

HANDBOOK OF Plant Disease Identification and Management



BALAJI AGLAVE



CRC Press
Taylor & Francis Group

Handbook of Plant Disease Identification and Management



Taylor & Francis

Taylor & Francis Group

<http://taylorandfrancis.com>

Handbook of Plant Disease Identification and Management

Balaji Aglave



CRC Press

Taylor & Francis Group

Boca Raton London New York

CRC Press is an imprint of the
Taylor & Francis Group, an **informa** business

CRC Press
Taylor & Francis Group
6000 Broken Sound Parkway NW, Suite 300
Boca Raton, FL 33487-2742

© 2019 by Taylor & Francis Group, LLC
CRC Press is an imprint of Taylor & Francis Group, an Informa business

No claim to original U.S. Government works

Printed on acid-free paper

International Standard Book Number-13: 978-1-138-58547-8 (Hardback)

This book contains information obtained from authentic and highly regarded sources. Reasonable efforts have been made to publish reliable data and information, but the author and publisher cannot assume responsibility for the validity of all materials or the consequences of their use. The authors and publishers have attempted to trace the copyright holders of all material reproduced in this publication and apologize to copyright holders if permission to publish in this form has not been obtained. If any copyright material has not been acknowledged please write and let us know so we may rectify in any future reprint.

Except as permitted under U.S. Copyright Law, no part of this book may be reprinted, reproduced, transmitted, or utilized in any form by any electronic, mechanical, or other means, now known or hereafter invented, including photocopying, microfilming, and recording, or in any information storage or retrieval system, without written permission from the publishers.

For permission to photocopy or use material electronically from this work, please access www.copyright.com (<http://www.copyright.com/>) or contact the Copyright Clearance Center, Inc. (CCC), 222 Rosewood Drive, Danvers, MA 01923, 978-750-8400. CCC is a not-for-profit organization that provides licenses and registration for a variety of users. For organizations that have been granted a photocopy license by the CCC, a separate system of payment has been arranged.

Trademark Notice: Product or corporate names may be trademarks or registered trademarks, and are used only for identification and explanation without intent to infringe.

Library of Congress Cataloging-in-Publication Data

Names: Aglave, Balaji, author.
Title: Handbook of plant disease identification and management / Balaji Aglave.
Description: Boca Raton, Florida : CRC Press, [2019]
Identifiers: LCCN 2018017131 | ISBN 9781138585478 (hardback : alk. paper) | ISBN 9780429504907 (ebook)
Subjects: LCSH: Plant diseases--Handbooks, manuals, etc. | Phytopathogenic microorganisms--Control.
Classification: LCC SB731 .A34 2018 | DDC 632/.3--dc23
LC record available at <https://lccn.loc.gov/2018017131>

Visit the Taylor & Francis Web site at
<http://www.taylorandfrancis.com>

and the CRC Press Web site at
<http://www.crcpress.com>

Contents

Preface.....	xxxvii
About the Author	xxxix
Chapter 1 Strawberry	1
1.1 Anthracnose of Strawberry	1
1.1.1 Causal Organism	1
1.1.2 Symptoms.....	2
1.1.2.1 Fruit Rot.....	2
1.1.2.2 Crown Rot.....	2
1.1.2.3 Petioles and Stolons	2
1.1.2.4 Leaf Spot.....	3
1.1.3 Cause and Disease Development.....	3
1.1.4 Favorable Conditions of Disease Development.....	3
1.1.5 Disease Cycle	4
1.1.6 Management	5
1.1.6.1 Chemical Control.....	5
1.1.6.2 Biological Control.....	5
1.1.6.3 Cultural Control.....	5
1.2 Powdery Mildew of Strawberry	6
1.2.1 Causal Organism	6
1.2.2 Symptoms.....	6
1.2.3 Cause and Disease Development.....	7
1.2.4 Favorable Conditions.....	7
1.2.5 Disease Cycle	9
1.2.6 Management	9
1.2.6.1 Chemical Control.....	11
1.2.6.2 Biological Control.....	11
1.2.6.3 Cultural Control.....	11
1.3 Leaf Scorch of Strawberry	11
1.3.1 Causal Organism.....	13
1.3.2 Symptoms.....	13
1.3.2.1 Leaves	13
1.3.2.2 Leaf Stems (Petioles)	14
1.3.2.3 Fruit	14
1.3.3 Cause and Disease Development.....	14
1.3.4 Favorable Conditions of Disease Development.....	15
1.3.5 Disease Cycle	15
1.3.6 Management	15
1.3.6.1 Chemical Control.....	15
1.3.6.2 Cultural Control.....	16
1.4 Crinkle Virus.....	16
1.4.1 Causal Organism	16
1.4.2 Species Affected.....	17
1.4.3 Symptoms.....	17
1.4.4 Means of Movement and Transmission.....	17
1.4.5 Prevention and Control.....	17

1.5	Latent C Virus (SLCV)	17
1.5.1	Causal Organism	17
1.5.2	Species Affected by SLCV	18
1.5.3	Symptoms.....	18
1.5.4	Means of Movement and Transmission.....	18
1.5.5	Prevention and Control.....	18
1.6	Mild Yellow Edge Virus.....	18
1.6.1	Causal Organism	18
1.6.2	Species Affected.....	18
1.6.3	Symptoms.....	19
1.6.4	Means of Movement and Transmission.....	19
1.6.5	Prevention and Control.....	19
1.7	Mottle Disease	19
1.7.1	Causal Organism	19
1.7.2	Species Affected.....	20
1.7.3	Symptoms.....	20
1.7.4	Means of Movement and Transmission.....	20
1.7.5	Prevention and Control.....	20
1.8	Necrotic Shock Virus	20
1.8.1	Causal Organism	20
1.8.2	Species Affected.....	20
1.8.3	Symptoms.....	20
1.8.4	Means of Movement and Transmission.....	21
1.8.5	Prevention and Control.....	21
1.9	Vein Banding <i>Caulimovirus</i>	21
1.9.1	Causal Organism	21
1.9.2	Species Affected.....	21
1.9.3	Symptoms.....	21
1.9.3.1	On <i>F. vesca</i>	21
1.9.3.2	On Commercial Strawberries	21
1.9.4	Means of Movement and Transmission.....	22
1.9.5	Prevention and Control.....	22
1.10	<i>Phomopsis</i> Leaf Blight of Strawberry	22
1.10.1	Causal Organism	22
1.10.2	Symptoms.....	23
1.10.3	Cause and Disease Development.....	24
1.10.4	Favorable Conditions of Disease Development.....	24
1.10.5	Disease Cycle	24
1.10.6	Management	24
1.11	Gray Mold.....	25
1.11.1	Causal Organism	25
1.11.2	Symptoms.....	25
1.11.3	Cause and Disease Development.....	26
1.11.4	Favorable Conditions of Disease Development.....	27
1.11.5	Disease Cycle	28
1.11.6	Management	29
1.11.6.1	Chemical Control.....	30
1.11.6.2	Biological Control.....	30
1.11.6.3	Cultural Control.....	30
1.12	Leather Rot of Strawberry.....	31
1.12.1	Causal Organism	31

1.12.2	Symptoms.....	31
1.12.3	Cause and Disease Development.....	32
1.12.4	Favorable Conditions of Disease Development.....	33
1.12.5	Disease Cycle	33
1.12.6	Management	34
1.12.6.1	Chemical Control.....	34
1.12.6.2	Cultural Control.....	34
1.12.6.3	Soil Solarization	34
1.13	<i>Rhizopus</i> Fruit Rot.....	35
1.13.1	Causal Organism	35
1.13.2	Symptoms.....	35
1.13.3	Cause and Disease Development.....	35
1.13.4	Favorable Conditions of Disease Development.....	35
1.13.5	Disease Cycle	35
1.13.6	Management	36
1.13.6.1	Cultural Control.....	36
1.13.6.2	Organically Acceptable Methods	36
1.13.6.3	Treatment Decisions	36
1.14	Red Stele Root Rot of Strawberry	36
1.14.1	Causal Organism	36
1.14.2	Symptoms.....	36
1.14.2.1	Aboveground.....	36
1.14.2.2	Below Ground.....	37
1.14.3	Cause and Disease Development.....	37
1.14.4	Favorable Conditions of Disease Development.....	38
1.14.5	Disease Cycle	38
1.14.6	Management	39
1.14.6.1	Chemical Control.....	39
1.14.6.2	Cultural Control.....	39
1.14.6.3	Biological Control.....	40
1.15	<i>Phytophthora</i> Crown Rot.....	40
1.15.1	Causal Organism	41
1.15.2	Symptoms.....	41
1.15.3	Cause and Disease Development.....	41
1.15.4	Favorable Conditions of Disease Development.....	41
1.15.5	Disease Cycle	42
1.15.6	Management	42
1.16	<i>Rhizoctonia</i> Root Rot	43
1.16.1	Causal Organism	43
1.16.2	Symptoms.....	43
1.16.3	Cause and Disease Development.....	44
1.16.4	Favorable Conditions of Disease Development.....	44
1.16.5	Management	45
1.17	Charcoal Rot of Strawberry.....	46
1.17.1	Causal Organism	46
1.17.2	Symptoms.....	46
1.17.3	Cause and Disease Development.....	46
1.17.4	Favorable Conditions of Disease Development.....	46
1.17.5	Management	47
1.18	Gnomonia Fruit Rot and Leaf Blotch.....	47
1.18.1	Causal Organism	47

1.18.2	Symptoms.....	47
1.18.3	Cause and Disease Development.....	48
1.19	Leaf Spot of Strawberry	49
1.19.1	Causal Organism.....	50
1.19.2	Symptoms.....	50
1.19.2.1	Leaves	50
1.19.2.2	Leaf Stems (Petioles), Runners, Fruit Stalks (Pedicels), Berry Caps (Calyxes)	50
1.19.2.3	Fruit	50
1.19.3	Cause and Disease Development.....	51
1.19.4	Favorable Conditions of Disease Development.....	51
1.19.5	Disease Cycle	51
1.19.6	Management	53
1.19.6.1	Chemical Control.....	53
1.19.6.2	Cultural Control.....	53
1.19.6.3	Preventive Treatment	53
1.20	Bacterial Leaf Spot of Strawberry.....	53
1.20.1	Causal Organism	54
1.20.2	Symptoms.....	54
1.20.3	Cause and Disease Development.....	56
1.20.4	Favorable Conditions of Disease Development.....	56
1.20.5	Disease Cycle	56
1.20.6	Management	57
1.21	The Dagger Nematode of Strawberry	57
1.21.1	Distribution of the Causal Organism	57
1.21.2	Symptoms.....	57
1.21.3	Disease Cycle	58
1.21.4	Management	58
1.21.4.1	Chemical Control.....	58
1.21.4.2	Cultural Control.....	58
1.22	The Foliar Nematode.....	59
1.22.1	The Causal Organism.....	59
1.22.2	Symptoms.....	60
1.22.3	Favorable Conditions.....	60
1.22.4	Disease Cycle	60
1.22.5	Management	61
1.23	The Lesion Nematode of Strawberry	61
1.23.1	The Causal Organism.....	61
1.23.2	Symptoms.....	62
1.23.3	Favorable Conditions.....	62
1.23.4	Disease Cycle	62
1.23.5	Management	63
1.23.5.1	Chemical Control.....	63
1.23.5.2	Cultural Control.....	64
1.24	The Sting Nematode	64
1.24.1	The Causal Organism.....	65
1.24.2	Symptoms.....	65
1.24.3	Disease Cycle	65
1.24.4	Management	66
1.24.4.1	Chemical Control.....	66

1.24.4.2 Cultural Control.....	67
1.24.4.3 Biological Control.....	67
1.25 Verticillium Wilt of Strawberry	67
1.25.1 Causal Organism	67
1.25.2 Symptoms.....	67
1.25.3 Cause and Disease Development.....	68
1.25.4 Favorable Conditions of Disease Development.....	68
1.25.5 Disease Cycle	68
1.25.6 Management	69
1.25.6.1 Chemical Control.....	69
1.25.6.2 Cultural Control.....	69
References	70
Chapter 2 Tomato.....	71
2.1 Anthracnose of Tomato	71
2.1.1 Causal Organism	71
2.1.2 Symptoms.....	71
2.1.3 Cause and Disease Development.....	73
2.1.4 Favorable Conditions of Disease Development.....	73
2.1.5 Disease Cycle	73
2.1.6 Management	74
2.2 Bacterial Canker of Tomato	74
2.2.1 Causal Organism	74
2.2.2 Symptoms.....	75
2.2.2.1 Seedlings.....	75
2.2.2.2 Leaf and Plant.....	75
2.2.2.3 Fruit	75
2.2.3 Cause and Disease Development.....	76
2.2.4 Favorable Conditions of Disease Development.....	76
2.2.5 Management	76
2.3 Bacterial Speck of Tomato	77
2.3.1 Causal Organism	77
2.3.2 Symptoms.....	77
2.3.3 Cause and Disease Development.....	78
2.3.4 Favorable Conditions of Disease Development.....	78
2.3.5 Management	78
2.4 Bacterial Spot of Tomato.....	79
2.4.1 Causal Organism	79
2.4.2 Symptoms.....	79
2.4.3 Cause and Disease Development.....	79
2.4.4 Favorable Conditions of Disease Development.....	80
2.4.5 Disease Cycle	80
2.4.6 Management	80
2.5 Septoria Leaf Spot on Tomato	81
2.5.1 Causal Organism	81
2.5.2 Symptoms.....	81
2.5.3 Cause and Disease Development.....	82
2.5.4 Favorable Conditions for Disease Development.....	82
2.5.5 Disease Cycle	82

