THE CORESTA COLLABORATIVE STUDY ON BACTERIAL WILT (*RALSTONIA SOLANACEARUM*) – 2002 REPORT

Anne Jack

Agronomy Department, University of Kentucky, Lexington, Kentucky 40546-0312, USA.

ABSTRACT

This, the final report of the bacterial wilt subgroup in its original format, summarizes the activities of the subgroup and the achievement of its objectives.

The bacterial wilt subgroup was initiated in 1995. Nine participants from six countries have regularly taken part.

The cultivar set included cultivars with all known sources of resistance; the polygenic resistance ex T.I.448A, the monogenic *Rps* resistance and the monogenic *Rxa* resistance.

Most of the objectives of this study have been achieved. It has been demonstrated that the T.I.448A resistance does hold up wherever tested, and that there is no evidence for the existence of different bacterial types, based on host reaction. Excellent sources of flue-cured resistance have been identified, although we have been less successful with burley. It is now clear that bacterial wilt resistance is additive, not recessive, as has been reported. With multi-site data, it is evident that the conflicting reports on the nature of the genetic control of bacterial wilt resistance are due to the different levels of disease pressure at the various sites.

Having achieved the objectives, there is no further information to be gained from the trial in its original format. The format has now been changed; these trials will be used as an international testing forum for new burley breeding material.

INTRODUCTION

The CORESTA bacterial wilt collaborative subgroup was set up in 1994 at the Harare CORESTA Congress, and the first trials were grown in the Southern hemisphere in the 1995/96 season. The mandate was to collect information on bacterial wilt (*Ralstonia solanacearum* (Smith) Yabuuchi *et al*.), addressing four specific objectives. Resistant cultivars were of particular interest because there is no other effective means of controlling this disease.

There is no known immunity to bacterial wilt; the resistance is not absolute and the resistant control will usually show some symptoms. The resistance in commercial cultivars, derived from T.I.448A, is polygenic. The monogenic resistance genes, *Rps* and *Rxa*, confer low to moderate resistance only.

This, the eighth report to be presented (Jack and Robertson, 1995, 1996, 1997, 1998; Jack 1999, 2000, 2001), covers the achievement of the objectives.

PARTICIPANTS AND CULTIVAR SET

Participants

Initially, 18 participants in 12 countries registered for this subgroup. However, some of these have never participated, some have withdrawn and some do not return data; we generally receive data from about nine participants in six countries.

Table 1: Participants in the CORESTA Bacterial Wilt Collaborative Study in the Southern and Northern Hemispheres

Country	Organization	Comments
Southern Hemisphere		
Brazil	DIMON do Brasil Tabacos Ltda (Dimon)	regular
	ProfiGen do Brasil Ltda (Profigen)	regular
South Africa	Lowveld Tobacco Growers Association (LTGA)	regular
	Institute for Industrial Crops (IIC)	regular – greenhouse trial
Zimbabwe	Tobacco Research Board (TRB)	regular
Northern hemisphere		
Bulgaria	Tobacco and Tobacco Products Institute (TTPI)	regular – no disease
China	Qingzhou Tobacco Research Institute (QTRI)	regular
Iran	Iranian Tobacco Company (ITC)	two years only – no disease
	Tirtash Tobacco Institute (Tirtash)	three years only – no disease
	Rasht Tobacco Research (Rasht)	three years only – no disease
Malaysia	R J Reynolds Tobacco Co (RJR)	one year only
Mexico	CIICA	one year only
USA	N.C. State University (NCSU)	regular
	Pee Dee Centre, Clemson University (PD)	regular

Cultivar set

Each participant grew nine (ten after 1999) prescribed cultivars (Tables 2 and 3) and up to three (two after 1999) optional local cultivars. When compiling the cultivar set, the objectives were to include all known sources of resistance, to include material from as wide a range of countries as possible, and to include material from all three major types of tobacco, flue-cured, burley and Oriental. The final cultivar set (Table 3) included all three known sources of

resistance, had material from five countries and had one burley, one Oriental, one air-cured (semi-Oriental) and six flue-cured entries.

The cultivar set was changed at the beginning of 1998, when new cultivars became available. NC 60, RK 3 and T 20 (Table 2) were replaced by Oxford 207, KB 101 and Xanthi (Table 3). Enshu AC was added in 1999 (Table 3).

Table 2: Prescribed Cultivar Set up to 1998

Cultivar	Origin	Type	Resistance
Hicks (S control)	USA	Flue-cured	Susceptible
NC 95 (R control)	USA	Flue-cured	Polygenes ex T.I. 448A
F_1 (NC 95 x Hicks)	–	Flue-cured	F_1 (R x S)
NC 60 #	USA	Flue-cured	Polygenes ex T.I. 448A
ADT 108/B40	South Africa	Flue-cured	Polygenes ex T.I. 448A
T 20 #	Zimbabwe	Flue-cured	Polygenes ex T.I. 448A
RK 3 #	Zimbabwe	Flue-cured	Polygenes ex T.I. 448A
Kokubu	Japan	Air-cured	*Rps* gene
Enshu FC *	Australia	Flue-cured*	*Rps* + polygenes?

