e., maize and sorghum). The levels of hatching and species composition are averages for each valley and the actual levels vary greatly among properties. The values in brackets to the right of % hatch and % *H. armigera* indicate the range among collections. For instance, for the period between September and December, in the Upper Namoi the % *H. armigera* is 17 and ranges from 8 to 25 among the properties sampled. The periods across the top of the table represent the dates at which the samples were identified to species, which is about 12 days after they were received as eggs or up to 7 days after larvae collections were received.

Table 1: Summary data for the samples collected in 2014-15.

Valley	Trait	**Sep-Dec**	**Jan-Apr**	**Season total**
Lower Namoi	No. of samples	951	768	1719
	% hatch	78 (66-96)	58 (37-82)	70 (37-96)
	% *H. armigera*	(0)	60 (22-100)	60 (0-100)
Upper Namoi	No. of samples	1538	0	1538
	% hatch	46 (46-96)	-	46 (46-96)
	% *H. armigera*	17 (8-25)	-	17 (8-25)
Emerald	No. of samples	1319	1740	3059
	% hatch	36 (4-73)	22 (2-61)	25 (2-73)
	% *H. armigera*	69 (41-97)	96 (83-100)	80 (41-100)
Darling Downs	No. of samples	459	760	1219
	% hatch	64 (41-89)	34 (3-64)	62 (3-89)
	% *H. armigera*	16 (0-35)	62 (30-100)	50 (0-100)
St George	No. of samples	1993	1174	3167
	% hatch	59 (21-94)	78 (44-100)	65 (21-100)
	% *H. armigera*	11 (0-26)	52 (0-100)	37 (0-100)
Gwydir	No. of samples	508	19	527
	% hatch	82 (70-96)	89 (89)	87 (70-96)
	% *H. armigera*	9 (9)	0 (0)	9 (0-9)
Macintyre	No. of samples	151	941	1092
	% hatch	59 (12-85)	65 (24-100)	56 (12-100)
	% *H. armigera*	0 (0)	74 (47-91)	74 (0-91)
MIA	No. of samples	33	421	454
	% hatch	75 (75)	69 (29-100)	68 (29-100)
	% *H. armigera*	0 (0)	25 (13-45)	25 (0-45)
Mungindi	No. of samples	624	1250	1874
	% hatch	58 (21-86)	50 (28-76)	47 (21-86)
	% *H. armigera*	0 (0)	50 (18-75)	50 (0-75)
Macquarie	No. of samples	620	714	1334
	% hatch	68 (51-94)	46 (0-90)	56 (0-90)
	% *H. armigera*	27 (14-50)	22 (0-46)	24 (0-50)

The following reports are based on data collected by the Bt resistance and insecticide resistance monitoring programs and are the final results for the season 2014/15. The take home message for both programs is that there has been no substantial change in frequency in Helicoverpa spp. in this year compared to recent years. It is imperative however to continue to practise good resistance management via adherence to the Bollgard II RMP and IRMS.

End of season results from CSIRO Bt resistance monitoring 2014/15

Changes to the CSIRO Bt Resistance Monitoring Program from 2013/14

From 2002 to 2012 CSIRO conducted two sorts of tests for Bt resistance (Figure 1). F_2 screens can detect heterozygote individuals even when the resistance is recessive (rS). They involve testing the grandchildren of pairs of moths raised from eggs collected from field populations, and therefore take about 10 weeks to run. This method tests both alleles from two field collected individuals (4 alleles total), identifies all previously detected and potentially new types of resistance.

In 2004 CSIRO developed protocols for testing resistance using a modified and shorter version of the F_2 method called an F_1 screen. F_1 screens involve testing the offspring of matings between a resistant moth (rr; isolated from F_2 screens and then reared as a colony in the laboratory) and a moth raised from eggs collected from field populations. They take around 5 weeks to conduct. This method tests both alleles from one field collected individuals (2 alleles total), and can only determine if the field moth has the same resistance as its mating partner.