2.5.6	Management	83
2.5.6.1	Cultural Control.....	83
2.5.6.2	Chemical Control.....	83
2.5.6.3	Resistance	83
2.6	Cucumber Mosaic Virus on Tomato	84
2.6.1	Causal Organism	84
2.6.2	Symptoms.....	84
2.6.3	Means of Movement and Transmission.....	84
2.6.4	Potato Virus Y	85
2.6.5	Symptoms.....	85
2.6.6	Means of Movement and Transmission.....	86
2.6.7	Tobacco Etch Virus	86
2.6.8	Symptoms.....	86
2.6.9	Means of Movement and Transmission.....	86
2.6.10	Prevention and Control of CMV, PVY, and TEV	86
2.7	Common Mosaic of Tomato (Tobacco/Tomato Mosaic)	87
2.7.1	Causal Organism	87
2.7.2	Symptoms.....	87
2.7.3	Means of Movement and Transmission.....	88
2.7.4	Prevention and Control.....	88
2.8	Tomato Spotted Wilt Virus.....	89
2.8.1	Causal Organism	89
2.8.2	Symptoms.....	89
2.8.3	Means of Movement and Transmission.....	90
2.8.4	Prevention and Control.....	90
2.9	Disorders of Tomato	90
2.9.1	Blossom-End Rot.....	90
2.9.1.1	Symptoms	90
2.9.1.2	Management	91
2.9.2	Catfacing	91
2.9.2.1	Cause.....	91
2.9.2.2	Symptoms	91
2.9.2.3	Management	92
2.9.3	Blotchy Ripening.....	92
2.9.3.1	Management	92
2.9.4	Sunscald	92
2.9.4.1	Symptoms	93
2.9.4.2	Management	93
2.9.5	Fruit Cracking	93
2.9.5.1	Radial Cracking.....	93
2.9.5.2	Concentric Cracking.....	94
2.9.5.3	Management	94
2.9.6	Physiological Leaf Roll	94
2.9.6.1	Cause.....	95
2.9.6.2	Symptoms	95
2.9.6.3	Management	95
2.9.7	Puffiness	96
2.9.7.1	Cause.....	96
2.9.7.2	Symptoms	96
2.9.7.3	Management	96
2.10	Early Blight of Tomato	96

2.10.1	Causal Organism.....	96
2.10.2	Symptoms.....	96
2.10.3	Cause and Disease Development.....	97
2.10.4	Favorable Conditions of Disease Development.....	98
2.10.5	Disease Cycle	98
2.10.6	Management	98
2.10.6.1	Cultural Control.....	98
2.10.6.2	Chemical Control.....	99
2.10.6.3	Resistance	99
2.11	Late Blight of Tomato.....	99
2.11.1	Causal Organism.....	99
2.11.2	Symptoms.....	100
2.11.2.1	On Tomato Leaves	100
2.11.2.2	On Tomato Petioles and Stems	100
2.11.2.3	On Tomato Fruits.....	101
2.11.3	Cause and Disease Development.....	101
2.11.4	Favorable Conditions of Disease Development.....	102
2.11.5	Disease Cycle	102
2.11.5.1	Dissemination	102
2.11.5.2	Inoculation	102
2.11.5.3	Infection and Pathogen Development.....	102
2.11.5.4	Symptom and Disease Development	102
2.11.6	Management	103
2.12	Southern Blight on Tomato.....	104
2.12.1	Causal Organism.....	104
2.12.2	Symptoms.....	104
2.12.3	Favorable Conditions of Disease Development.....	105
2.12.4	Disease Cycle	105
2.12.5	Management	105
2.12.5.1	Integrated Management	105
2.12.5.2	Chemical Control.....	106
2.12.5.3	Biological Control.....	107
2.13	Gray Mold on Tomato.....	107
2.13.1	Causal Organism.....	107
2.13.2	Symptoms.....	107
2.13.3	Cause and Disease Development.....	108
2.13.4	Favorable Conditions of Disease Development.....	109
2.13.5	Disease Cycle	109
2.13.6	Management	110
2.13.6.1	Cultural Control.....	110
2.13.6.2	Chemical Control	110
2.13.6.3	Resistance Management	110
2.14	Root Knot Nematode.....	110
2.14.1	Symptoms and Damage.....	111
2.14.2	Life Cycle	111
2.14.3	Management	112
2.15	Stubby-Root Nematode.....	112
2.15.1	Symptoms and Damage.....	113
2.15.2	Management	113
2.15.3	Sting Nematode on Tomato.....	113
2.15.4	Symptoms.....	113

2.16	Fusarium Crown and Root Rot.....	113
2.16.1	Causal Organism	114
2.16.2	Symptoms.....	114
2.16.3	Cause and Disease Development.....	115
2.16.4	Favorable Conditions of Disease Development.....	115
2.16.5	Disease Cycle	115
2.16.6	Management	115
2.16.6.1	Cultural control.....	115
2.16.6.2	Chemical Control.....	117
2.16.6.3	Biological Control.....	118
2.16.6.4	Integrated Management	120
2.17	Fusarium Wilt of Tomato	121
2.17.1	Causal Organism	121
2.17.2	Symptoms.....	121
2.17.3	Cause and Disease Development.....	121
2.17.4	Favorable Conditions of Disease Development.....	121
2.17.5	Disease Cycle	122
2.17.6	Management	122
2.17.6.1	Integrated Pest Management Strategies.....	122
2.18	Verticillium Wilt of Tomato	122
2.18.1	Causal Organism	122
2.18.2	Symptoms.....	122
2.18.3	Favorable Conditions of Disease Development.....	123
2.18.4	Disease Cycle	123
2.18.5	Management	124
2.19	Powdery Mildew of Tomato	124
2.19.1	Causal Organism	125
2.19.2	Symptoms.....	125
2.19.3	Favorable Conditions of Disease Development.....	126
2.19.4	Disease Cycle	126
2.19.5	Management	126
2.19.5.1	Sulfur	126
	References	127
Chapter 3	Citrus	129
3.1	<i>Alternaria</i> Brown Spot of Citrus	129
3.1.1	Causal Organism	129
3.1.2	Symptoms.....	129
3.1.3	Favorable Conditions for Disease Development.....	129
3.1.4	Disease Cycle	131
3.1.5	Management	131
3.2	Citrus Black Spot.....	131
3.2.1	Causal Organism	132
3.2.2	Symptoms.....	132
3.2.2.1	Hard Spot.....	132
3.2.2.2	False Melanose	132
3.2.2.3	Cracked Spot.....	133
3.2.2.4	Virulent Spot.....	133
3.2.2.5	Symptoms on Leaves and Stem.....	133
3.2.3	Cause and Disease Development.....	134

3.2.4	Favorable Conditions of Disease Development.....	134
3.2.5	Disease Cycle	134
3.2.6	Management	135
3.3	Greasy Spot of Citrus	136
3.3.1	Causal Organism.....	136
3.3.2	Symptoms.....	136
3.3.3	Cause and Disease Development.....	136
3.3.4	Favorable Conditions of Disease Development.....	137
3.3.5	Disease Cycle	137
3.3.6	Management	138
3.4	Alternaria Rot of Citrus.....	139
3.4.1	Causal Organism.....	139
3.4.2	Symptoms.....	139
3.4.3	Favorable Conditions for Disease Development.....	139
3.4.4	Disease Cycle	139
3.4.5	Management	141
3.5	Citrus Brown Rot.....	141
3.5.1	Causal Organism.....	141
3.5.2	Symptoms.....	141
3.5.3	Cause and Disease Development.....	142
3.5.4	Favorable Conditions of Disease Development.....	143
3.5.5	Disease Cycle	143
3.5.6	Management	143
3.5.6.1	Cultural Practices	143
3.5.6.2	Fungicidal Protection.....	143
3.6	Phytophthora Foot Rot and Root Rot of Citrus	144
3.6.1	Causal Organism	144
3.6.2	Symptoms.....	144
3.6.2.1	Foot Rot/Gummosis.....	144
3.6.2.2	Root Rot.....	144
3.6.3	Cause and Disease Development.....	146
3.6.4	Disease Cycle	146
3.6.5	Management	147
3.7	Anthracnose of Citrus.....	147
3.7.1	Causal Organism	147
3.7.2	Symptoms.....	147
3.7.2.1	Leaf	147
3.7.2.2	Fruit	147
3.7.3	Cause and Disease Development.....	147
3.7.4	Favorable Conditions of Disease Development.....	152
3.7.5	Disease Cycle	153
3.7.6	Management	153
3.8	Citrus Canker.....	154
3.8.1	Causal Organism.....	154
3.8.2	Symptoms.....	154
3.8.2.1	Leaf	154
3.8.2.2	Fruit	155
3.8.2.3	Twigs.....	156
3.8.3	Cause and Disease Development.....	156
3.8.4	Favorable Conditions of Disease Development.....	156
3.8.5	Disease Cycle	157

3.8.6	Management	158
3.8.6.1	Cultural Practices	158
3.8.6.2	Chemical Control.....	158
3.8.6.3	Integrated Management Programs	159
3.9	Citrus Greening	159
3.9.1	Causal Organism	160
3.9.2	Symptoms.....	160
3.9.3	Transmission of the Disease.....	160
3.9.4	Disease Cycle	161
3.10	Citrus Variegated Chlorosis.....	161
3.10.1	Causal Organism	161
3.10.2	Symptoms.....	162
3.10.2.1	Leaf	162
3.10.2.2	Fruit	162
3.10.2.3	Whole Tree	162
3.10.3	Favorable Conditions for Disease Development and Disease Transmission	163
3.10.4	Disease Cycle	163
3.10.5	Management	163
3.11	Green Mold on Citrus.....	164
3.11.1	Causal Organism	164
3.11.2	Symptoms.....	164
3.11.3	Cause and Disease Development.....	164
3.11.4	Favorable Conditions of Disease Development.....	165
3.11.5	Disease Cycle	165
3.11.6	Management	166
3.12	Citrus Melanose.....	166
3.12.1	Causal Organism	166
3.12.2	Symptoms.....	166
3.12.2.1	Leaf	166
3.12.2.2	Fruit	166
3.12.3	Cause and Disease Development.....	167
3.12.4	Favorable Conditions of Disease Development.....	167
3.12.5	Disease Cycle	168
3.12.5.1	Dispersal	168
3.12.5.2	Infection.....	168
3.12.6	Management	168
3.13	Postbloom Fruit Drop	168
3.13.1	Causal Organism	169
3.13.2	Symptoms.....	169
3.13.3	Cause and Disease Development.....	170
3.13.4	Favorable Conditions of Disease Development.....	171
3.13.4.1	Rainfall	171
3.13.4.2	Leaf Wetness	171
3.13.5	Disease Cycle	171
3.13.6	Management	171
3.14	Sooty Blotch of Citrus	172
3.14.1	Causal Organism	172
3.14.2	Symptoms.....	172
3.14.3	Favorable Conditions of Disease Development.....	173
3.14.4	Management	173

3.15	Sweet Orange Scab.....	173
3.15.1	Causal Organism.....	174
3.15.2	Symptoms.....	174
3.15.3	Cause and Disease Development.....	175
3.15.4	Favorable Conditions of Disease Development.....	175
3.15.5	Disease Cycle	175
3.15.6	Management	175
	References	175
Chapter 4	Apple	177
4.1	Alternaria Blotch of Apple	177
4.1.1	Causal Organism.....	177
4.1.2	Symptoms.....	177
4.1.3	Cause and Disease Development.....	179
4.1.4	Favorable Conditions of Disease Development.....	179
4.1.5	Disease Cycle	179
4.1.6	Management	179
4.2	Apple Scab.....	179
4.2.1	Causal Organism.....	179
4.2.2	Symptoms.....	179
4.2.3	Cause and Disease Development.....	181
4.2.4	Favorable Conditions of Disease Development.....	181
4.2.5	Disease Cycle	181
4.2.6	Management	181
4.3	Fire Blight of Apple.....	182
4.3.1	Causal Organism.....	182
4.3.2	Symptoms.....	183
4.3.3	Favorable Conditions of Disease Development.....	186
4.3.4	Disease Cycle	186
4.3.5	Management	187
4.4	Apple Powdery Mildew	189
4.4.1	Causal Organism.....	189
4.4.2	Symptoms.....	190
4.4.3	Cause and Disease Development.....	190
4.4.4	Favorable Conditions of Disease Development.....	191
4.4.5	Disease Cycle	191
4.4.6	Management	191
4.5	Sooty Blotch and Flyspeck of Apple	191
4.5.1	Causal Organism.....	192
4.5.2	Symptoms.....	192
4.5.2.1	Sooty Blotch	192
4.5.2.2	Flyspeck	192
4.5.3	Cause and Disease Development.....	192
4.5.3.1	<i>Sooty Blotch</i>	192
4.5.3.2	<i>Flyspeck</i>	192
4.5.4	Favorable Conditions of Disease Development.....	193
4.5.4.1	<i>Sooty Blotch</i>	193
4.5.4.2	<i>Flyspeck</i>	193
4.5.5	Disease Cycle	193
4.5.6	Management	194