Replaced in 1998

* This accession, obtained from Australia, is a flue-cured cultivar, but the original Japanese accession is air-cured. Source of resistance is unknown.

Table 3: Prescribed Cultivar Set from 1998

Cultivar	Origin	Type	Resistance
Hicks (S control)	USA	Flue-cured	Susceptible
NC 95 (R control)	USA	Flue-cured	Polygenes ex T.I. 448A
F_1 (NC 95 x Hicks)	–	Flue-cured	F_1 (R x S)
KB 101 #	Korea	Burley	Polygenes ex T.I. 448A
ADT 108/B40	South Africa	Flue-cured	Polygenes ex T.I. 448A
Oxford 207 #	USA	Flue-cured	Polygenes ex T.I. 448A
Xanthi #	Turkey	Oriental	*Rxa* gene
Kokubu	Japan	Air-cured	*Rps* gene
Enshu FC *	Australia	Flue-cured*	*Rps* + polygenes ?
Enshu AC ##	Japan	Air-cured	*Rps* + polygenes

New entries in 1998

* This accession, obtained from Australia, is a flue-cured cultivar, but the original Japanese accession (##) is air-cured. Source of resistance is unknown.

New entry in 1999

The original cultivar set (Table 2), included material from five countries, and two of the three known sources of resistance; the third was not available at that time. Most of the cultivars listed in Table 2 carry polygenic resistance derived from T.I. 448A (Smith and Clayton, 1948). Kokubu, which carries the monogenic partially dominant *Rps* resistance, has been reported to be moderately resistant only (Matsuda and Ohashi, 1973). Enshu FC is a flue-cured accession obtained from Australia. It was originally entered in the belief that it was the Japanese accession of Enshu, and was assumed to carry the *Rps* resistance gene as well as polygenes. However, it bears no resemblance to the original Japanese air-cured accession, and as its

pedigree is unknown, we have no way of knowing if it does indeed carry the *Rps* gene. It has been designated “FC” to distinguish it from the original air-cured Japanese Enshu.

Four new cultivars were added in 1998 and 1999 (Table 3). KB 101 is a Korean burley cultivar with resistance derived from Burley 64, and is one of the few burley cultivars reported to have resistance to bacterial wilt (Kim, 1993). Oxford 207 is a recently released USA flue-cured cultivar, reported to have the highest resistance of all flue-cured cultivars (Sisson, 1999). Both of these cultivars carry the T.I. 448A polygenic resistance. Xanthi, an Oriental cultivar from Turkey, carries the monogenic partially dominant *Rxa* resistance (Matsuda, 1977), reported to be moderately resistant only (Clayton and Smith, 1942). The Japanese air-cured Enshu AC, reported to be highly resistant, carries the *Rps* resistance gene as well as the polygenes found in the other cultivars (Matsuda and Ohashi, 1973). It has been designated “AC” to distinguish it from the Australian flue-cured Enshu FC.

DESIGN, ASSESSMENT AND DATA ANALYSIS

Details of experimental design, assessment methods and data analysis are given in the 1999 and 2001 reports (Jack, 1999, 2001).

ACHIEVEMENT OF OBJECTIVES

The objectives of the trial were:

1. To investigate the apparent anomaly that the most widely used source of resistance, derived from T.I.448A, does not appear to hold up in some countries, notably South Africa.
2. To establish whether different bacterial types exist, based on host reaction.
3. To identify sources of resistance which will be useful to breeders.
4. To establish whether the T.I.448A-derived resistance is recessive, as reported.

These objectives have mostly been achieved.

1. Reported anomaly of susceptible NC 95

This objective has been achieved; the apparent anomaly does not exist. Results from this study show that the resistance in NC 95 (derived from T.I.448A) does hold up in South Africa. Initial screening of this cultivar in South Africa showed that a local accession of NC 95 was susceptible (Englebrecht and van Heerden, 1992). However, data from this study showed that NC 95 was considerably more resistant than Hicks (Figure 1), and that it was no less resistant