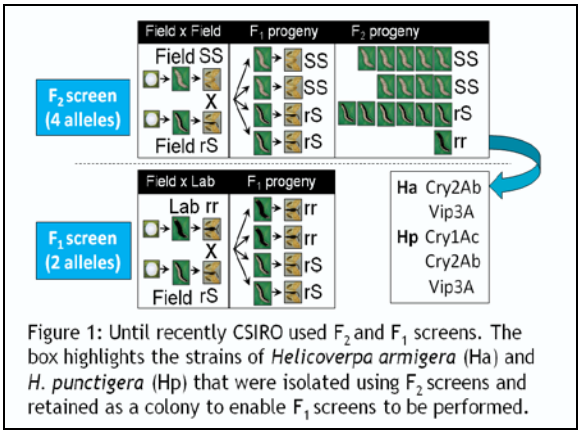

Figure 1: Until recently CSIRO used F_2 and F_1 screens. The box highlights the strains of *Helicoverpa armigera* (Ha) and *H. punctigera* (Hp) that were isolated using F_2 screens and retained as a colony to enable F_1 screens to be performed.

From 2002 to 2012 CSIRO performed experiments that showed that each of the isolates of Cry2Ab resistance detected using F_2 screens was the same type of resistance that was initially identified. Similarly, work on Vip3A from 2009 to 2012 showed that all newly isolated resistances were the same type that was initially identified. In other words, although frequencies of resistance to some Bt toxins is higher than expected, it seems that there is only one key type of resistance for each Bt toxin.

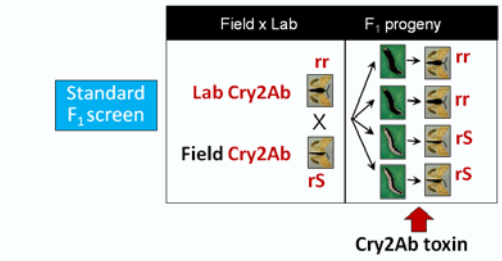

Figure 2a: This diagram shows the standard F_1 screen using Cry2Ab as an example. A moth from a known rr lab colony (isolated using F_2 screens) is crossed with a field moth and the offspring are screened against the same toxin that the rr lab colony resists (in this case Cry2Ab).

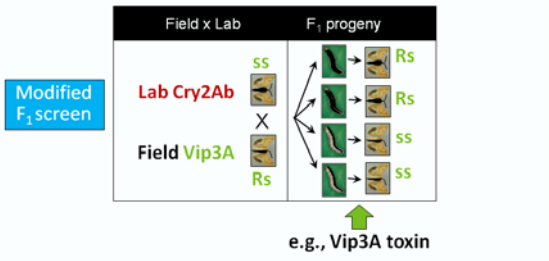

Figure 2b: This diagram shows the modified F_1 screen. A moth from a known rr lab colony (in this case resistant to Cry2Ab and susceptible to Vip3A) is crossed with a field moth and the offspring are screened against a different toxin (in this case Vip3A) to the one that the rr lab colony resists.

In 2013 CSIRO shifted to performing only F_1 screens to focus on the frequencies of the known common resistances. As well as screening F_1 families against the toxin of interest (e.g., Cry2Ab: Figure 2a), we introduced screens against all relevant Bt toxins (e.g., Cry1Ac and Vip3A: Figure 2b) to detect any new forms of resistance that are dominant. Every 4 or 5 years CSIRO will incorporate F_2 screens into the program to check for any new resistances that are recessive.

A graphical summary of F_2 screen data until 2012/13

The following graphs provide the F_2 screen data for the CSIRO program until 2012/13 and are useful background for interpreting the F_1 screen data, particularly in cases where the latter data exists for only a few seasons.

F_2 screens for Cry1Ac and Cry2Ab commenced in 2002/03. For *H. armigera* and *H. punctigera* the cumulative frequency of alleles conferring resistance to Cry1Ac is 3/5936 (0.001) and 5/6866 (0.001) respectively. For *H. armigera* and *H. punctigera* the cumulative frequency of alleles conferring resistance to Cry2Ab is 60/6336 (0.010) and 53/7834 (0.007) respectively. F_2 screens for Vip3A, the additional toxin in Bollgard 3, commenced in 2009/10. For *H. armigera* and *H. punctigera* the cumulative frequency of alleles conferring resistance to Vip3A is 54/2094 (0.026) and 18/1742 (0.010) respectively.