4.6	Apple Mosaic Virus.....	195
4.6.1	Causal Organism	195
4.6.2	Symptoms.....	195
4.6.3	Means of Movement and Transmission.....	195
4.6.4	Prevention and Control.....	197
4.7	Apple Stem Grooving Virus.....	197
4.7.1	Causal Organism	197
4.7.2	Symptoms.....	197
4.7.3	Means of Movement and Transmission.....	197
4.7.4	Prevention and Control.....	198
4.8	Apple Stem Pitting Virus	198
4.8.1	Causal Organism	198
4.8.2	Symptoms.....	198
4.8.3	Means of Movement and Transmission.....	198
4.9	Apple Union Necrosis and Decline	198
4.9.1	Causal Organism	199
4.9.2	Symptoms.....	199
4.9.3	Means of Movement and Transmission.....	199
4.9.4	Prevention and Control.....	199
4.10	Blister Spot of Apple	199
4.10.1	Causal Organism	199
4.10.2	Symptoms.....	199
4.10.3	Cause and Disease Development.....	200
4.10.4	Favorable Conditions of Disease Development.....	200
4.10.5	Disease Cycle	200
4.10.6	Management	200
4.11	Blue Mold of Apple	201
4.11.1	Causal Organism	201
4.11.2	Symptoms.....	201
4.11.3	Cause and Disease Development.....	202
4.11.4	Favorable Conditions of Disease Development.....	203
4.11.5	Disease Cycle	203
4.11.6	Management	203
4.12	Botrytis Rot of Apple	204
4.12.1	Causal Organism	204
4.12.2	Symptoms.....	204
4.12.2.1	In the orchard.....	204
4.12.2.2	In Store.....	204
4.12.2.3	On Other Apple Varieties Botrytis Rot is Mainly Mid-Brown	204
4.12.3	Cause and Disease Development.....	205
4.12.4	Favorable Conditions of Disease Development.....	205
4.12.5	Disease Cycle	206
4.12.6	Management	206
4.12.6.1	Cultural Control.....	206
4.12.6.2	Biological Control.....	207
4.12.6.3	Chemical Control.....	207
4.13	Disorders of Apple.....	207
4.13.1	Bitter Pit	207
4.13.1.1	Management	208
4.13.2	Water Core.....	208

4.13.2.1	Management	209
4.13.3	Brown Heart	209
4.13.3.1	Management	210
4.13.4	Apple Sunburn (Sunscald).....	210
4.13.4.1	Management	210
4.13.5	Scald.....	211
4.13.5.1	Management	211
References		211
Chapter 5	Banana.....	213
5.1	Banana Bacterial Wilt	213
5.1.1	Causal Organism.....	213
5.1.2	Symptoms.....	213
5.1.3	Cause and Disease Development.....	213
5.1.4	Management	215
5.1.4.1	Disease Cycle.....	215
5.2	Banana Bract Mosaic Virus.....	215
5.2.1	Causal Organism	216
5.2.2	Symptoms.....	216
5.2.3	Means of Movement and Transmission.....	218
5.2.4	Prevention and Control.....	218
5.3	Banana Mild Mosaic Virus	218
5.3.1	Causal Organism	218
5.3.2	Symptoms.....	218
5.3.3	Means of Movement and Transmission.....	218
5.3.4	Prevention and Control.....	219
5.4	Banana Streak Virus.....	219
5.4.1	Causal Organism	219
5.4.2	Symptoms.....	219
5.4.3	Means of Movement and Transmission.....	220
5.4.4	Prevention and Control.....	220
5.5	CMV	220
5.5.1	Causal Organism	221
5.5.2	Symptoms.....	221
5.5.3	Means of Movement and Transmission.....	222
5.5.4	Prevention and Control.....	222
5.6	Burrowing Nematode	222
5.6.1	Symptoms.....	222
5.6.2	Life Cycle	223
5.6.3	Conditions that Favor Development	223
5.6.4	Prevention and Control.....	223
5.7	Root Knot Nematode	224
5.7.1	Life Cycle, Symptoms, and Damage	224
5.7.2	Management	224
5.8	Cigar-End Rot of Banana.....	225
5.8.1	Causal Organism	226
5.8.2	Symptoms.....	226
5.8.3	Favorable Conditions of Disease Development.....	226
5.8.4	Management	226
5.9	Disorders.....	227

5.9.1	Choke Throat.....	227
5.9.2	Management	227
5.9.3	Chilling Injury.....	228
5.9.4	Potassium Deficiency	228
	References	228
Chapter 6	Pepper.....	231
6.1	Anthracnose of Pepper	231
6.1.1	Causal Organism	231
6.1.2	Symptoms.....	231
6.1.3	Cause and Disease Development.....	231
6.1.4	Favorable Conditions of Disease Development.....	232
6.1.5	Disease Cycle	232
6.1.6	Management	233
6.2	Bacterial Spot of Pepper.....	233
6.2.1	Causal Organism	233
6.2.2	Symptoms.....	233
6.2.3	Cause and Disease Development.....	233
6.2.4	Favorable Conditions of Disease Development.....	234
6.2.5	Disease Cycle	234
6.2.6	Management	235
6.3	Cercospora Leaf Spot.....	235
6.3.1	Causal Organism	236
6.3.2	Symptoms.....	236
6.3.3	Cause and Disease Development.....	236
6.3.4	Favorable Conditions of Disease Development.....	236
6.3.5	Disease Cycle	236
6.3.6	Management	237
6.4	CMV, PVY, TEV.....	237
6.4.1	Cucumber Mosaic Virus on Pepper	237
6.4.1.1	Causal Organism	237
6.4.1.2	Symptoms	238
6.4.1.3	Means of Movement and Transmission	238
6.4.2	Potato Virus Y.....	238
6.4.2.1	Symptoms	238
6.4.2.2	Means of Movement and Transmission	238
6.4.3	Tobacco Etch Virus	238
6.4.3.1	Symptoms	239
6.4.3.2	Means of Movement and Transmission	239
6.4.4	Prevention and Control of CMV, PVY, and TEV	239
6.5	Pepper Mild Mottle Virus	239
6.5.1	Causal Organism	239
6.5.2	Symptoms.....	239
6.5.3	Means of Movement and Transmission.....	240
6.5.4	Prevention and Control.....	240
6.6	TMV on Pepper.....	241
6.6.1	Causal Organism	241
6.6.2	Symptoms.....	241
6.6.3	Means of Movement and Transmission.....	242
6.6.4	Prevention and Control.....	242

6.7	Tomato Spotted Wilt Virus.....	243
6.7.1	Causal Organism	243
6.7.2	Symptoms.....	243
6.7.3	Means of Movement and Transmission.....	243
6.7.4	Prevention and Control.....	244
6.8	Blossom-End Rot of Pepper.....	244
6.8.1	Symptoms.....	244
6.8.2	Management	245
6.8.3	Sunscald	245
6.8.4	Symptoms.....	245
6.8.5	Management	246
6.8.6	Misshapen Fruit.....	246
6.8.6.1	Cause.....	246
6.8.6.2	Prevention	246
6.9	Fusarium Stem and Fruit Rot of Pepper.....	246
6.9.1	Causal Organism	246
6.9.2	Symptoms.....	247
6.9.3	Cause and Disease Development.....	248
6.9.4	Management	248
6.10	Southern Blight on Pepper.....	248
6.10.1	Causal Organism	248
6.10.2	Symptoms.....	248
6.10.3	Favorable Conditions of Disease Development.....	249
6.10.4	Disease Cycle	249
6.10.5	Management	249
6.10.5.1	Integrated Management	249
6.10.5.2	Chemical Control.....	250
6.10.5.3	Biological Control.....	251
6.11	Powdery Mildew of Pepper	251
6.11.1	Causal Organism	251
6.11.2	Symptoms.....	251
6.11.3	Cause and Disease Development.....	252
6.11.4	Favorable Conditions of Disease Development.....	252
6.11.5	Disease Cycle	252
6.11.6	Management	253
6.12	Phytophthora Blight.....	253
6.12.1	Causal Organism	253
6.12.2	Symptoms.....	253
6.12.3	Cause and Disease Development.....	255
6.12.4	Favorable Conditions of Disease Development.....	255
6.12.5	Disease Cycle	255
6.12.6	Management	256
6.13	Root Knot Nematode	256
6.13.1	Symptoms and Damage.....	256
6.13.2	Life Cycle	257
6.13.3	Management	257
6.13.4	Sting Nematode on Pepper	258
6.13.5	Symptoms.....	258
6.14	Verticillium Wilt of Pepper	258
6.14.1	Causal Organism	258
6.14.2	Symptoms.....	258

6.14.3	Cause and Disease Development.....	258
6.14.4	Favorable Conditions of Disease Development.....	259
6.14.5	Disease Cycle	259
6.14.6	Management	259
	References	260
Chapter 7	Potato.....	261
7.1	Bacterial Soft Rot and Blackleg	261
7.1.1	Causal Organism	261
7.1.2	Symptoms.....	261
7.1.2.1	Soft Rot.....	261
7.1.2.2	Blackleg	263
7.1.3	Favorable Conditions of Disease Development.....	263
7.1.4	Disease Cycle	263
7.1.4.1	Soft Rot	263
7.1.4.2	Blackleg	264
7.1.5	Management	264
7.2	Bacterial Wilt of Potato.....	264
7.2.1	Causal Organism.....	264
7.2.2	Symptoms.....	265
7.2.2.1	Aboveground.....	265
7.2.2.2	Belowground	265
7.2.3	Cause and Disease Development.....	266
7.2.4	Favorable Conditions of Disease Development.....	266
7.2.5	Disease Cycle	266
7.2.6	Management	267
7.3	Fusarium Dry Rot.....	267
7.3.1	Causal Organism	267
7.3.2	Symptoms.....	268
7.3.3	Cause and Disease Development.....	268
7.3.4	Favorable Conditions of Disease Development.....	269
7.3.5	Disease Cycle	269
7.3.6	Management	269
7.3.6.1	Cultural Control.....	269
7.3.6.2	Chemical Control.....	269
7.3.6.3	Biological Control.....	269
7.4	Pink Rot of Potato	270
7.4.1	Causal Organism	270
7.4.2	Symptoms.....	270
7.4.3	Cause and Disease Development.....	271
7.4.4	Favorable Conditions of Disease Development.....	271
7.4.5	Disease Cycle	271
7.4.6	Management	272
7.4.6.1	Fungicide Application.....	272
7.5	Bacterial Ring Rot of Potato	273
7.5.1	Causal Organism	273
7.5.2	Symptoms.....	273
7.5.3	Favorable Conditions of Disease Development.....	273
7.5.4	Disease Cycle	274
7.5.5	Management	274

7.6	Sclerotinia Stem Rot of Potato	275
7.6.1	Causal Organism	275
7.6.2	Symptoms.....	275
7.6.3	Favorable Conditions of Disease Development.....	275
7.6.4	Disease Cycle	276
7.6.5	Management	276
7.7	Pink Eye of Potato	276
7.7.1	Causal Organism	277
7.7.2	Symptoms.....	277
7.7.3	Favorable Conditions of Disease Development.....	277
7.7.4	Disease Cycle	278
7.7.5	Management	278
7.8	Black Dot Disease of Potato.....	278
7.8.1	Causal Organism	278
7.8.2	Symptoms.....	278
7.8.3	Cause and Disease Development.....	279
7.8.4	Favorable Conditions of Disease Development.....	279
7.8.5	Disease Cycle	280
7.8.6	Management	280
7.8.6.1	Seed	280
7.8.6.2	Field	280
7.8.6.3	Storage	280
7.9	Common Scab of Potato.....	280
7.9.1	Causal Organism	280
7.9.2	Symptoms.....	281
7.9.3	Cause and Disease Development.....	281
7.9.4	Disease Cycle	281
7.9.5	Management	282
7.10	<i>Dickeya Solani</i> on Potato	282
7.10.1	Causal Organism	282
7.10.2	Symptoms.....	283
7.10.3	Management	283
7.11	Disorders in Potato	283
7.11.1	Brown Center and Hollow Heart.....	283
7.11.1.1	To Reduce Hollow Heart	285
7.11.2	Growth Cracks	285
7.11.3	Potato Greening.....	285
7.12	Early Blight of Potato	286
7.12.1	Causal Organism	286
7.12.2	Symptoms.....	287
7.12.3	Cause and Disease Development.....	287
7.12.4	Favorable Conditions of Disease Development.....	288
7.12.5	Disease Cycle	288
7.12.6	Management	289
7.12.6.1	Cultural Practices	289
7.12.6.2	Chemical Practices	289
7.12.6.3	Resistant Cultivars.....	289
7.13	Late Blight of Potato.....	289
7.13.1	Causal Organism	289
7.13.2	Symptoms.....	290
7.13.3	Cause and Disease Development.....	290

7.13.4	Favorable Conditions of Disease Development.....	290
7.13.5	Disease Cycle	291
7.13.6	Management	291
7.14	Potato Black Ringspot Virus (PBRSV).....	292
7.14.1	Symptoms.....	292
7.14.2	Means of Movement and Transmission.....	292
7.14.3	Prevention and Control.....	292
7.15	Potato Leafroll Virus.....	293
7.15.1	Causal Organism	293
7.15.2	Symptoms.....	293
7.15.3	Means of Movement and Transmission.....	294
7.15.4	Prevention and Control.....	294
7.16	Potato Virus Y	295
7.16.1	Causal Organism	295
7.16.2	Symptoms.....	295
7.16.3	Means of Movement and Transmission.....	295
7.16.4	Prevention and Control.....	296
7.17	Root Knot Nematode on Potato.....	296
7.17.1	Symptoms.....	296
7.17.2	Spread.....	296
7.17.3	Management	297
7.17.4	Root Lesion Nematode	297
7.17.5	Symptoms.....	297
7.17.6	Management	297
7.18	Rhizoctonia Canker.....	297
7.18.1	Causal Organism	298
7.18.2	Symptoms.....	298
7.18.3	Cause and Disease Development.....	299
7.18.4	Favorable Conditions of Disease Development.....	299
7.18.5	Disease Cycle	299
7.18.6	Management	300
7.19	Silver Scurf of Potato	300
7.19.1	Causal Organism	300
7.19.2	Symptoms.....	300
7.19.3	Cause and Disease Development.....	301
7.19.4	Favorable Conditions of Disease Development.....	301
7.19.5	Disease Cycle	301
7.19.6	Management	302
	References	303
Chapter 8	Onion.....	305
8.1	Basal Plate Rot.....	305
8.1.1	Pathogen	305
8.1.2	Symptoms.....	305
8.1.3	Disease Development and Epidemiology.....	305
8.1.4	Control Measures	306
8.2	Fusarium Rot Disease (Yellows)	306
8.2.1	Symptoms	306
8.2.2	Pathogen Detection.....	307
8.2.3	Pathogen Life.....	307
8.2.4	Control Measures	307