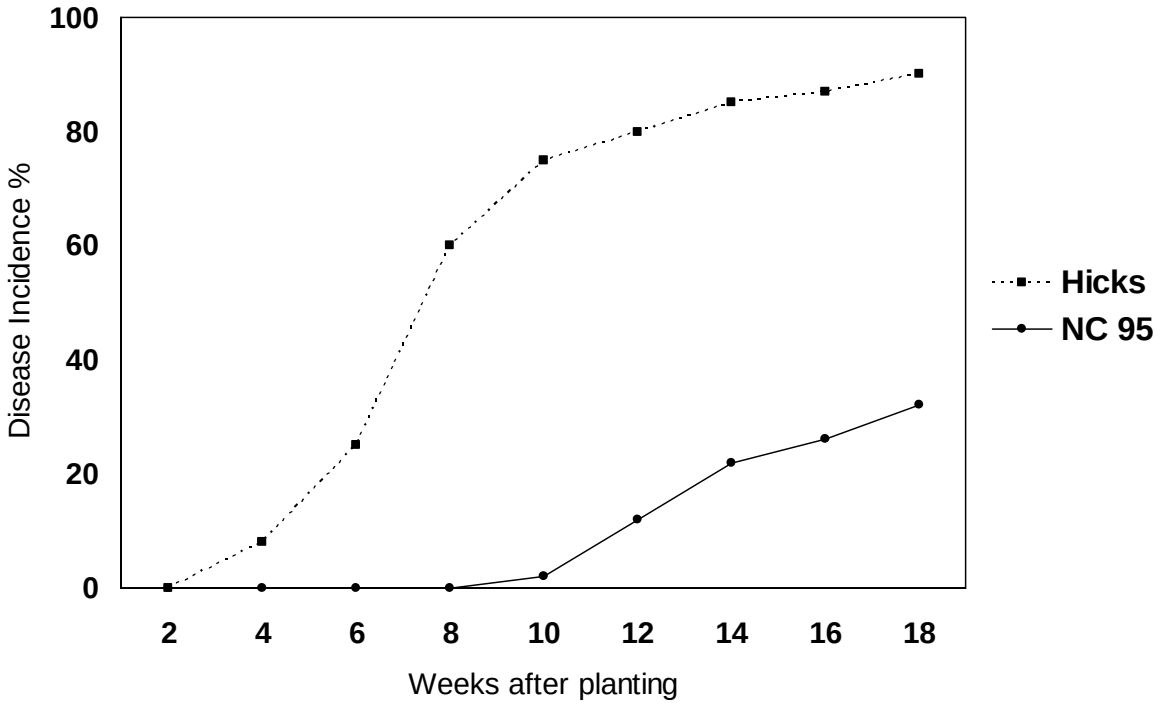

Figure 1: Disease Progress Curves – Hicks and NC 95 in South Africa in 1998/99.
Disease incidence (% infected plants)

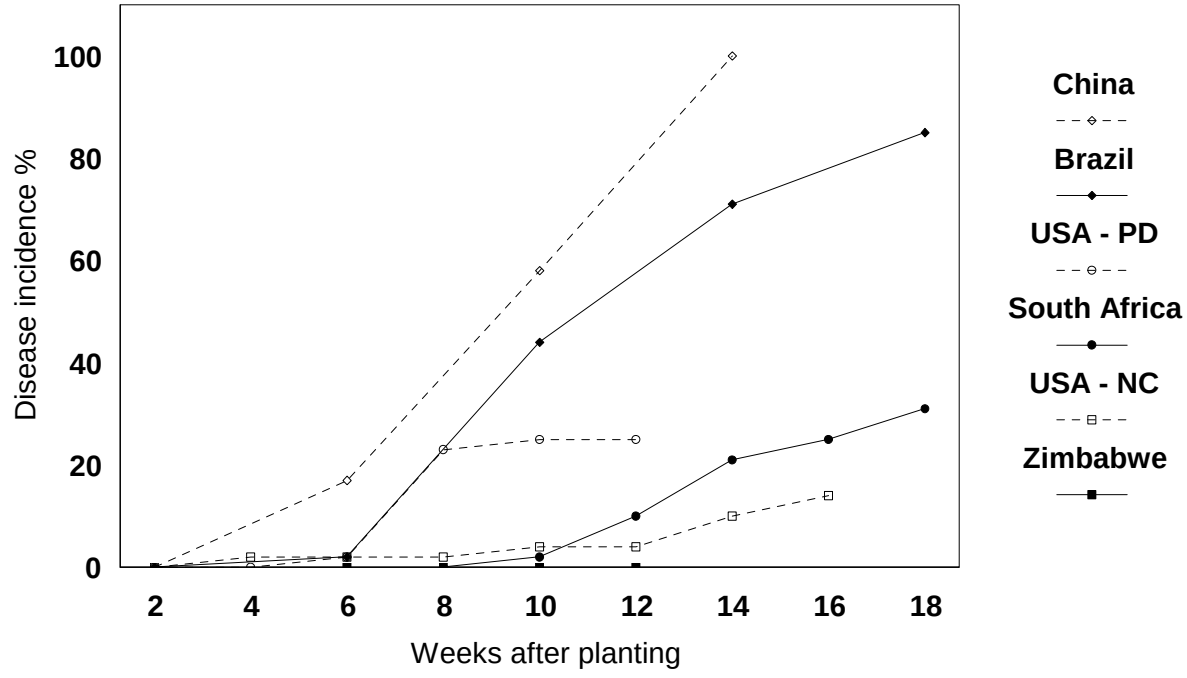

Figure 2: NC 95 – control at all sites in 1998/99 and 1999.
Disease incidence (% infected plants)

in South Africa than in other countries (Figure 2). As the T.I.448A resistance clearly does hold up in South Africa, it seems likely that the apparently susceptible NC 95 was the wrong accession.