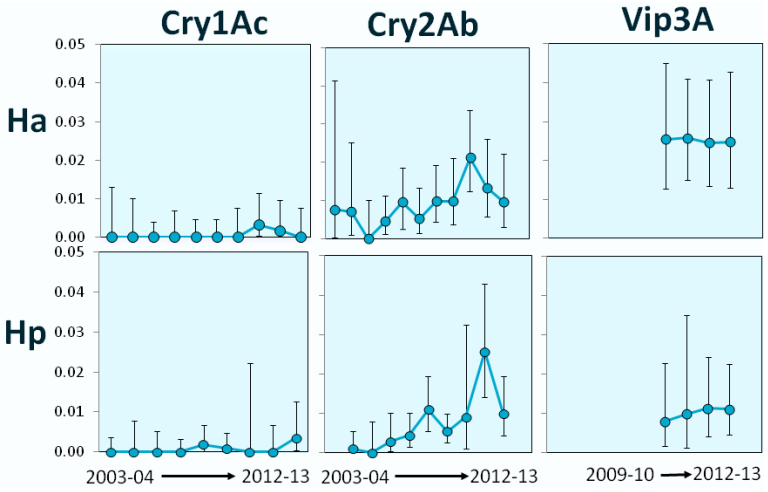

Figure 3: Proportion of resistant alleles from CSIRO F_2 screens until 2012/13. Data are presented separately for *H. armigera* (Ha) and *H. punctigera* (Hp).

Standard F_1 screens

Cry1Ac

Several families of *H. armigera* that carry a gene conferring resistance to Cry1Ac have been isolated using F_2 screens (Figure 3) but none have been retained as colonies. Therefore in *H. armigera* it is not possible to perform F_1 screens against Cry1Ac.

In 2014/15 we screened 394 alleles from *H. punctigera* and isolated 1 case (0.002) conferring resistance to Cry1Ac (see Table 2); this frequency is lower than for 2013/14. For *H. punctigera* the cumulative frequency of alleles conferring resistance to Cry1Ac since 2013/14 is 3/892 (0.003).

Table 2: Summary of results from F_1 screens of *H. punctigera* against Cry1Ac. Data are presented as the frequency for that testing season. For each entry we have indicated the number of homozygous (rr) resistant individuals that contributed to the frequency.

Species	Year	Cry1Ac F_1 screen			
		alleles tested	scored positive	Freq. of r	No. rr
H. punctigera	2013/14	498	2	0.004	0
	2014/15*	**394**	**1**	**0.003**	**0**
	Total*	892	3	0.003	0

Cry2Ab

In 2014/2015 we screened 720 alleles from *H. armigera* and isolated 15 cases (0.021) conferring resistance to Cry2Ab (see Table 3); this frequency is higher than for 2013/14. Of these alleles, two were contributed from one individual that was homozygous (rr) for resistance. For *H. armigera* the cumulative frequency of alleles conferring resistance to Cry2Ab since the CSIRO program began (2007/08) is 271/9908, and analyses on this F_1 screen data show that there has not been a significant change in frequency over time.

In 2014/2015 we screened 488 alleles from *H. punctigera* and isolated 5 cases (0.011) conferring resistance to Cry2Ab (see Table 3); this frequency is almost the same as for 2013/14. Of these alleles, four were contributed from two individuals that was homozygous (rr) for resistance. For *H. punctigera* the cumulative frequency of alleles conferring resistance to Cry2Ab since the CSIRO program began (2007/08) is 98/4654, and analyses on this F_1 screen data show that there has not been a significant change in frequency over time.

Table 3: Summary of results from F_1 screens of *H. armigera* and *H. punctigera* against Cry2Ab. Data are presented as the frequency for that testing season. For each entry we have indicated the number of homozygous (rr) resistant individuals that contributed to the frequency.

Species	Year	Cry2Ab F_1 screen			
		alleles tested	scored positive	Freq. of r	No. rr
H. punctigera	2007/08	194	2	0.010	0
	2008/09	640	30	0.047	1
	2009/10	1138	15	0.013	1
	2010/11	358	10	0.028	0
	2011/12	736	24	0.033	3
	2012/13	518	7	0.014	2
	2013/14	582	5	0.010	0
	2014/15*	**488**	**5**	**0.011**	**2**
	Total	4654	98	0.020	9
H. armigera	2007/08	278	9	0.032	0
	2008/09	3104	69	0.022	1
	2009/10	1710	37	0.022	0
	2010/11	1810	80	0.044	3
	2011/12	832	33	0.040	1
	2012/13	770	18	0.023	2
	2013/14	684	10	0.015	1
	2014/15*	**720**	**15**	**0.021**	**1**
	Total	9908	271	0.027	9

Vip3A

To develop a robust resistance management plan for Bollgard 3 it is important to know the baseline frequency of Vip3A resistance genes in populations of *H. armigera* and *H. punctigera*.