8.3	Onion Leaf Rot and Leaf Blight.....	308
8.3.1	Pathogen	308
8.3.2	Symptoms.....	308
8.3.3	Epidemiology	308
8.3.4	Control Measures	310
8.4	Onion Neck Rot.....	313
8.4.1	<i>B. aclada</i> : The pathogen	313
8.4.2	Symptoms.....	313
8.4.3	Control Measures and Disease Management	314
8.5	Onion White Root Rot.....	315
8.5.1	Symptoms.....	315
8.5.2	Disease Cycle	315
8.5.3	Control Measures	316
8.5.3.1	Sanitation	316
8.5.3.2	Cultural Controls	317
8.5.3.3	Sclerotia Germination Stimulants	317
8.5.3.4	Chemical Controls	317
8.5.3.5	Organically Acceptable Practices / Biological Control	317
8.6	Downy Mildew	317
8.6.1	Pathogen	317
8.6.2	Symptoms.....	318
8.6.3	Epidemiology	318
8.6.4	Control and Management of Disease	318
8.6.4.1	Cultural Management	318
8.6.4.2	Chemical Treatment.....	320
8.6.4.3	Disease Forecast	320
8.7	Onion Smut.....	320
8.7.1	Pathogen	320
8.7.2	Symptoms.....	320
8.7.3	Factors Affecting the Disease	321
8.7.4	Control Measures	321
8.8	Pink Root.....	322
8.8.1	Pathogen	322
8.8.2	Disease Development and Symptoms	322
8.8.3	Epidemiology	322
8.8.4	Control Measures	323
8.8.4.1	Crop Rotation	323
8.8.4.2	Resistant Cultivars	323
8.8.4.3	Chemical Methods for Curing.....	323
8.8.4.4	Solarization.....	323
8.8.5	Preventive Measure	323
8.9	Purple Blotch	323
8.9.1	Symptoms.....	323
8.9.2	Epidemiology	324
8.9.3	Epidemic.....	325
8.9.4	Control Measures	325
8.9.4.1	Crop Rotation.....	325
8.9.4.2	Sanitation and Crop Debris	325
8.9.4.3	Plantation and Crop Handling	325
8.9.4.4	Chemical Control.....	325
8.9.4.5	Resistant Cultivars	325
	References	326

Chapter 9	Chili.....	327
9.1	Anthracnose of Chili	327
9.1.1	Causal Organism	327
9.1.2	Symptoms.....	327
9.1.3	Cause and Disease Development.....	328
9.1.4	Favorable Conditions of Disease	328
9.1.5	Disease Cycle	329
9.1.6	Management	329
9.1.6.1	Chemical Control.....	329
9.1.6.2	Biological Control.....	329
9.1.6.3	Cultural Control.....	330
9.2	Bacterial Leaf Spot of Chili	330
9.2.1	Causal Organism	330
9.2.2	Symptoms.....	330
9.2.3	Cause and Disease Development.....	332
9.2.4	Favorable Conditions.....	332
9.2.5	Disease Cycle	332
9.2.6	Management	332
9.2.6.1	Chemical Control.....	332
9.2.6.2	Biological Control.....	333
9.2.6.3	Cultural Control.....	333
9.3	Cercospora Leaf Spot of Chili.....	334
9.3.1	Causal Organism	334
9.3.2	Symptoms.....	334
9.3.3	Cause and Disease Development.....	334
9.3.4	Favorable Conditions.....	334
9.3.5	Management	336
9.3.5.1	Chemical Control.....	336
9.4	Pepper Mottle Mosaic Virus (PMMV)	336
9.4.1	Causal Organism	336
9.4.2	Symptoms.....	336
9.4.3	Cause and Disease Development.....	337
9.4.4	Management	337
9.5	Phytophthora Blight of Pepper/Chili	338
9.5.1	Causal Organism	338
9.5.2	Symptoms.....	338
9.5.3	Cause and Disease Development.....	339
9.5.4	Favorable Conditions.....	339
9.5.5	Disease Cycle	339
9.5.6	Management	340
9.5.6.1	Chemical Control.....	340
9.5.6.2	Biological Control.....	340
9.5.6.3	Cultural Control.....	340
9.6	Powdery Mildew of Chili	340
9.6.1	Causal Organism	341
9.6.2	Symptoms.....	341
9.6.3	Cause and Disease Development.....	342
9.6.4	Favorable Conditions.....	342
9.6.5	Disease Cycle	343
9.6.6	Management	343

9.6.6.1	Chemical Control.....	343
9.6.6.2	Biological Control.....	343
9.7	Verticillium Wilt of Chili	344
9.7.1	Causal Organism	344
9.7.2	Symptoms.....	344
9.7.3	Cause and Disease Development.....	347
9.7.4	Favorable Conditions.....	347
9.7.5	Disease Cycle	347
9.7.6	Management	348
	References	349
Chapter 10	Cucurbits	351
10.1	Anthracnose of Cucurbits.....	351
10.1.1	Causal Organism	351
10.1.2	Symptoms.....	351
10.1.3	Cause and Disease Development.....	352
10.1.4	Favorable Conditions.....	353
10.1.5	Disease Cycle	353
10.1.6	Management	354
10.1.6.1	Chemical Control.....	354
10.1.6.2	Biological Control.....	354
10.1.6.3	Cultural Control.....	354
10.2	Alternaria Leaf Blight (Spot) of Cucurbits.....	355
10.2.1	Causal Organism	355
10.2.2	Symptoms.....	356
10.2.3	Favorable Conditions of Disease Development.....	357
10.2.4	Disease Cycle	357
10.2.5	Management	357
10.2.5.1	Chemical Control.....	358
10.2.5.2	Biological Control.....	359
10.2.5.3	Cultural Control.....	359
10.3	Gummy Stem Blight of Cucurbits	359
10.3.1	Causal Organism	360
10.3.2	Symptoms.....	360
10.3.2.1	Stems.....	360
10.3.2.2	Fruits.....	360
10.3.2.3	Leaves	360
10.3.3	Cause and Disease Development.....	362
10.3.4	Favorable Conditions.....	362
10.3.5	Disease Cycle	362
10.3.6	Management	363
10.3.6.1	Chemical Control.....	363
10.3.6.2	Cultural Control.....	364
10.3.6.3	Fungicides	364
10.4	Angular Leaf Spot of Cucurbits	364
10.4.1	Causal Organism	364
10.4.2	Symptoms.....	366
10.4.3	Cause and Disease Development.....	367
10.4.4	Favorable Conditions.....	367
10.4.5	Disease Cycle	367

10.4.6	Management	368
10.4.6.1	Chemical Control.....	368
10.4.6.2	Biological Control.....	368
10.4.6.3	Cultural Control.....	368
10.5	Cercospora Leaf Spot of Cucurbits	368
10.5.1	Causal Organism	368
10.5.2	Symptoms.....	368
10.5.3	Cause and Disease Development.....	370
10.5.4	Favorable Conditions.....	370
10.5.5	Disease Cycle	370
10.5.6	Management	370
10.5.6.1	Chemical Control.....	370
10.5.6.2	Biological Control.....	371
10.5.6.3	Cultural Control.....	371
10.6	Charcoal Rot of Cucurbits	371
10.6.1	Causal Organism	371
10.6.2	Symptoms.....	371
10.6.3	Cause and Disease Development.....	371
10.6.4	Favorable Conditions of Disease Development.....	373
10.6.5	Management	373
10.7	Choanephora Wet Rot of Cucurbits.....	373
10.7.1	Causal Organism	373
10.7.2	Symptoms.....	374
10.7.3	Cause and Disease Development.....	374
10.7.4	Favorable Conditions.....	374
10.7.5	Disease Cycle	375
10.7.6	Management	375
10.8	Cucumber Mosaic Virus Disease	375
10.8.1	Causal Organism	375
10.8.2	Symptoms.....	375
10.8.3	Cause and Disease Development.....	376
10.8.4	Favorable Conditions.....	376
10.8.5	Disease Cycle	376
10.8.6	Management	377
10.8.6.1	Chemical Control and Biological Control	377
10.8.6.2	Cultural Control.....	377
10.9	Root Knot Nematode of Cucurbits	377
10.9.1	Causal Organism	378
10.9.2	Symptoms.....	378
10.9.3	Cause and Disease Development.....	378
10.9.4	Favorable Conditions.....	379
10.9.5	Disease Cycle	379
10.9.6	Management	381
10.9.6.1	Chemical Control.....	381
10.9.6.2	Biological Control.....	381
10.9.6.3	Cultural Control.....	381
10.10	Downy Mildew of Cucurbits	381
10.10.1	Causal Organism	382
10.10.2	Symptoms.....	382
10.10.3	Cause and Disease Development.....	383
10.10.4	Favorable Conditions	384

10.10.5 Disease Cycle	384
10.10.6 Management	385
10.10.6.1 Chemical Control.....	385
10.10.6.2 Biological Control.....	385
10.10.6.3 Cultural Control.....	386
10.11 Powdery Mildew of Cucurbits.....	386
10.11.1 Causal Organism	387
10.11.2 Symptoms.....	387
10.11.3 Cause and Disease Development.....	387
10.11.4 Favorable Conditions.....	388
10.11.5 Disease Cycle	388
10.11.6 Management	388
10.11.6.1 Chemical Control.....	388
10.11.6.2 Biological Control.....	389
10.11.6.3 Cultural Control.....	389
10.12 Fusarium Wilt Disease of Cucurbits	389
10.12.1 Causal Organism	390
10.12.2 Symptoms.....	390
10.12.3 Cause and Disease Development.....	392
10.12.4 Favorable Conditions.....	393
10.12.5 Disease Cycle	393
10.12.6 Management	393
10.12.6.1 Chemical Control.....	393
10.12.6.2 Biological Control.....	393
10.12.6.3 Cultural Control.....	394
10.13 Pythium Disease of Cucurbits	394
10.13.1 Causal Organism	395
10.13.2 Symptoms.....	395
10.13.2.1 Fruit Rot.....	395
10.13.2.2 Root Rot.....	395
10.13.3 Cause and Disease Development.....	395
10.13.4 Disease Cycle	397
10.13.5 Management	397
10.13.5.1 Chemical Control.....	397
10.13.5.2 Biological Control.....	397
10.13.5.3 Cultural Control.....	397
10.14 Scab or Gummosis of Cucurbits.....	397
10.14.1 Causal Organism	399
10.14.2 Symptoms.....	399
10.14.3 Cause and Disease Development.....	401
10.14.4 Favorable Conditions.....	401
10.14.5 Disease Cycle	401
10.14.6 Management	401
References	405
Chapter 11 Ginger.....	407
11.1 Ginger Wilt	407
11.1.1 Causal Organism	407
11.1.2 Symptoms.....	407
11.1.3 Disease Cycle	408

11.1.4 Cause and Disease Development.....	409
11.1.5 Favorable Conditions of Disease Development.....	411
11.1.6 Management and Control	412
11.1.7 Seed Selection	412
11.2 Bacterial Wilt of Ginger	413
11.2.1 Causal Organism	413
11.2.2 Symptoms.....	414
11.2.3 Pathogen Detection and Disease Diagnosis	414
11.2.3.1 Bioassay	415
11.2.3.2 Immunoassay	415
11.2.3.3 Polymerase Chain Reaction and DNA Test.....	415
11.2.4 Management of the Disease and Control Measures.....	415
11.2.4.1 Chemical Procedures.....	416
11.2.4.2 Crop Rotation and Inter-cropping.....	416
11.2.5 Storage.....	417
11.3 Fungal Soft Rot.....	417
11.3.1 Disease Cycle	418
11.3.2 Disease Management.....	419
11.4 Fusarium Rot Disease (Yellows)	420
11.4.1 Symptoms.....	420
11.4.2 Pathogen detection	421
11.4.3 Pathogen Life.....	421
11.4.4 Control Measures	421
11.5 Phyllosticta Leaf Spot.....	422
11.5.1 The Pathogen.....	422
11.5.2 Symptoms.....	422
11.5.3 Disease Cycle	422
11.5.4 Management	423
References	423
Chapter 12 Maize.....	425
12.1 Anthracnose of Maize	425
12.1.1 Causal Organism	425
12.1.2 Symptoms.....	425
12.1.3 Cause and Disease Development.....	426
12.1.4 Favorable Conditions	426
12.1.5 Disease Cycle	426
12.1.6 Management	427
12.1.6.1 Chemical/Biological Control.....	427
12.1.6.2 Cultural Practices	427
12.2 Common Rust of Corn.....	427
12.2.1 Causal Organism	428
12.2.2 Symptoms of Common Rust	428
12.2.3 Symptoms of Southern Rust.....	428
12.2.4 Cause and Disease Development.....	429
12.2.5 Favorable Conditions.....	429
12.2.6 Disease Cycle	430
12.2.7 Management	430
12.2.7.1 Chemical Control.....	430
12.2.7.2 Cultural Control.....	431