2. Different bacterial types

There is no evidence to suggest that there are different bacterial types based on host reaction. There are known strains of this pathogen, and at least 12 different avirulence genes (Salanoubat *et al*, 2002). However, these strains are pathogenic or not pathogenic to tobacco; they do not vary in their relative effect on cultivars. Biochemical tests have distinguished several biovars, but again, all biovars seem to have the same relative effect on the various cultivars. The most resistant and the most susceptible cultivars have generally been constant across sites and years. However, there is no doubt that isolates vary considerably in virulence, even once the confounding effect of environment has been removed (Robertson, 1998).

Where locally bred optional entries were included, these were never more resistant than the resistant control (Jack, 1999). Although these local entries are no doubt better suited to local conditions in terms of agronomic characteristics, there does not appear to be any advantage in selecting against the local bacterial wilt *per se*.

Where there were inconsistencies in the relative ranking of cultivars, they could usually be explained by variation in level of disease pressure, or by the presence of other diseases.

In most cases, Xanthi performed no better than the susceptible control. However, in Zimbabwe it performed significantly better than the susceptible control (Figure 3). This was because the moderate-low resistance of the *Rxa* gene in Xanthi is sufficient under the very low disease pressure in Zimbabwe; under higher pressure, this resistance is swamped and is indistinguishable from the susceptible control.

ADT 108/B40 was as good as or better than the resistant control at most sites. However, in South Africa it had significantly higher scores than did the resistant control (Figure 4). This was because ADT 108/B40 is very susceptible to black shank, and the test site was infested with black shank. Plants scored as infected with bacterial wilt were later found to be infected with black shank.

At the NCSU site in the USA, there were several anomalies that could not be explained. Xanthi *(Rxa* gene) performed much better than expected, under high disease pressure (Figure 3). Both ADT 108/B40 (polygenes) and Enshu AC (*Rps* gene and polygenes) did not perform as well as expected – both of these cultivars are usually as good as or better than the resistant control (Figure 4). This is inconclusive, but it is possible that there is something unique about this site.

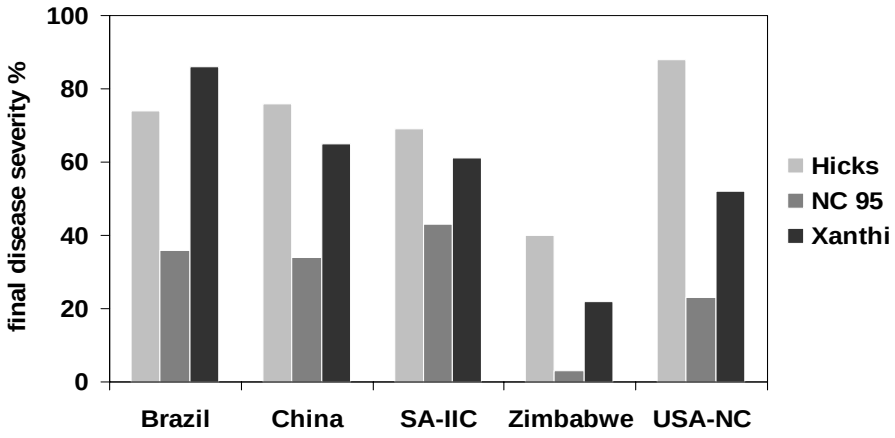

Figure 3: Xanthi vs. susceptible and resistant controls.
Disease severity (% infection)

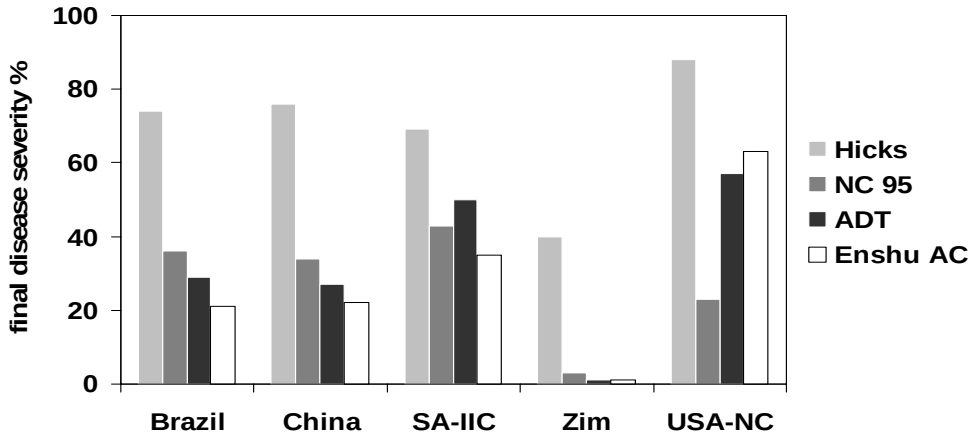

Figure 4: ADT and Enshu AC vs. susceptible and resistant controls.
Disease severity (% infection)

3. Identification of useful sources of resistance

This objective has been achieved in the case of flue-cured tobacco, but has been less successful with burley. There are several reasons for this. There are few burley cultivars with good resistance in the public domain. The American breeders have generally been the leaders in flue-cured bacterial wilt resistance breeding, but bacterial wilt is not a problem on American burley. Most importantly, it seems to be more difficult to incorporate resistance into burley, and this may be related to its yellow color.