In 2014/2015 we screened 626 alleles from *H. armigera* and isolated 10 cases (0.016) conferring resistance to Vip3A (see Table 4); this frequency is higher than for 2013/14. Of these alleles, two were contributed from one individual that was homozygous (rr) for resistance. For *H. armigera* the cumulative frequency of alleles conferring resistance to Vip3A since 2013/14 is 16/1268.

Since 2009/10 we began F_1 screens against Vip3A in *H. punctigera.* In 2011/12, the frequency was 0.095 (7/74); since it was obtained from a relatively small sample it is excluded from the overall summary results and in analyses. In 2014/2015 we screened 414 alleles from *H. punctigera* and isolated 1 case (0.002) conferring resistance to Vip3A (see Table 4); this frequency is lower than 2013/14. For *H. punctigera* the cumulative frequency of alleles conferring resistance to Vip3A since the CSIRO program began (2007/08) is 31/2544, and analyses on this F_1 screen data show that there has not been a significant change in frequency over time.

Table 4: Summary of results from F_1 screens of *H. armigera* and *H. punctigera* against Vip3A. Data are presented as the final frequency for that testing season. For each entry we have indicated the number of homozygous (rr) resistant individuals that contributed to the frequency. *Note the very small sample for 2011/12 has been excluded from the total estimates.

Species	Year	Vip3A F_1 screen			
		alleles tested	scored positive	Freq. of r	No. rr
H. punctigera	2009/10	1144	16	0.014	0
	2010/11	172	3	0.017	0
	2011/12	74	7	0.095	0
	2012/13	284	5	0.018	2
	2013/14	530	6	0.011	2
	2014/15*	**414**	**1**	**0.002**	**0**
	Total*	2544	31	0.012	4
H. armigera	2013/14	642	6	0.009	1
	2014/15*	**626**	**10**	**0.016**	**1**
	Total*	1268	16	0.013	2

Modified F_1 screens

In 2013/14 in *H. punctigera* we screened alleles to identify new dominant resistances for Cry1Ac (n=196), Cry2Ab (n=230) and Vip3A (n=204) and found none. In *H. armigera* we screened alleles to identify new dominant resistances for Cry1Ac (n=186), Cry2Ab (n=156) and Vip3A (n=284) and found none.

In 2014/15 in *H. punctigera* we screened alleles to identify new dominant resistances for Cry1Ac (n=140), Cry2Ab (n=176) and Vip3A (n=126) and found none. In *H. armigera* we screened alleles to identify new dominant resistances for Cry1Ac (n=246), Cry2Ab (n=312) and Vip3A (n=240) and found none.

Take home messages

- In *H. punctigera* and *H. armigera* the first isolations of alleles conferring resistance to Cry1Ac were recently detected but remain relatively rare.
- Cry2Ab resistance genes and Vip3A resistance genes were present at detectable levels before Bt cotton expressing these traits was widespread.
- F_1 data demonstrate that currently in *H. armigera* 4% of individuals in the population are heterozygous (rS) for the Cry2Ab resistance gene and there is no indication of changes in resistance frequency since testing started in 2007/08.
- F_1 data demonstrate that currently in *H. punctigera* 2% of individuals in the population are heterozygous (rS) for the Cry2Ab resistance gene and there is no indication of changes in resistance frequency since testing started in 2007/08.
- Currently in *H. armigera* 3% of individuals in the population are heterozygous (rS) for the Vip3A resistance gene (based on F_1 data).
- Currently in *H. punctigera* 1% of individuals in the population are heterozygous (rS) for the Vip3A resistance gene (based on combined F_2 and F_1 data).
- No new dominant resistances to Cry1Ac, Cry2Ab or Vip3A have been detected.

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Mid-season preliminary results from NSW DPI insecticide resistance monitoring 2014/15

Changes to the NSW DPI Insecticide Resistance Monitoring Program from 2012/13

1. Introduction of F_2 screens. Prior to 2014, NSW DPI conducted topical resistance testing for all synthetic insecticides using F_0 insects (ie. test insects were collected as eggs and a discriminating concentration of insecticide applied directly to insect larvae in the late 3rd /early 4th instar). This is a well established method for detection of resistance to synthetic broad-spectrum insecticides active by contact, and has been the industry standard for monitoring resistance in Australian populations of *Helicoverpa spp.* since monitoring began in the early 1980s.