12.3	Common Smut of Corn.....	431
12.3.1	Causal Organism	431
12.3.2	Symptoms.....	433
12.3.3	Cause and Disease Development.....	433
12.3.4	Favorable Conditions.....	434
12.3.5	Disease Cycle	434
12.3.6	Management	435
12.4	Downy Mildew of Corn (Crazy Top of Corn)	436
12.4.1	Causal Organism	436
12.4.2	Symptoms.....	436
12.4.3	Cause and Disease Development.....	437
12.4.4	Favorable Conditions.....	437
12.4.5	Disease Cycle	437
12.4.6	Management	438
12.5	Eyespot Disease of Corn.....	438
12.5.1	Causal Organism	438
12.5.2	Symptoms.....	438
12.5.3	Cause and Disease Development.....	439
12.5.4	Favorable Conditions.....	439
12.5.5	Disease Cycle	439
12.5.6	Management	440
12.5.6.1	Chemical Control.....	440
12.5.6.2	Biological Control.....	440
12.5.6.3	Cultural Control.....	440
12.6	Gray Leaf Spot of Corn	441
12.6.1	Causal Organism	441
12.6.2	Symptoms.....	441
12.6.3	Cause and Disease Development.....	442
12.6.4	Favorable Conditions.....	443
12.6.5	Disease Cycle	443
12.6.6	Management	443
12.6.6.1	Chemical Control.....	443
12.6.6.2	Headline EC.....	444
12.6.6.3	Quilt.....	444
12.6.6.4	Proline 480 SC.....	444
12.6.6.5	Tilt 250 and Bumper 418 EC	445
12.6.6.6	Biological Control.....	445
12.6.6.7	Cultural Control.....	445
12.7	Maize Dwarf Mosaic Virus.....	445
12.7.1	Causal Organism	446
12.7.2	Symptoms.....	446
12.7.3	Cause and Disease Development.....	446
12.7.4	Favorable Conditions.....	447
12.7.5	Disease Cycle	447
12.7.6	Management	447
12.8	Northern Corn Leaf Blight of Maize.....	448
12.8.1	Causal Organism	448
12.8.2	Symptoms.....	448
12.8.3	Cause and Disease Development.....	449
12.8.4	Favorable Conditions.....	449
12.8.5	Disease Cycle	449

12.8.6	Management	450
12.8.6.1	Chemical Control.....	450
12.8.6.2	Biological Control.....	450
12.8.6.3	Cultural Control.....	451
12.9	Southern Corn Leaf Blight	451
12.9.1	Causal Organism	451
12.9.2	Symptoms.....	451
12.9.3	Cause and Disease Development.....	452
12.9.4	Favorable Conditions.....	452
12.9.5	Disease Cycle	452
12.9.6	Management	453
12.10	Stewart's Bacterial Wilt.....	454
12.10.1	Causal Organism	454
12.10.2	Symptoms.....	454
12.10.3	Cause and Disease Development.....	454
12.10.4	Favorable Conditions.....	455
12.10.5	Disease Cycle	456
12.10.6	Management	456
12.10.6.1	Chemical Control.....	456
12.10.6.2	Cultural Control.....	457
12.10.6.3	Biological Control.....	457
	References	457
Chapter 13	Grape	459
13.1	Anthracnose of Grapes	459
13.1.1	Causal Organism	459
13.1.2	Symptoms.....	459
13.1.3	Cause and Disease Development.....	461
13.1.4	Favorable Conditions of Disease Development.....	462
13.1.5	Disease Cycle	462
13.1.6	Management	463
13.1.7	Control.....	463
13.2	Alternaria Rot	464
13.2.1	Causal Organism	464
13.2.2	Taxonomy	464
13.2.3	Symptoms.....	465
13.2.4	Cause and Disease Development.....	465
13.2.5	Favorable Conditions of Disease Development.....	466
13.2.6	Management	466
13.3	Gray Mold (<i>Botrytis Cinerea</i>)	467
13.3.1	Causal Organism	467
13.3.2	Symptoms.....	467
13.3.3	Cause and Disease Development.....	467
13.3.4	Favorable Conditions of Disease Development.....	468
13.3.5	Management	468
13.4	Black Rot (<i>Guignardia bidwellii</i>)	469
13.4.1	Causal Organism	469
13.4.2	Symptoms.....	469
13.4.3	Cause and Disease Development.....	471
13.4.4	Favorable Conditions for the Disease Development.....	472

13.4.5 Management	473
13.5 Armillaria Root Rot in Grapes.....	476
13.5.1 Causal Organism	477
13.5.2 Symptoms.....	477
13.5.3 Cause and Disease Development.....	477
13.5.4 Favorable Conditions.....	478
13.5.5 Disease Cycle	478
13.5.6 Management	478
13.5.6.1 Chemical Control.....	478
13.5.6.2 Biological Control.....	479
13.5.6.3 Cultural Control.....	479
13.6 Downy Mildew on Grapes.....	479
13.6.1 Causal Organism	479
13.6.2 Symptoms.....	479
13.6.3 Cause and Disease Development.....	480
13.6.4 Favorable Conditions of Disease Development.....	481
13.6.5 Management	482
13.7 Powdery Mildew.....	484
13.7.1 Causal Organism	485
13.7.2 Symptoms.....	485
13.7.3 Favorable Conditions of Disease Development.....	487
13.7.4 Management	488
13.7.5 Control.....	488
13.8 Bacterial Blight (Bacterial Nerosis)	490
13.8.1 Causal Organism	490
13.8.2 Symptoms.....	493
13.8.3 Cause and Disease Development.....	494
13.8.4 Favorable Conditions of Disease Development.....	494
13.8.4.1 Dispersal	494
13.8.4.2 Survival.....	494
13.8.5 Disease Cycle	494
13.8.6 Management	495
13.8.6.1 Chemical Control.....	495
13.8.6.2 Cultural Control.....	495
13.9 Crown Gall of Grape	495
13.9.1 Causal Organism	496
13.9.2 Symptoms.....	496
13.9.3 Cause and Disease Development.....	497
13.9.4 Favorable Conditions of Disease Development.....	497
13.9.5 Disease Cycle	498
13.9.6 Management	498
13.9.6.1 Chemical Control.....	498
13.9.6.2 Biological Control.....	498
13.9.6.3 Cultural Control.....	498
13.10 Eutypa dieback	499
13.10.1 Causal Organism	499
13.10.2 Symptoms.....	499
13.10.2.1 Shoot-Leaves Symptom	499
13.10.2.2 Wood Symptom	499
13.10.3 Cause and Disease Development.....	500
13.10.4 Disease Cycle	501

13.10.5 Management	501
13.10.5.1 Chemical Control.....	501
13.10.5.2 Biological Control.....	501
13.10.5.3 Cultural Control.....	502
13.11 Grape Leaf Roll Disease	502
13.11.1 Causal Organism.....	502
13.11.2 Symptoms.....	503
13.11.3 Cause and Disease Development.....	504
13.11.4 Management	504
13.11.4.1 Chemical (Vector) Control	505
13.11.4.2 Biological and Cultural Control.....	505
13.12 Grapevine Fan Leaf Disease	506
13.12.1 Causal Organism.....	506
13.12.2 Symptoms.....	507
13.12.3 Cause and Disease Development.....	507
13.12.3.1 Favorable Conditions	508
13.12.4 Management	509
13.12.4.1 Chemical Control.....	509
13.12.4.2 Cultural Control.....	509
13.13 Pierce's Disease	509
13.13.1 Causal Organism.....	510
13.13.2 Symptoms.....	510
13.13.3 Cause, Favorable Conditions, and Disease Development.....	511
13.13.4 Disease Cycle	513
13.13.5 Management	513
13.13.5.1 Chemical Control.....	514
13.13.5.2 Cultural Control.....	514
13.13.5.3 Vector Management.....	514
13.14 Phomopsis Cane and Leaf Spot.....	515
13.14.1 Causal Organism.....	515
13.14.2 Symptoms.....	515
13.14.2.1 Leaves	516
13.14.2.2 Fruit	516
13.14.2.3 Green Shoots.....	517
13.14.2.4 Canes	517
13.14.3 Cause and Disease Development.....	518
13.14.4 Favorable Conditions.....	518
13.14.5 Disease Cycle	519
13.14.6 Management	519
13.14.6.1 Chemical Control.....	519
13.14.6.2 Cultural Control.....	520
13.15 Rugose Wood of Grapevines	520
13.15.1 Causal Organism.....	521
13.15.2 Symptoms.....	521
13.15.2.1 Wood.....	521
13.15.2.2 Vine Growth	522
13.15.2.3 Foliage	522
13.15.2.4 Fruit	522
13.15.3 Cause and Disease Development.....	522
13.15.3.1 Virus Movement and Disease Development.....	522

13.15.4 Management	523
References	523
Chapter 14 Sugarcane	525
14.1 Red Rot of Sugarcane	525
14.1.1 Causal Organism	525
14.1.2 Symptoms	525
14.1.2.1 Stalk Symptoms	527
14.1.2.2 Leaf Symptoms	529
14.1.3 Cause and Disease Development	529
14.1.4 Favorable Conditions of Disease Development	529
14.1.5 Disease Cycle	530
14.1.6 Management	530
14.1.6.1 Chemical Control	530
14.1.6.2 Biological Control	532
14.1.7 Control	532
14.2 Wilt on Sugarcane	532
14.2.1 Causal Organism	533
14.2.2 Symptoms	533
14.2.3 Cause and Disease Development	533
14.2.4 Favorable Conditions of Disease Development	533
14.2.5 Disease Cycle	536
14.2.6 Management and Control	536
14.3 Smut Disease of Sugarcane	536
14.3.1 Casual Organism	537
14.3.2 Symptoms	537
14.3.3 Cause of Disease and Development	539
14.3.4 Favorable Conditions for Disease Development	539
14.3.5 Disease Cycle	539
14.3.6 Management	540
14.3.7 Control	542
14.4 Sugarcane Grassy Shoot Disease (SCGS)	543
14.4.1 Causal Organism	543
14.4.2 Symptoms	544
14.4.3 Cause of Disease and Development	544
14.4.4 Favorable Conditions for Disease Development	544
14.4.5 Disease Cycle	545
14.4.6 Control	545
14.5 Yellow Leaf Virus of Sugarcane	545
14.5.1 Casual Organism	545
14.5.2 Symptoms	546
14.5.3 Cause of Disease and Development	546
14.5.4 Favorable Conditions for Disease Development	546
14.5.5 Disease Cycle	546
14.5.6 Management and Control	548
14.6 Banded Disease	549
14.6.1 Symptoms	549
14.6.2 Control Measures	549
14.7 Gummosis or Gummimg Disease	550
14.7.1 Symptoms	550

14.7.2 Disease Cycle	551
14.7.3 Control.....	551
14.8 Root Knot Nematode	551
14.8.1 Symptoms.....	552
14.8.2 Disease Cycle	552
14.8.3 Control.....	552
14.9 Eye Spot in Sugarcane.....	553
14.9.1 Symptoms	553
14.10 Red Stripe	553
14.10.1 Symptoms.....	554
14.10.2 Disease Cycle	554
14.10.3 Control.....	554
References	555
Chapter 15 Guava.....	557
15.1 Anthracnose of Guava	557
15.1.1 Causal Organism	557
15.1.2 Symptoms.....	557
15.1.3 Cause and Disease Development.....	558
15.1.4 Favorable Conditions of Disease Development.....	560
15.1.5 Disease Cycle	560
15.1.6 Management	560
15.1.7 Control.....	560
15.2 Canker [Pestalotia Psidii Pat.]	560
15.2.1 Symptoms.....	560
15.2.2 Mode of Spread and Reason for Severity.....	561
15.2.3 Management	561
15.3 Algal leaf and Fruit Spot	561
15.3.1 Symptoms.....	561
15.3.2 Management	561
15.4 Cercospora Leaf Spot	562
15.4.1 Symptoms.....	562
15.4.2 Management	562
15.5 Sooty Mold	562
15.5.1 Symptoms	562
15.5.2 Management	563
15.6 Damping Off of Seedlings	563
15.6.1 Symptoms.....	563
15.6.2 Management	563
15.7 Phytophthora Fruit Rot	564
15.7.1 Symptoms.....	564
15.7.2 Mode of Spread	564
15.7.3 Reason for Severity	565
15.7.4 Management	565
15.8 Stylar End Rot	565
15.8.1 Symptoms.....	565
15.8.2 Management	566
15.9 Soft Watery Rot	566
15.9.1 Symptoms	566
15.9.2 Management	566

15.10 Botryosphaeria Rot	567
15.10.1 Symptoms	567
15.10.2 Management	567
15.11 Hyaloderma Leaf Spot.....	567
15.11.1 Symptoms.....	567
15.11.2 Management	568
15.12 Parasites.....	568
15.12.1 Symptoms.....	568
15.12.2 Management	569
References	569
Index	571



Taylor & Francis
Taylor & Francis Group
<http://taylorandfrancis.com>

Preface

Plant diseases are becoming major constraints in agricultural production with increases in the number of hybrid and genetically modified cultivators who are focused on increasing yield. Climatic changes are also creating favorable conditions for most of the diseases. At the same time, there are many reports of new pathogens that can cause considerable damage to agricultural crop. Disease identification plays a key role in overall diseases management. One disease may show different symptoms; on the contrary, different diseases may show same kind of symptoms. Scope and depth of our knowledge of plant and crop physiology are rapidly expanding, and plant physiologists are continuously making new discoveries.

The *Handbook of Plant Disease Identification and Management* will be a plant pathology and diseases related work, which will center around the topic of crop diseases. This handbook will provide fundamental knowledge about how to identify the disease, how to track disease development, and IPM (Integrated pest management) by using diverse ways like chemical, biological, and physical methods. It is a unique, comprehensive, and complete collection of the topics in plant pathology to serve as an all-inclusive resource and up-to-date reference to effectively cover the information relevant to plant diseases.