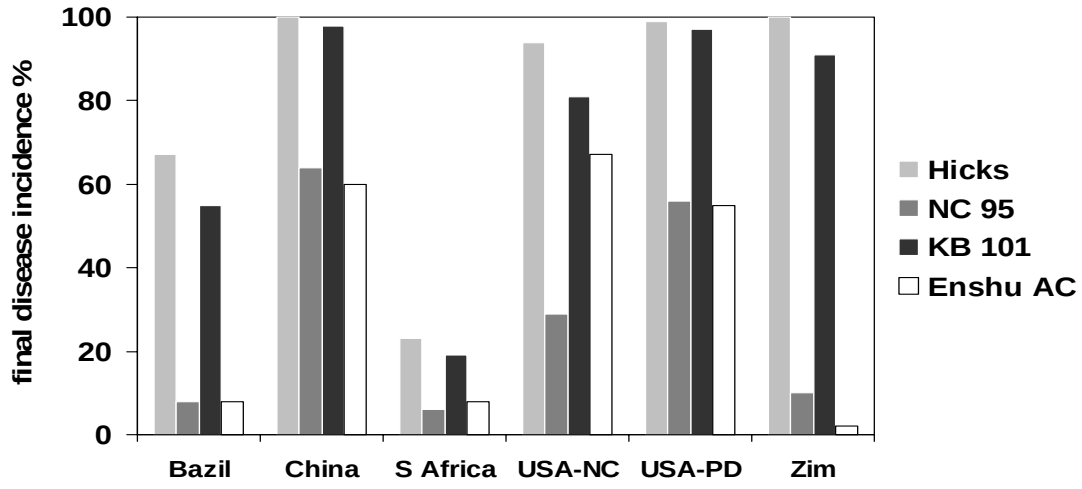

Figure 5: KB 101 and Enshu AC vs. susceptible and resistant controls. Disease incidence (% infected plants)

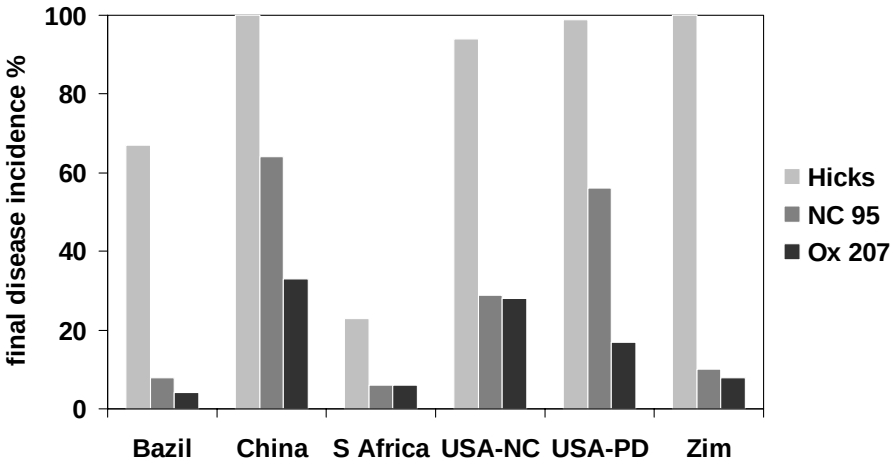

Figure 6: Oxford 207 vs. susceptible and resistant controls. Disease incidence (% infected plants)

The only burley cultivar so far tested in this trial, the Korean KB 101, has proved to have a level of resistance too low to be of interest to breeders; it was usually little better than the susceptible control (Figure 5). However, the Japanese air-cured cultivar Enshu AC (carrying the *Rps* gene + polygenes), looks very promising (Figure 5). Being an air-cured type, it would be a very useful source of resistance for burley breeders. The South Africans report that this cultivar also has good black shank resistance.

Of the flue-cured cultivars, Oxford 207 was consistently the best over years and sites (Figure 6). Enshu FC and the South African breeding line ADT 108/B40 also had good resistance at most sites; all three of these cultivars would be useful parents for breeders. Enshu FC is a flue-cured cultivar which bears no resemblance to the original Japanese air-cured Enshu

(designated Enshu AC). It was originally entered in the trial under the mistaken assumption that it was the Japanese cultivar, and was assumed to carry the *Rps* gene, but as its pedigree is unknown, we have no way of knowing if it does carry this gene in addition to the polygenes. However, the mistake has been a fruitful one, because Enshu FC has excellent resistance and is a flue-cured type.

Enshu AC, although an air-cured type, could be of interest to flue-cured breeders because of the possibility of stacking the monogenic and polygenic resistance. If a marker for the *Rps* gene could be identified, it would be possible to a) establish whether Enshu FC does indeed carry the *Rps* gene and b) stack the *Rps* resistance with the very high polygenic resistance found in Oxford 207, giving a level of resistance higher than any yet identified.