Although testing of F_0 insects is highly effective for detecting dominant forms of resistance (as is the case for broad-spectrum insecticides such as SPs, OPs and carbamates), it is less sensitive for detecting non-conspicuous (recessive) resistance alleles. This is because heterozygous carriers of resistance (rS) could be killed by the discriminating concentration of insecticide, thereby underestimating the frequency of early-stage resistance in insect populations.

Therefore, in 2013 NSW DPI introduced an F_2 screening technique as a means of increasing capacity for detecting non-dominant resistance alleles which, as described above, involves testing the grandchildren of pairs of moths raised from eggs collected from field populations (Figure 1 above). This is particularly important for detecting alleles that may enhance survival to selective insecticides indoxacarb, emamectin benzoate and rynaxypyr (chlorantraniliprole).

2. Introduction of feeding bioassays. The use of traditional topical bioassays in insecticide resistance monitoring was originally developed for broad-spectrum insecticides whereby toxicity is mediated primarily through contact mode of entry. However, indoxacarb, emamectin benzoate, and rynaxypyr intoxicate insects via both contact and ingestion, with the later considered the primary route whereby insects accumulate a lethal dose of insecticide (Wing et al. 2004, Lasota and Dybas 1991, Temple et al. 2009).

Results from NSW DPI research supports these previous findings and suggests that delivery by ingestion using a feeding method of bioassay (where insecticide is incorporated into insect diet) is highly effective for assessing toxicity of these chemistries and would be an appropriate alternative method for use in resistance monitoring programs. Therefore in 2013-14, the use of diet incorporation bioassay was implemented as a new industry standard for monitoring resistance to indoxacarb, emamectin benzoate, and rynaxypyr.

In summary, the adoption of F_2 screens where a discriminating dose is delivered by ingestion by a diet incorporation method of bioassay will improve the efficiency for detecting low frequency (early-stage) resistance to key selective insecticides. This method is also highly compatible with pre-emptive mitigation of resistance to prevent in-field failures of insecticidal products.

Insecticide resistance results – H. armigera

Synthetic pyrethroid. Synthetic pyrethroid resistance is well established in Australian populations of *H. armigera* at variable but generally high frequencies. Monitoring has historically involved the use of fenvalerate. While no longer registered for use in *Helicoverpa spp.* control, fenvalerate provides a reliable indicator of general (α-cyano) pyrethroid resistance.

In 2011 the frequency of resistance increased by 30% for both general pyrethroid and the resistance-breaking product, bifenthrin (Table 5). The average frequency of fenvalerate resistance across all regions sampled was around 90% for the three seasons from 2011-12 to 2013-14, with regional

frequencies ranging between 84% and 100%. In 2011 resistance to the pyrethroid bifenthrin (Talstar®) also increased by 30% with the average regional frequencies ranging between 39% and 43% from 2011-12 to 2013-14.

The consistency in frequency data over a three year period confirms this increase, and reflects a return to high level pyrethroid resistance which is widespread in Australian populations *H. armigera*. While the use of pyrethroid products in the cotton system does not account for the magnitude of this increase, pyrethroid use in other cropping systems that play host to *Helicoverpa spp.* has been significant. For example α-cyano products have been used prophylactically for *Helicoverpa spp.* control in chickpea, and for midge control in sorghum.

In 2014-15 resistance to fenvalerate was high at 93% (*n* = 1053) and moderate to bifenthrin at 55% (*n* = 1047).

Organophosphates. Resistance to organophosphates (chlorpyrifos) remains present in *H. armigera* populations since being detected again in 2001/02. However, resistance frequencies have declined to very low levels (<3%) which have been maintained over the last eight seasons. Results from 2011-12 to 2013-14 indicate a continuing trend of low frequency organophosphate resistance (Table 5) with regional frequencies ranging from 0 to 3%.

In 2014-15 resistance to chlorpyrifos remains low at 2% (*n* = 979).

Carbamates. Resistance to carbamates (methomyl) has been present at moderate frequencies for over ten years, with typical frequencies of 20 - 30%. Results from the last three seasons show that resistance to carbamates remains at moderate and stable levels (Table 5). Regional frequencies range from 7% to 46%.