The sociology of crop pathology is discussed in this handbook as well as a review of a great variety of techniques for the diagnosis of crop disease, losses due to crop diseases, and theory behind the disease management. It also explores topics on how society is constraining the possibilities for management; management of diseases through changing the environment; biological control of crop diseases; weed management through pathogens; and the epidemiologic and genetic concepts of managing host genes.

Subsequent chapters present the management of crop disease with chemicals and some examples of diseases that benefit man and even a few that benefit plants. This book also describes the organization and operation of society-supported disease management activities, as well as important advisory services provided by the industry.

This handbook will act as a complete guide for academic researchers, students, and growers to understand the basics of plant disease identification. In this handbook I have tried to explain in layman's terms the disease cycle with favorable conditions for disease development that will help growers to manage the diseases and help researchers in their future study. This work is intended for an individual conducting research in plant pathology to broaden his views, stimulate his thinking, and help to synthesize ideas. This book will be very beneficial for industrial professionals as well.

Balaji Aglave



Taylor & Francis
Taylor & Francis Group
<http://taylorandfrancis.com>

About the Author

Balaji A. Aglave was born in Yermala, Maharashtra, India. He obtained his Bachelor of Science degree in Agricultural Sciences, 2002, and Master of Science degree in Agricultural Biotechnology, 2004 from Marathwada Agricultural University, Parbhani Maharashtra (India). His Ph.D. was in Biotechnology with specialization in Plant Pathology, and he obtained the degree from Government Institute of Science, Aurangabad, Maharashtra (India) in 2009. After completing his master's education, Dr. Aglave worked for National Research Center for Citrus, Nagpur M.S. India as a Senior Research Fellow. As a Senior Research Fellow, his areas of study were molecular and serological diagnosis of citrus viruses, diagnosis and characterization of major citrus viruses, development of non-radioactive probes, and diagnostic kits for plant virus detection. In 2005, he was hired as the Head of the Department of Biotechnology by H.P.T. Arts and R.Y.K. Science College, Nashik, M.S. India (University of Pune). He worked as Head of the Department for four years after which, in 2010, he was hired by Florida Ag Research as a Scientist and Lab Director for the Division of Plant Pathology and Nematology at East Coast research station of Pacific Ag Research, San Luis Obispo, California. In addition to his Ph.D., he mastered the area of plant nematology by completing *Plant Parasitic Nematode Identification* course at Clemson University. Dr. Aglave is an expert in plant diseases and common plant parasites, and his insights in these topics are invaluable to the field. Presently, his work as Scientist and Lab Director aims at developing innovative technologies to improve crop production and environmental quality, testing product effectiveness against nematodes and diseases, studying crop/fruit rot and decay, soil fumigation studies, etc.

Dr. Aglave has published two books, viz. *Biotechnology Review*, A&A Publisher, Tampa, Florida, in 2012 and *Techniques in Chemistry, biophysics and Instrumentation*, Shanti Prakashan, Delhi, India, in 2011. From 2012, he has been working as an active member of various well reputed committees like the Industry Committee, Nematology Committee, and Soil Microbiology and Root Disease committee of American Phytopathology Society. He has acted as the Chairman of Board of studies in Biotechnology subcommittee in Applied Biotechnology, University of Pune, India and Chairman of Board of studies in Microbiology subcommittee in Applied Wine Technology, University of Pune, India. He is currently working as an Editorial Board Member of Advances in Plants and Agriculture Research, African Journal of Food Science, and as a Reviewer for several journals in Agriculture sciences. He has 20 years of academic, research, and industry experience in the field of plant pathology and nematology.

From 2008 to present, Dr. Aglave has conducted multiple Conferences and Workshops covering diverse areas of biotechnology like techniques in molecular biology, techniques in genetic engineering, trends in biotechnology, etc. He delivered more than 50 seminars and plenary lectures in various scientific meetings and at various institutes in India and the United States. His research work is also published in over 100 journal research articles, review articles, and abstracts. Also, he holds numerous internationally published research abstracts and technical summaries completed on topics such as disease and insect management. Currently, Dr. Aglave is continuing his research work on biotechnology at Florida Ag Research, Thonotosassa, Florida.



Taylor & Francis
Taylor & Francis Group
<http://taylorandfrancis.com>

1 Strawberry

Strawberry, *Fragaria × ananassa* (Weston) Duchesne ex Rozier is a small fruit, grown throughout the world. It has a peculiar red color with a unique shape and flavor. Different berry plant cultivars or cultivated varieties have been developed for different regions and climates throughout the world. The characteristics that distinguish varieties may include fruit ripening time frame, plant disease resistance, cold tolerance, and specific berry traits such as size, shape, firmness, and flavor. Strawberries are a high-value crop in the United States, which is the world's largest producer of strawberries, accounting for nearly 1/3 of the world's total production. Other major strawberry producing countries of the world are South Korea, Japan, Spain, Poland, and the Russian Federation.

In Brittany, France, during the late eighteenth century, the first garden strawberry was grown. Prior to this, wild strawberries and cultivated selections from wild strawberry species were the common sources of the fruit (Figure 1.1).

Strawberry plants can be affected by many diseases. For example, the leaves of strawberry may be infected by powdery mildew, leaf spot caused by the fungus *Sphaerella fragariae*, leaf blight caused by the fungus *Phomopsis obscurans*, and by a variety of slime molds. The crown and roots may fall victim to black root rot, red stele, verticillium wilt, and nematodes. The fruits are subject to damage from *Rhizopus* rot, gray mold, and leather rot. Thus, it is crucial to have different preventive methods and ways to manage strawberry diseases. The methods discussed in this chapter are very useful for strawberry cultivation and disease prevention.

1.1 ANTHRACNOSE OF STRAWBERRY

Anthracnose is the term used to identify strawberry diseases caused by the fungus *Colletotrichum*. Anthracnose is an important disease of strawberry that can affect foliage, runners, crowns, and fruit. The disease is caused by several species of fungi in the genus *Colletotrichum*: *C. acutatum*, *C. fragariae*, and *C. gloeosporioides*. They all cause similar or nearly identical symptoms on the strawberry plant. The 2 most destructive forms of the disease are crown rot, usually associated with *C. fragariae*, and fruit rot, usually associated with *C. acutatum*.

Historically, anthracnose has generally been restricted to the southern United States and was not common in the northern United States. It has generally been considered to be a “warm weather” or “southern disease” of strawberry. Epidemics of anthracnose fruit rot caused by *C. acutatum* have occurred in Ohio, but the crown rot phase has been observed only a few times in the mid-1980s.¹

Over the past few years, the incidence of anthracnose fruit rot in northern production areas has increased, and there is a concern about the potential impact of this disease in northern, perennial-production systems. Although the disease occurs sporadically and is not common in most plantings in Ohio, when it does occur, it can be devastating, resulting in 100% loss of fruit.

1.1.1 CAUSAL ORGANISM

Anthracnose is caused by distinct species of *Colletotrichum*, mentioned in table below.

Species	Associated Disease Phase	Economic Importance
<i>C. acutatum</i>	Fruit rot	High
<i>C. gloeosporioides</i>	Crown rot	Low to moderate
<i>C. fragariae</i>	Crown rot	Low



FIGURE 1.1 Strawberry fruit.

1.1.2 SYMPTOMS

1.1.2.1 Fruit Rot

Anthracnose fruit rot is caused by *C. acutatum*. It can infect green fruit but is found most often on ripe fruit. Round, firm, sunken spots develop on ripening fruit (Figure 1.2). Spots may range from tan to dark brown. Under rainy or humid conditions, masses of fungal spores develop around the center of spots in a cream to salmon-colored slimy matrix (Figure 1.3). Spots often enlarge until the entire fruit is affected. Diseased fruit frequently become mummified.

1.1.2.2 Crown Rot

Crown Rot is caused by *C. gloeosporioides* and *C. fragariae*. The first visible symptoms of crown rot are sudden leaf wilting and plant death. Crown infections are initiated by spores splashing or washing into central buds from leaves or petioles or by the fungus growing directly into crown tissue.

When crowns of wilted plants are split lengthwise, reddish brown streaking/marbling is visible (Figure 1.4). Although strawberry crowns show discoloration regardless of the cause of death, the reddish marbling pattern is most characteristic of anthracnose. Anthracnose crown rot is active during warm weather but becomes dormant during colder months. However, disease progression resumes when the soil warms in spring. Other symptoms like spots on petioles and stolons and leaf spot are also observed.

1.1.2.3 Petioles and Stolons

Small, dark lesions (dead spots) can appear on stolons and petioles anytime during warm weather. Lesions gradually become black, dry, and sunken. When a lesion girdles a stolon, the unrooted daughter plant beyond the lesion dies. Similarly, a lesion on the petiole often results in death of the attached leaf. Anthracnose symptoms on petioles and stolons are often confused with diseases caused by *Rhizoctonia* and various other leaf spot fungi.



FIGURE 1.2 Sunken spots on fruit due to anthracnose.



FIGURE 1.3 Masses of fungal spores develop in decayed areas on infected fruit.

1.1.2.4 Leaf Spot

Small, round, black to gray spots can appear on expanding leaflets even before petiole or stolon symptoms are noticed. Spores produced in these lesions can wash down into crowns and initiate crown rot.

1.1.3 CAUSE AND DISEASE DEVELOPMENT

Strawberry anthracnose is caused by at least two species of the fungus *Colletotrichum*. *C. fragariae* is primarily a crown rotting fungus, and *C. acutatum* primarily rots fruit. *C. acutatum* is the most common species causing fruit rot in Ohio. The disease is probably introduced into new plantings on infected plants. Recent research indicates that the fungus can grow and produce spores on the surface of apparently healthy leaves. These fungi can persist in infected plants as dormant spores or other fungal structures.

1.1.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

During warm and rainy or humid weather, the fungi become active and rapidly initiate disease development. Once the disease is established in the field, the fungus can overwinter on infected plants and plant debris, such as old dead leaves and mummified fruit. Spore production, spore germination, and infection of strawberry fruits are favored by warm, humid weather and rainfall. In spring and early summer, spores are produced in abundance on previously infected plant debris.



FIGURE 1.4 Reddish-brown discoloration of strawberry crown.

The spores are spread by splashing rain, wind-driven rain, and by people or equipment moving through the field. They are not airborne, so they do not spread over long distances in the wind. Spores require free water on the plant surface to germinate and infect.

The optimum temperature for infection on both immature and mature fruit is between 25°C and 30°C. Under favorable conditions, the fungus produces secondary spores on infected fruit. These spores are spread by rain and result in new infections throughout the growing season. Disease development can occur very rapidly. Up to 90% of the fruit can be infected within a week or less. Both immature and mature fruit are susceptible to infection; however, the disease is most common on ripening or matured fruit.

Short distance disease spread can occur in the field via

- Rain splash
- Overhead irrigation water
- Movement of contaminated equipment

Long distance spread is accomplished by

- Movement of strawberry transplants from the nursery to the grower

1.1.5 DISEASE CYCLE

Infected transplants and soil from infected transplants appear to be the primary source of inoculum in most instances, especially in annual production systems. This may be especially true for *C. fragariae*, which has a limited host range and does not survive in soil over the summer. In perennial systems, the fungi may overseason in infected plants and debris, providing inoculum for the following fruiting season. Spores (conidia) may be dispersed in the field by wind-driven rain, splashing water, insects, movement of workers, equipment, or animals. Disease development and spread are minimal in most cases under cool, dry conditions. Crown infections often occur in the nursery but do not appear until after planting. The fungus continues to develop in newly planted nursery infected plants, which may suddenly die during warm weather in the fall or early spring of the following year (Figure 1.5).

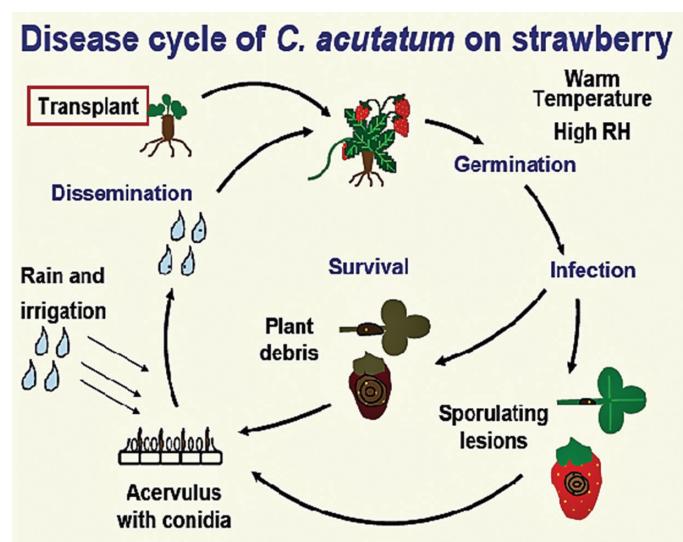


FIGURE 1.5 Disease cycle of *C. acutatum* on strawberry.