It is clear that the polygenic T.I.448A resistance is much superior to both monogenic sources of resistance, as previously reported by Clayton and Smith (1942) and Matsuda and Ohashi (1973).

It seems likely that while the *Rps* gene alone (as in Kokubu) gives adequate resistance at some sites, the resistance is overcome under moderate to heavy disease pressure and needs to be combined with polygenes to be effective.

The *Rxa* gene, carried by the Oriental cultivar Xanthi, does not have a very high level of resistance, as has been reported; like the *Rps* resistance, the *Rxa* resistance alone is not adequate for most sites. Although it might be possible to produce good resistance by stacking this gene with the polygenic resistance, it has been found to be unsuitable as a source of flue-cured resistance, because of its close association with undesirable agronomic characteristics (Sisson, 1992).

There have been no major advances in bacterial wilt resistance breeding since the release of NC 95 in 1963. There have been small increments, maximized in Oxford 207, but no major advances.

4. Genetic control of resistance

This objective has been achieved, and has provided the most valuable information to come out of this trial. However, further work is needed to confirm and quantify the mode of inheritance. For many years, there have been conflicting reports on the nature of the genetic control of resistance to bacterial wilt. Early work in the USA reported that the T.I.448A-derived resistance was polygenic and recessive (Smith and Clayton, 1948), but the Zimbabweans always reported that the resistance had some dominance (Tobacco Research Board, 1961; Schweppenhauser, 1966; Jack, 1994).

It now appears that the T.I.448A-derived resistance is in fact additive, like most other resistance from within the *Nicotiana tabacum* species. Most genes in the *N. tabacum* species are expressed in an additive fashion, with a general lack of dominance (or recessiveness); genes expressing complete dominance tend to be of interspecific origin (Dr E. Wernsman, pers. comm.). It seems that the conflicting reports are due to the different disease pressure at the various sites, sometimes resulting in the inability to distinguish different levels of resistance.

NC 95 gives a much better indication of the level of disease pressure at the various sites than does Hicks; Hicks is so susceptible that the differences between sites are not always clear. Based on the disease progress curves for NC 95 (Figure 1), we classified China and Brazil as high pressure sites, South Africa and the two USA sites as moderate pressure sites and Zimbabwe as a low pressure site.

If the resistance is additive, the resistant x susceptible F_1 (moderate resistance) should fall approximately midway between the susceptible parent (no resistance) and the resistant parent (high resistance). However, under certain levels of disease pressure, it may be difficult to distinguish the F_1 from one of the parents. If the resistance is recessive, the F_1 should always be the same as the susceptible parent; if it is dominant, it should always be the same as the resistant parent - regardless of the level of disease pressure.

Figures 7 to 10 show disease incidence with time for the resistant and susceptible parents and the F_1. Table 4 shows the deviations of the F_1 from the resistant and susceptible parents and from the mid-parent. If the F_1 is significantly different from both parents, but not from the mid-parent, this indicates additive resistance. If the F_1 is significantly different from the resistant parent and the mid-parent, but not from the susceptible parent, this indicates recessive resistance. If the F_1 is significantly different from the susceptible parent and the mid-parent, but not from the resistant parent, this indicates dominant resistance.

The level of resistance in the resistant x susceptible F_1 is generally not very high, and can be overcome under high disease pressure, although it is evident under moderate disease pressure. Figure 7 shows the disease progress curve for the South African site, typical of that for a moderate site (Figure 1); the graphs for the two USA sites are similar. It clearly shows that the F_1 is intermediate between the parents i.e. the resistance is additive. When the significance of the deviations is tested for the NCSU site (Table 4), both the disease incidence and the disease severity data are consistent with additive resistance ie the F_1 is significantly different from both parents, but not from the mid-parent. None of the deviations is significant at the other two sites.

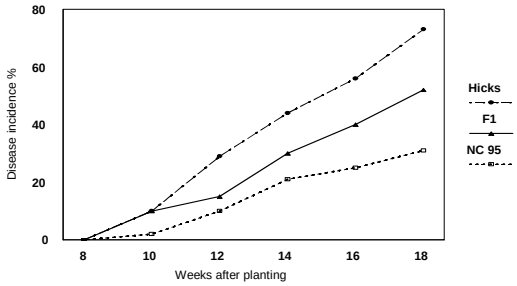

Figure 7: South Africa (moderate pressure site). Disease incidence (% infected plants) for the resistant and susceptible controls and their F_1s.

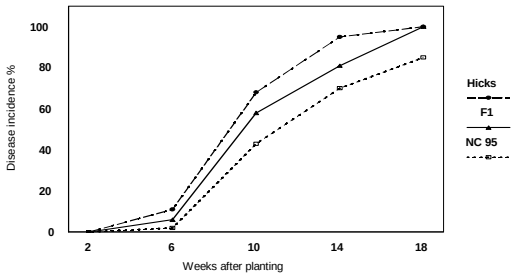

Figure 8: Brazil - Profigen (high pressure site). Disease incidence (% infected plants) for the resistant and susceptible controls and their F_1s.