In 2014-15 average resistance frequency to methomyl has increased to 49% (*n* = 951).

Table 5. Resistance frequencies in *H. armigera* determined from F_0 bioassays conducted by topical bioassay (annual average all regions).

	% Resistance (*n*)				
Insecticide	**2010-11**	**2011-12**	**2012-13**	**2013-14**	**2014-15**
Indoxacarb (Steward®)	0.4 (917)	0.3 (1646)	0.6 (2754)		
Emamectin benzoate (Affirm®)	0.2 (1277)	0.4 (1497)	1 (2354)		
Rynaxypyr (Altacor®)	0.2 (1460)	0.1 (1519)	0.2 (2530)		
Chlorpyrifos - OP	0.6 (670)	1 (1051)	1 (2037)	2 (601)	2 (979)
Methomyl - carbamate	17 (602)	34 (1049)	23 (1965)	28 (598)	49 (951)
Bifenthrin (Talstar®) – pyrethroid	8 (404)	40 (1197)	39 (2215)	43 (636)	55 (1047)
Fenvalerate - pyrethroid	61 (246)	91 (902)	91 (2446)	90 (581)	93 (1053)

Indoxacarb, emamectin benzoate & rynaxypyr 2011-12 & 2012-13. Since the introduction of Bollgard II, resistance has stabilised for the three key selective products indoxacarb, emamectin benzoate and rynaxypyr. During the seasons 2011-12 and 2012-13 resistance frequencies for these products were monitored by using traditional screening techniques based on topical application of a discriminating concentration of insecticide in the F_0. Based on these methods, resistance to these

chemistries was shown to be consistently low across all regions in the seasons with very few survivors detected (Table 5).

In 2012-13 the indoxacarb window was brought forward to provide an early season 'soft option' for *Helicoverpa spp.* control. Monitoring results indicate that this has not led to detectable changes in resistance frequency. Nevertheless, continued compliance with resistance management guidelines is essential for minimising resistance risk associated with the use of indoxacarb particularly because extended use of this product potentially targets successive generations of *H. armigera*.

We cannot necessarily assume that resistance alleles selected by exposure to products used in non-cotton systems (chickpea for example) will be eliminated by exposure to Bollgard II in the subsequent generation. This is because the large majority of *H. armigera* will not preferentially establish on cotton but instead will favour alternative hosts present in a mixed cropping landscape such as maize and sorghum.

Indoxacarb, emamectin benzoate & rynaxypyr 2013-15. New standards for measuring resistance frequency in selective insecticides based on F_2 screens and the use of a feeding (insecticide-incorporated diet) bioassay technique (described above) were implemented in the 2013-14 season and replaced topical F_0 screening methods previously used to monitor resistance to these products.

Combined data from all regions in 2013-14 shows that none of the iso-female lines examined for resistance to emamectin benzoate or rynaxypyr scored positive against the discriminating concentration of these insecticides (Table 6). In 2014-15, there were again no positive tests recorded for emamectin benzoate (n = 1302) and only a single positive test recorded for rynaxypyr (n = 1310), resulting in a resistance frequency of 0.008 for this product (Table 6).

In 2013-14 a total of 1096 alleles from *H. armigera* were screened against indoxacarb. Combined data from all regions shows that 9 iso-females lines tested positive for resistance to this insecticide (9/1096) (Table 6). Based on these data and assuming non-dominant resistance, the estimated R frequency for alleles conferring resistance to indoxacarb was 0.008 (0.8%). Positive tests from indoxacarb screens included individuals from the lower Namoi valley (3; n = 390 alleles), upper Namoi valley (2; n = 252 alleles), Gwydir (2; n = 46 alleles), Burdekin (1; n = 92 alleles), and MIA (1; n = 56 alleles).

In 2014-15 a total of 1324 alleles were screened for indoxacarb resistance. Combined data from all regions shows that 25 iso-female lines tested positive for resistance indicating a resistance frequency for indoxacarb of 0.019 (1.9%) (Table 6). Positive tests from indoxacarb screens were again distributed throughout a range of geographic locations including the lower Namoi valley (2; n = 154), upper Namoi valley (10; n = 610), Gwydir (2; n = 164), Macquarie (2; n = 32), Mungindi (1; n = 76), St. George (3; n = 74) and Emerald (5; n = 100).

In 2014-15 we did not detect any cases of resistance to emamectin benzoate (n = 1302 alleles).