1.1.6 MANAGEMENT

Diseases can be managed using the following strategies:

- Chemical control
- Biological control
- Cultural control
- Integrated pest management

1.1.6.1 Chemical Control

Anthracnose fruit rot is a common problem in many areas, and its occurrence is increasing across the Midwest United States. The disease is very important in plasticulture systems. Once anthracnose fruit rot is established in a planting, it is difficult to control and can be very severe, resulting in complete loss of the crop. Captan and Thiram are protectant fungicides that have some activity against anthracnose. If used in a protectant program, they will provide some level of control. Abound, Cabrio, and Pristine are strobilurin fungicides and are labeled for control of anthracnose on strawberry. They have good activity against anthracnose on strawberry of all currently registered fungicides. For purposes of fungicide resistance management and increased efficacy, Abound, Cabrio, and Pristine should be used in rotation with or in combination with Captan or Thiram. Abound, Cabrio, and Pristine are the same class of chemistry so they should not be alternated with each other as a fungicide-resistance strategy. The label states that no more than two applications of one of these fungicides can be made without switching to a fungicide with a different mode of action. Switch has also been reported to have moderate to good activity against anthracnose fruit rot. Therefore, Switch may be used in alternation with Abound, Cabrio, or Pristine for anthracnose control and fungicide resistance management.²

1.1.6.2 Biological Control

Trichoderma isolates are known for their ability to control plant pathogens. It has been shown that various isolates of *Trichoderma*, including *T. harzianum* isolate T-39 from the commercial biological control product TRICHODEX, were effective in controlling anthracnose (*C. acutatum*) in strawberry, under controlled and greenhouse conditions. Three selected *Trichoderma* strains, namely T-39, T-161, and T-166, were evaluated in large-scale experiments using different timing application and dosage rates for the reduction of strawberry anthracnose. All possible combinations of single, double, or triple mixtures of *Trichoderma* strains, applied at 0.4% and 0.8% concentrations, and at seven- or ten-day intervals, resulted in reduction of anthracnose severity; the higher concentration (0.8%) was superior in control whether used with single isolates or because of combined application of two isolates, each at 0.4%. Isolates T-39 applied at 0.4% at two-day intervals, T-166 at 0.4%, or T-161 combined with T-39 at 0.4% were as effective as the chemical fungicide fenhexamid.

1.1.6.3 Cultural Control

Using drip irrigation and clean planting stock is important components of managing this disease. Thoroughly washing all soil from plants before planting will reduce disease in crowns and fruit. It may be worthwhile to dip trays of long-term cold storage (-2°C) transplants into a hot water bath for seven minutes right before planting to reduce occurrence of this disease. Prepare plants for this treatment by thoroughly washing them to remove all dirt; then place them in a circulating water bath that is held at a constant temperature of 49°C. Afterward, submerge them in very cold water and then plant them as soon as possible. (This treatment is not recommended for fresh-dug transplants that have only been stored at 0.5°C.)

Clean field equipment before using it to ensure that contaminated soil and plant parts are not transported into a field or from an infested part of the field to a non-infested section. Crop rotation with a non-host crop can also help in reducing levels of this pathogen in the soil. Also important is good weed management in and around the field to destroy any weeds that may harbor the pathogen.

Recent research has demonstrated the importance of removing the weeds from the fields after they are destroyed because the pathogen can still produce spores even though the weeds are dead.

The following strategies can be followed:

1.1.6.3.1 Use Disease-Free Planting Material

The disease is introduced to the field with infected plant material. The best way to avoid the disease is to begin with disease-free planting material. Although there are no nurseries that can certify plants to be free of fungal and bacterial plant pathogens, inspection of plants for the disease before planting is recommended.

1.1.6.3.2 Proper Irrigation

If the field was previously infected, or the disease is present in the field, minimize the amount of overhead irrigation used. The fungus is spread by splashing water. Avoid the use of overhead irrigation and use drip irrigation if possible.

1.1.6.3.3 Mulching

Plastic mulch increases the level of splash-dispersal of the pathogen. Mulching with straw is recommended in perennial matted row plantings to reduce water splash and disease spread.

1.1.6.3.4 Remove Infected Plant Parts

Infected plant parts serve as a source of inoculum for the disease. Remove as much old, infected plant debris as possible. Try to remove infected berries from the planting during harvest.

Table 1.1 lists materials in order of usefulness in an integrated pest management (IPM) Program, considering efficacy. Also, consider the general properties of the fungicide as well as information relating to environmental impact. Not all registered pesticides are listed. Always read the label of the product being used (Table 1.2).

1.2 POWDERY MILDEW OF STRAWBERRY

Powdery mildew is considered a moderate disease that can affect fruit, leaves, and flowers. This disease produces white patches of web-like growth that develop on both the lower and upper leaf surface. The edges of the leaves may curl upward. Immature fruit may fail to ripen, become hard, crack, and turn a reddish color with raised seeds. Powdery mildew is favored by warm, dry conditions followed by moisture on leaves from overnight dew or rainfall. Spores can be spread by wind and can overwinter in trash from the previous and current crops. The disease affects all cultivated strawberries worldwide. No variety is resistant, but each differs in susceptibility.

1.2.1 CAUSAL ORGANISM

Powdery mildew is mostly caused by the fungi mentioned as follows:

Species	Associated Disease Phase	Economic Importance
<i>Podosphaera aphanis</i> (Previously known as <i>Sphaerotheca macularis</i>)	Leaves, fruits, and flowers	High

1.2.2 SYMPTOMS

P. aphanis infects leaves, flowers, and fruit. Early foliar infections are characterized by small white patches of fungus growing on the lower leaf surface. On susceptible cultivars, dense mycelia growth

TABLE 1.1
Materials in IPM Program

Common Name (Trade Name)	Amount/Acre ^a	R.E.I. ^b (Hours)	P.H.I. ^b (Days)
METHYL BROMIDE/CHLOROPICRIN	300–400 lb	48	0
<i>Sequential application of:</i>			
1,3 - DICHLOROPROPENE/CHLOROPICRIN (Telone C5) OR.....	9–12 gal (shank)	5 days	0
1,3 - DICHLOROPROPENE/CHLOROPICRIN (InLine) OR.....	28–33 gal (drip)	5 days	0
CHLOROPICRIN (MetaPicrin) (Tri-Clor)	15–30 gal (shank) 15–21.85 gal (drip)	48 48	0 0
<i>Followed 5–7 days later by:</i>			
METAM SODIUM (Vapam HL, Sectagon 42) OR.....	37.5–75 gal 30–60 gal	48 48	0 0
METAM POTASSIUM (K-Pam HL)			
<i>AT PLANTING:</i> AZOXYSTROBIN (Abound)	5–8 fl oz/100 gal	4	0
<i>FOLIAR FUNGICIDES:</i> CYPRODINIL/FLUDIOXONIL (Switch) 62.5WG	11–14 oz	12	0
CAPTAN 50WP	4 lb	24	0
AZOXYSTROBIN (Abound)	6.2–15.4 fl oz	4	0

^a Apply all materials in 200-gal water/acre to ensure adequate coverage.

^b Restricted entry interval (R.E.I.) is the number of hours (unless otherwise noted) from treatment until the treated area can be safely entered without protective clothing. Preharvest interval (P.H.I.) is the number of days from treatment to harvest. In some cases, the REI exceeds the PHI. The longer of two intervals is the minimum time that must elapse before harvest.

and numerous chains of conidia give these patches a powdery appearance (Figure 1.6). Under favorable conditions, the patches expand and coalesce until the entire lower surface of the leaf is covered (Figure 1.7). In some strawberry cultivars, relatively little mycelium is produced, making it difficult to see the white patches. Instead, irregular yellow or reddish-brown spots develop on colonized areas on the lower leaf surface, and eventually break through to the upper surface (Figure 1.8). The edges of heavily infected leaves curl upward (Figure 1.9). At times, dark round structures (cleistothecia) are produced in the mycelia on the undersides of leaves. Cleistothecia are initially white but turn black as they mature. The fungus also infects flowers, which may produce aborted or malformed fruit. In addition, *P. aphanis* colonizes older fruit producing a fuzzy mycelial growth on the seeds (Figure 1.10). Both types of infection may reduce fruit quality and marketable yields.

1.2.3 CAUSE AND DISEASE DEVELOPMENT

P. aphanis is an obligate parasite that only infects living tissue of wild or cultivated strawberry. The fungus readily infects living, green leaves in the nursery. Thus, infected transplants are normally the primary source of inoculum for fruiting fields in Florida. When conditions are favorable, conidia produced on infected plants are wind dispersed.

1.2.4 FAVORABLE CONDITIONS

Development and spread of powdery mildew are favored by moderate to high humidity and temperatures between 15.5°C and 27°C. Rain, dew, and overhead irrigation inhibit the fungus. Because

TABLE 1.2**Fungicides Registered for Control of Powdery Mildew of Strawberries in Florida**

Product Name (Active Ingredient)	Fungicide Group	Maximum Rate Per Acre Per: Application Season	Min. Days To Harvest	Remarks
Abound (azoxystrobin)	11	15.4 fl oz. 1.92 qt	0	Do not make more than 2 consecutive appl. and no more than 4 appl./crop year. See label for instructions on dipping transplants.
Bumper 41.8 EC (propiconazole)	3	4 fl oz. 16 fl. oz.	0	Do not make more than 2 consecutive applications.
Cabrio EG (pyraclostrobin)	11	14 fl. oz. 70 fl. oz.	0	Do not make more than 2 consecutive applications and no more than 5 appl./crop year.
Nova 40 W (myclobutanil)	3	5 oz. 30 oz.	0	Do not plant rotational crops until 30 days after last application.
Orbit (propiconazole) (potassium bicarbonate) many brands ^a	3 NC	4 fl.oz. varies	0 1	Do not make more than 2 consecutive applications. Do not mix with highly acidic products.
Pristine (pyraclostrobin + boscalid)	11+7	23 oz. 115 oz.	0	Do not make more than 2 consecutive appl. and no more than 5 appl./ crop.
Procure 50WS (triflumizole)	3	8 oz. 32 oz.	1	Do not plant leafy vegetables within 30 days or root vegetables within 60 days or rotational crops not on label for one year after application.
Quintec (quinoxyfen)	13	6 fl. oz. 24 fl. oz.	1	Do not make more than 2 consecutive applications or more than 4 applications per crop. Do not plant crops not on label for 30 days after application.
Rally 40W (myclobutanil)	3	5 oz. 30 oz.	0	Do not plant rotational crops until 30 days after last application.
Sonoma 40 WSP (myclobutanil) (sulfur) many brands ^b	3 M1 or M9	5 oz. varies	0 1	Do not plant rotational crops until 30 days after last application. Do not use when temperatures exceed 27°C to 30°C.
Switch 62.5 WG (cyprodinil + fludioxonil)	9+12	14 oz. 56 oz.	0	Do not make more than 2 consecutive applications. Do not plant crops not on the label for 30 days after last application.
T-Methyl 70 W WSB (thiophanate-methyl)	1	1 lb. 4 lb.	1	Fungicides from different chemical groups should be used in spray program for disease resistance management.
Topsin 4.5 FL (thiophanate-methyl)	1	20 fl. oz. 80 fl. oz.	1	Fungicides from different chemical groups should be used in spray program for disease resistance management.

(Continued)

TABLE 1.2 (CONTINUED)**Fungicides Registered for Control of Powdery Mildew of Strawberries in Florida**

Product Name (Active Ingredient)	Fungicide Group	Maximum Rate Per Acre Per: Application Season	Min. Days To Harvest	Remarks
Topsin M 70 WP ^a	1	1 lb.	4 lb.	1
Topsin M WSB ^b (thiophanate-methyl)				Fungicides from different chemical groups should be used in spray program for disease resistance management.

^a e.g. Kaligreen, Armicarb 100, Milstop

^b e.g. Micro Sulf., Sulfur 90W, Super-Six, Microthiol Disperss, Wettable Sulfur, Kumulus DF, Dusting Sulfur-IAP, Thioperse 80%, Yellow Jacket Dusting Sulfur, Yellow Jacket Wettable sulfur.

^c Fungicide group (FRAC Code): Numbers (1-37) and letters (M) are used to distinguish the fungicide mode of action groups. All fungicides within the same group (with same number or letter) indicate same active ingredient or similar mode of action. This information must be considered for fungicide resistance management decisions. M = Multi site inhibitors, fungicide resistance risk is low; NC = not classified.

Source: <http://www.frac.info/> (FRAC = Fungicide Resistance Action Committee).

dry conditions and high humidity are common in greenhouses and plastic tunnels, powdery mildew is typically more severe in protected culture. In open fields in central Florida, the disease is typically most severe in November and December and usually subsides in January and early February but may reappear in late February and March.

1.2.5 DISEASE CYCLE

Little is known about either the life cycle of the pathogen or the disease cycle in Powdery Mildew. Disease may occur in the fall, allowing the pathogen to overwinter on strawberry plants in a particular field. Mycelium from the previous fall infection apparently initiates new disease in the spring.

1.2.6 MANAGEMENT

The disease can be managed using the following strategies:

- Chemical control
- Biological control
- Cultural control
- Integrated pest management



FIGURE 1.6 Mycelia of *S. macularis* on strawberry leaf surface.



FIGURE 1.7 Lower leaf surface of strawberry covered with powdery mildew.



FIGURE 1.8 Reddish-brown spot reaction caused by *S. macularis*.



FIGURE 1.9 Curling leaves on severely infected plants.



FIGURE 1.10 *S. macularis* on seeds.

1.2.6.1 Chemical Control

Fungicides should be applied at the first sign of disease to control powdery mildew on susceptible cultivars. This is especially important when using protectant fungicides such as elemental sulfur. Systemic fungicides have some limited curative action. These include Rally, whose active ingredient is myclobutanil, and which was formerly named Nova. Sonoma is a competing brand that also contains myclobutanil. Fungicides in the same chemical class as Rally and Sonoma include Procure, Bumper, and Orbit. These products are treated as a group since they belong to the same fungicide class and have similar properties. All share a common, single mode of action and, for this reason, should be rotated with other fungicides with different properties to avoid the development of resistance. Quintec is a recently introduced and effective fungicide with a different mode of action than other powdery mildew products. Other rotational options include the benzimidazole fungicide Topsin M and the strobilurin fungicides Abound, Cabrio, and Pristine, but caution should be taken to not exceed four applications of these products per season. In addition, powdery mildew was recently added to the label of Switch. Controlling foliar infections helps to prevent fruit infections.