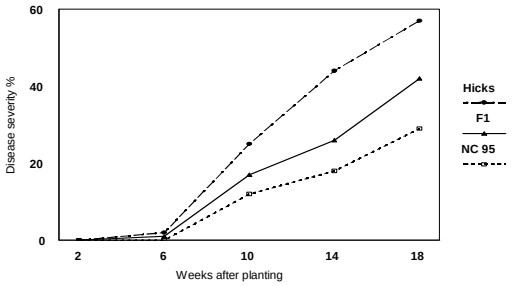

Figure 9: Brazil - Profigen (high pressure site). Disease severity (% infection) for the resistant and susceptible controls and their F_1s.

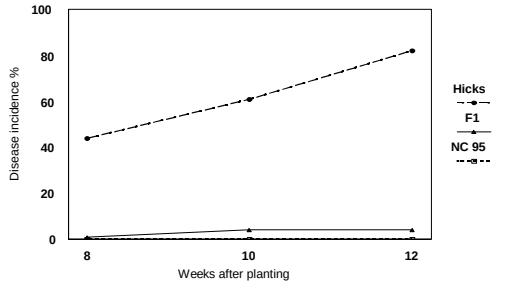

Figure 10: Zimbabwe (low pressure site). Disease incidence (% infected plants) for the resistant and susceptible controls and their F_1s.

Table 4: Deviations of the F_1 from the susceptible parent (S), the resistant parent (R) and the mid-parent (mp)

		S	R	mp	F_1	F_1 - S	F_1 - R	F_1 - mp	$LSD_{0.05}$	CV%
USA NCSU	DI_{16wap}	87.5	14.6	51.1	49.2	-38.3*	34.6*	-1.9^{NS}	31.47	59.5
	DS_{16wap}	85.4	12.5	49.0	43.6	-41.8*	31.1*	-5.4^{NS}	28.61	59.3
	AUDPC	564.6	47.9	306.3	154.1	-410.5*	106.2^{NS}	-152.2^{NS}	261.00	73.3
USA P D	DI_{13wap}	74.7	26.6	50.7	56.6	-18.1	30.0	5.9	-	-
	DI_{13wap}	*1.1*	*0.5*	*0.8*	*0.9*	*-0.2^{NS}*	*0.4^{NS}*	*0.1^{NS}*	*0.41*	*35.6*
	DS	-	-	-	-	-	-	-	-	-
	AUDPC	-	-	-	-	-	-	-	-	-
S Africa LTGA	DI_{18wap}	72.9	31.3	52.1	52.1	-20.8^{NS}	20.8^{NS}	0.0^{NS}	21.07	46.4
	DS_{18wap}	-	-	-	-	-	-	-	-	-
	AUDPC	-	-	-	-	-	-	-	-	-
China QTRI	DI_{14wap}	100.0	100.0	100.0	100.0	0.0^{NS}	0.0^{NS}	0.0^{NS}	8.24	8.6
	DI_{10wap}	100.0	58.3	79.2	87.5	-12.5^{NS}	29.2*	8.3^{NS}	14.83	2.6
	DS_{10wap}	55.2	16.7	36.0	33.9	-21.3*	17.2*	-2.1^{NS}	6.30	24.2
	AUDPC	743.8	458.3	601.1	620.8	-123.0*	162.5*	19.7^{NS}	59.80	11.8
Brazil Profigen	DI_{18wap}	100.0	85.4	92.7	100.0	0.0^{NS}	14.6*	7.3^{NS}	10.43	11.1
	DI_{14wap}	95.8	70.8	83.3	81.2	-14.6^{NS}	10.4^{NS}	-2.1^{NS}	16.53	23.1
	DS_{18wap}	57.3	29.2	43.3	42.7	-14.6*	13.5*	-0.6^{NS}	6.90	18.9
	AUDPC	405.4	183.3	294.4	264.6	-140.8*	81.3*	-29.8^{NS}	64.69	28.2
Zimbabwe TRB	DI_{12wap}	82.1	0.0	41.1	4.2	-77.9*	4.2^{NS}	-36.9*	16.60	120.4
	DS_{12wap}	53.8	0.0	26.9	1.0	-52.8*	1.0^{NS}	-25.9*	10.11	124.2
	AUDPC	135.6	0.0	67.8	3.1	-132.5*	3.1^{NS}	-64.7*	19.92	125.6

DI = disease incidence DS = disease severity AUDPC = Area Under Disease Progress Curve
DI = Arcsine transformation

However, this is not the case with high pressure sites such as China and Brazil (Figure 1). Figure 8 (disease progress curve for Brazil; China was similar) shows that under such circumstances, if one considers only final disease incidence (18 w.a.p.), it may appear that the F_1 resistance is no better than that of the susceptible parent i.e. that the resistance is fully recessive. If one considers earlier disease incidence (14 w.a.p.), the difference between the F_1 and the susceptible parent is generally evident (Figure 8). Although the deviations for earlier disease incidence are not significant in the pattern consistent with additive resistance, they do follow the right trend (Table 4). Alternatively, one can use a finer measurement, such as disease severity, instead of disease incidence (Figure 9) to separate the susceptible parent and the F_1. For both China and Brazil, the deviations for disease severity were consistent with additive resistance (Table 4). The F_1 and the susceptible parent do, therefore, have different

levels of resistance, but they cannot be distinguished phenotypically - at a high pressure site, at the final assessment of disease incidence. This is important because additive resistance enables breeders to distinguish between heterozygous and susceptible plants, which is not possible with recessive resistance.