Of the iso-female families tested for rynaxypyr resistance (n = 1310 alleles) a single positive test was recorded, resulting in a frequency of resistance to this product of 0.0008 (0.08%).

Of the iso-female families tested for indoxacarb resistance (n = 1324 alleles), 25 scored positive for resistance indicating a resistance frequency for this product of 0.019 (1.9%).

These results indicate that there is currently very low resistance to selective insecticides in Australian population of *H. armigera*.

Table 6. Indoxacarb, emamectin benzoate and rynaxypyr resistance frequencies in *H. armigera* determined from F_2 bioassays conducted on insecticide-incorporated diet (annual average all regions).

Insecticide	Year	Total tests	Total positives	Total alleles	%R
Emamectin Benzoate					
	2013-14	500	0	1000	0
	2014-15	651	0	1302	0
Rynaxypyr					
	2013-14	525	0	1050	0
	2014-15	655	1	1310	0.08
Indoxacarb					
	2013-14	548	9	1096	0.8
	2014-15	662	25	1324	1.9

Insecticide resistance results – H. punctigera

Products tested against *H. punctigera* include pyrethroid and abamectin. This species is tested in the F_0 generation using a topical bioassay. While *H. armigera* has repeatedly demonstrated the ability to develop resistance to conventional insecticidal chemistries, *H. punctigera* has maintained a susceptible status with only a single report of this species developing field resistance to an insecticide (fenvalerate) in field populations (Gunning et al. 1997).

From 2011 to 2014 there was ample opportunity to test resistance in *H. punctigera* because of the strong dominance of this species in all regions, particularly in 2013-14 where approximately 4000 individuals were tested against both chemistries (Table 7). Survivorship in all years was extremely low indicating full susceptibility of this species to fenvalerate and abamectin.

In 2014-15 resistance continues to be at low levels in *H. punctigera.*

Table 7. Fenvalerate and abamectin resistance frequencies in *H. punctigera* determined from F_0 bioassays conducted by topical bioassay (annual average all regions).

Insecticide	% Resistance (*n*)			
	2011-12	2012-13	2013-14	2014-15
Fenvalerate	0.07 (1477)	0.2 (2472)	0.2 (3976)	1.1 (1346)
Abamectin	0 (1757)	0.1 (2402)	0.02 (4051)	0.2 (1219)

Take home messages and implications for management

- Resistance to pyrethroids has returned to high levels in *H. armigera* while *H. punctigera* remains susceptible. The implications of this for management are that applications of pyrethroid on *H. armigera* dominant populations will provide little or no control unless applied in combination with a synergist such as piperonyl butoxide (PBO).

- Carbamate resistance remains widespread in the *H. armigera* population. A further increase in resistance to methomyl in 2014-15 indicates that field performance of this product against *H. armigera* may be highly variable.

- Low resistance frequencies to organophosphates, indoxacarb, emamectin benzoate and rynaxypyr in *H. armigera*, indicate these products will continue to be effective in the control of *H. armigera*. **Nevertheless, the cotton IRMS should be followed to ensure that selection pressure applied by any one of these chemical groups is minimized across multiple generations of Helicoverpa.** The IRMS recommends:

  1. Avoiding repeated applications of products from the same group.
  2. The use of chemical rotations even when targeting different pests.
  3. Compliance with maximum number of recommended sprays for any one chemical group.

- The introduction of an F_2 screen for monitoring resistance to selective insecticides has significantly increased capacity for detecting resistance alleles that are rare in the *H. armigera* population, and which would otherwise remain undetected using traditional screening methods.

  The introduction of these methods has not only improved the accuracy of resistance detection, but has also resulted in the first cases of genetic resistance to indoxacarb isolated from field populations of *H. armigera* in Australia.

  NSW DPI is working toward full characterization of indoxacarb resistance in *H. armigera*. So far we have conducted preliminary quantitative genetic analysis of indoxacarb-selected strains to determine the degree of dominance of the putative indoxacarb resistance allele. Results indicate that the allele(s) involved autosomal (not sex-linked) and is inherited as a partially dominant trait. Further characterization will increase our understanding of the role of population genetics in indoxacarb resistance evolution, and help to pre-emptively mitigate resistance risk associated with the incidence of this allele.

For further information regarding conventional insecticide resistance monitoring please contact lisa.bird@dpi.nsw.gov.au

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