Conversely, it can be difficult to separate high and moderate resistance at a site with low disease pressure. A similar argument applies to the interpretation of the Zimbabwean results. This site had very low disease pressure (Figure 1), and Figure 10 shows that the F_1 is virtually indistinguishable from the resistant parent i.e. the resistance appears to be dominant (Table 4). However, this is only because of the difficulty of separating the moderately resistant F_1 from the highly resistant parent under low disease pressure, where neither show any symptoms. Again, the F_1 and the resistant parent do have inherently different levels of resistance, but they cannot be distinguished phenotypically - at a low pressure site.

The nature of the genetic control of bacterial wilt has considerable impact on the breeder's choice of selection strategy; the disease pressure at the site and the assessment method are critical. Ideally, in the early stages of the breeding programme, when the identification of heterozygotes is necessary, one should use a site with moderate disease pressure. If this is not possible, one should consider earlier rather than final assessments and one should use the finer measurement of disease severity or AUDPC rather than the coarser measurement of disease incidence. Failure to recognize that heterozygotes can be distinguished from susceptible plants will inevitably result in the rejection of potentially valuable material. In the later stages of the breeding programme, where it is important to separate moderate and high resistance, a high pressure site or an artificially inoculated site would be ideal. At this stage, where rigorous selection is required, one would consider final rather than earlier assessments.

The nature of the genetic control of bacterial wilt also has a bearing on the type of breeding programme employed. If the resistance is dominant or additive, hybrids with only one resistant parent are a feasible option; if the resistance is recessive, this is not an option. With the additive bacterial wilt resistance, this type of hybrid would be suitable only for low pressure sites. In Zimbabwe, all bacterial wilt resistant cultivars are hybrids with one resistant parent; with the low disease pressure in that country, the resistance of these hybrids is quite adequate (Jack, 1998).

It is important to note that these conclusions have been possible only because multi-site data were available. It would not have been possible to reconcile the historically conflicting reports with single site data.

ACKNOWLEDGEMENTS

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APPENDIX - CURRENT PARTICIPANTS

Brazil, Dimon

Mr B Hoppe

Mr I Mallman

DIMON do Brasil Tabacos Ltda
BR 471 Km 48
Cep: 96835-640
Santa Cruz do Sul
RS Brazil

tel: + 55 51 718.7200
fax: + 55 51 718.7249
e-mail: bhoppe@dimon.com.br
imallmann@dimon.com.br

Brazil, Profigen

Mr E Weiss

ProfiGen do Brasil Ltda
P O Box 034
Santa Cruz do Sul
96.800-970
RS - Brazil

tel: + 55 51 715.3309
fax: + 55 51 713.3866
e-mail: profigen@viavale.com.br

Bulgaria, TTPI

Mr H Bozukov

Tobacco & Tobacco Products Institute
4108 Plovdiv
Bulgaria

tel: + 359 32 672.364
fax: + 359 32 775.156

China, QTRI

Prof Z Xianchao

Qingzhou Tobacco Research Institute
11 Xiangshan Road
Qingzhou
Shandong
262500
China

tel: + 86 536 328.0967
fax: + 86 536 328.0667
e-mail: "bh"<phyto@public.wfptt.sd.cn>

Iran, Tirtash

R A Mahtabi

Tobacco Research Institute of Tirtash
P O Box 48515 - 155
Behshahr
Iran

tel: + 98 1572 210.56
fax: + 98 1572 210.55

South Africa, LTGA
Mr A Scholtz
Lowveld Tobacco Growers Association
P O Box 60
Nelspruit
Mpumalanga 1200
South Africa

tel: + 27 13 752.8411
fax: + 27 13 752.2186
e-mail: ltga@lk.co.za

South Africa, IIC
Mrs J Terblanche
Institute for Industrial Crops
P O Box 82075
Rustenburg 0300
South Africa

tel: + 27 14 536.3150
fax: + 27 14 536.3113
e-mail: jody@nitk1.agric.za

USA, PD
Prof B Fortnum
Pee Dee Centre
2200 Pocket Road
Florence SC
29506 9706
USA

tel: + 1 803 662.3526 ext 235
fax: + 1 803 661.5676
e-mail: bfrtnm@clemson.edu

USA, NCSU
Dr V Sisson
Oxford Tobacco Research Station
P O Box 1555
Oxford
NC 27565
USA

tel: + 1 919 693.5151
fax: + 1 919 693.3870
e-mail: verne_sisson@ncsu.edu