1.2.6.2 Biological Control

- Serenade MAX (*Bacillus subtilis* strain QST 713) at 1–3 lb/A is registered for suppression only. As such it is not recommended for use in the Pacific Northwest. Four-hour re-entry.
- Sonata (*Bacillus pumilis* strain QST 2808) at 2–4 quarts/A is registered for suppression only. As such it is not recommended for use in the PNW. May be applied up to and including the day of harvest. Four-hour re-entry.

1.2.6.3 Cultural Control

Avoid overhead irrigation and excess use of nitrogen and use resistant cultivars where practical. Destroying old leaves by renovating plants after harvest may help reduce inoculums. Plant resistant cultivars.

The following materials are listed in order of usefulness in an IPM Program, considering efficacy. Also, consider the general properties of the fungicide as well as information relating to environmental impact. Not all registered pesticides are listed. Always read the label of the product being used (Table 1.3).

1.3 LEAF SCORCH OF STRAWBERRY

Leaf scorch is caused by the fungus *Diplocarpon earlianum*. The leaf scorch fungus can infect leaves, petioles, runners, fruit stalks, and caps of strawberry plants. Leaf scorch is common on older leaves

TABLE 1.3**Fungicides Registered for Control of *Botrytis* Fruit Rot of Strawberries in Florida^a**

Product Name (Active Ingredient)	Fungicide Group	Maximum Rate Per Acre Per Application Season	Min. Days to Harvest	Remarks
Abound (azoxystrobin)	11	15.4 fl oz	1.92 qt	0 For suppression of <i>Botrytis</i> on the foliage. Do not make more than 2 sequential applications of Group 11 fungicides and no more than 4 applications of Group 11 fungicides per crop year.
Cabrio EG (pyraclostrobin)	11	14 fl oz	70 fl oz	0 For suppression of <i>Botrytis</i> on the foliage. Do not make more than 2 sequential applications of Group 11 fungicides and no more than 5 applications of Group 11 fungicides per crop year.
Captan 80 WDG (captan)	M4	3.75 lb	30 lb	1 Rate per treated acre.
Captec 4L (captan)	M4	3 qt	24 qt	1 Rate per treated acre.
Captevate 68 WDG (captan + fenhexamid)	M4 + 17	5.25 lb	21 lb	0 Do not make more than 2 consecutive applications of Group 17 fungicides and no more than 4 applications of Group 17 fungicides per crop year.
Elevate 50 WDG (fenhexamid)	17	1.5 lb	6 lb	0 Do not make more than 2 consecutive applications of Group 17 fungicides and no more than 4 applications of Group 17 fungicides per crop year.
Iprodione 4L AG (iprodione)	3	2 pt	2 pt	N/A Do not make more than 1 application per season. Do not apply after first fruiting flower.
Pristine (pyraclostrobin + boscalid)	11 + 7	23 oz	115 oz	0 Do not make more than 2 sequential applications of Group 11 fungicides and no more than 5 applications of Group 11 fungicides per crop year.
Rovral 4 Flowable (iprodione)	2	2 pt	2 pt	N/A Do not make more than 1 application per season. Do not apply after bloom initiation.
Scala SC (pyrimethanil)	9	18 fl. oz	54 fl. oz	1 Do not make more than 2 consecutive applications of Group 9 fungicides. Do not use more than 2 of 6 applications of Group 9 fungicides in any one season.
Serenade ASO	44	6 qt.	-	0 For improved performance, use in a tank mix or rotational program with other registered fungicide.
Serenade Max	44	3 lb.	-	0 For improved performance, use in a tank mix or rotational program with other registered fungicide.
Switch 62.5 WG (cyprodinil + fludioxonil)	9 + 12	14 oz	56 oz	0 Do not make more than 2 consecutive applications. Do not plant crops not on the label for 30 days after last application.

(Continued)

TABLE 1.3 (CONTINUED)**Fungicides Registered for Control of *Botrytis* Fruit Rot of Strawberries in Florida^a**

Product Name (Active Ingredient)	Fungicide Group	Maximum Rate Per Acre Per Application Season	Min. Days to Harvest	Remarks
Thiram 65 WSB (thiram)	M2	5 lb	25 lb	3
Thiophanate-methyl 85 WDG (thiophanate-methyl)	1	0.8 lb	3.2 lb	1 Should always be tank-mixed or alternated with a product of a different fungicide group.
T-Methyl 70 W WSB (thiophanate-methyl)	1	1 lb	4 lb	1 Should always be tank-mixed or alternated with a product of a different fungicide group.
Topsin4.5FL(thiophanate-methyl)	1	20 fl. oz	80 fl. oz	1 Should always be tank-mixed or alternated with a product of a different fungicide group.
Topsin M 70 WP ^b Topsin M WSB ^b (thiophanate-methyl)	1	1 lb	4 lb	1 Should always be tank-mixed or alternated with a product of a different fungicide group.

Consult the product label for specific use requirements and restrictions.

^a Recommendations given in this fact sheet are based on experimentation and statements from the manufacturer.

^b Fungicide group (FRAC Code): Numbers (1–37) and letters (M) are used to distinguish the fungicide mode of action groups. All fungicides within the same group (with same number or letter) indicate same active ingredient or similar mode of action. This information must be considered for fungicide resistance management decisions. M = Multi site inhibitors, fungicide resistance risk is low.

Source: <http://www.frac.info/> (FRAC = Fungicide Resistance Action Committee).

and at the end of the season, but can also affect leaf stalks, fruit stalks, flowers, and fruit. This disease produces small purple spots that first appear on older leaves and gradually enlarge, join other spots and finally produce large dead patches giving the leaves a scorched appearance.

1.3.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>D. earliana</i>	Leaf scorch	High

1.3.2 SYMPTOMS

1.3.2.1 Leaves

Leaf spots (lesions) may take two forms: pinpoint lesions in large or small numbers, and blotchy type lesions measuring 1/4 to 1/2 inches in diameter (Figure 1.11). Lesions are typically reddish to purple, coalescing to give a burned appearance to the plants. They often appear as numerous irregular, purplish to brownish blotches, 1–5 mm in diameter, developing on the leaf surface (laminae). The centers of these lesions do not become white or gray, as with leaf spot (*Mycosphaerella fragariae*). The blotches coalesce irregularly when numerous, and tissue between the blotches turns purplish to bright red. As the disease progresses, leaves turn brown,



FIGURE 1.11 Advanced scorch symptoms on strawberry leaves.

dry out, and turn up at the margins, assuming a burned or “scorched” appearance, as indicated in the name leaf scorch.

1.3.2.2 Leaf Stems (Petioles)

Lesions are typically elongated, sunken, purplish-brown or reddish-brown spots or streaks (Figure 1.12). Advanced lesions can girdle the petiole and kill the leaf.

1.3.2.3 Fruit

All parts of the flower truss and fruits may be infected. Peduncles and pedicels may develop elongated lesions and purplish streaks. In severe cases, tissues are girdled, resulting in the death of flowers and fruits. Infected petals wither and fall off. Irregular brown areas form on infected sepals, often on the margins or tips. These infections lead to fruit with dead calyxes (“dead cap”, “dead burr”) which are less attractive to consumers, resulting in lower market grades. Signs (visible presence of the pathogen)—using a hand lens, look for small dark spots or fungal fruiting bodies (acervuli) with glistening spore masses (Figure 1.13). As leaf lesions enlarge, they may gradually resemble drops of tar due to the production of large numbers of the minute black acervuli. Rarely, you might see apothecia develop on advanced lesions in leaves and strawberry leaf residues.

1.3.3 CAUSE AND DISEASE DEVELOPMENT

The fungus overwinters on infected leaves that survive the winter. In the spring, conidia are produced on both leaf surfaces in speck-sized black acervuli. The fungus also produces ascospores in the early spring, within disk-shaped apothecia (fungal fruiting structures) that appear as black dots



FIGURE 1.12 Strawberry leaf scorch on petioles.



FIGURE 1.13 Red blotches on the leaves and on the unripened fruit.

in old lesions on the lower surface of diseased leaves that died during winter. In the presence of moisture, ascospores germinate within 24 hours and infect the plant through the lower leaf surface. After symptom development, conidia are produced on the leaf spots in large numbers throughout the growing season. Therefore, repeated infections occur whenever weather conditions are favorable. Conidia are spread mainly by splashing water.

1.3.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

The fungus overwinters on infected leaves. The fungus produces spore-forming structures in the spring on both surfaces of dead leaves. These structures produce spores abundantly in midsummer. The disease is favored most in warm conditions. The disease is most severe at temperatures from 20°C to 25°C. In the presence of free water, these spores can germinate and infect the plant within 24 hours. Older and middle-aged leaves are infected more easily than young ones.

1.3.5 DISEASE CYCLE

Leaf scorch can progress year-round in most climates but dry conditions, and temperatures above 35°C and below freezing markedly reduce the rate of disease. In North America, scorch can continue to develop in foliage beneath snow cover at temperatures around -4°C–3°C. Symptoms appear quickly on leaves of early growth in spring when scorch is more commonly severe, increasing in late spring and late summer to mid-fall. Infection by ascospores has received little attention. Acervuli can remain dormant for long periods in dry leaves but mature quickly during wet periods. The sticky conidia are dispersed from the acervuli by splashing rain, dew, sprinkler water, and probably by arthropods. Conidia directly penetrate the cuticle and develop into a subcuticular intercellular mycelium. Lesions begin to appear 6–15 days after infection at favorable temperatures (15°C–30°C) provided a post-infection wetness period of nine-hours occurs (18-hours for very young leaves). Mature acervuli form 1–25 days after infection when the microclimate is favorable (leaf age dependent). (Figure 1.14).

1.3.6 MANAGEMENT

The disease can be managed using the following strategies:

- Chemical control
- Cultural control

1.3.6.1 Chemical Control

Several fungicides are registered for control of strawberry leaf diseases. Topsin M, Captan, Thiram, Nova, and Syllit (previously marketed as Cyprex) are all registered for use on strawberries. The

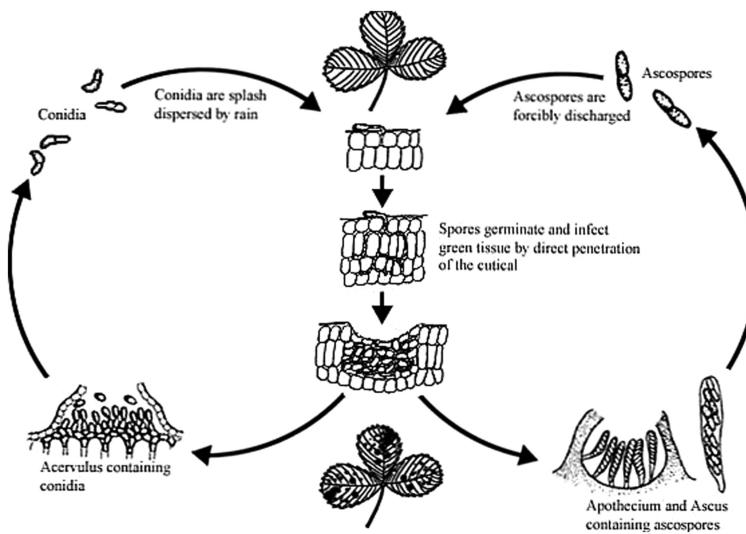


FIGURE 1.14 Strawberry leaf scorch (Red Spot) disease cycle.

label states that Topsin M cannot be applied before early bloom; thus, applications made very early in the season (as new growth starts) should use Syllit, Captan, Nova, or Thiram. The strobilurin fungicides (Cabrio, Abound, and Pristine) also have excellent activity against leaf diseases. If leaf diseases are a serious problem, post-harvest or post-renovation applications of these fungicides may be required. Nova and the strobilurin fungicides have the highest level of activity against leaf diseases. An alternating program of Nova and a strobilurin fungicide should provide excellent control of leaf diseases as well as fungicide resistance management.

1.3.6.2 Cultural Control

- Cultivars differ greatly in their resistance to leaf scorch. If it is a serious problem, use a more resistant cultivar.
- Don't use too much nitrogen fertilizer. It can cause soft, succulent foliage that is more susceptible to leaf scorch.
- Allow good air circulation for optimum drying by spacing plants appropriately and keeping weeds under control.
- Summer renovation will help reduce inoculum levels. In Oregon, after every two to four weeks of each harvest it is recommended to renovate crop types.

1.4 CRINKLE VIRUS

Strawberry Crinkle Virus (SCV), also known as Strawberry Frizz Virus, is a viral disease first reported in *F. vesca*. Its normal vector is the aphid *Chaetosiphon fragaefolii* and *C. jacobi*. The virus is carried by mechanical inoculation.

1.4.1 CAUSAL ORGANISM

The causal virus is *Cytorhabdovirus*, of family *Rhabdoviridae*. SCV is transmitted in a persistent propagative manner by the principal natural aphid vector *C. fragaefolii*. Infectivity of aphids is retained lifelong. The length of a transmission cycle in nature depends on temperature conditions since lower temperatures extend the incubation period in strawberry and the latent period in the vector. The virus also multiplies in aphid species other than *C. fragaefolii* when injected.

1.4.2 SPECIES AFFECTED

SCV has a narrow natural host range among species of *Fragaria*. It occurs on the wild species *F. vesca*, *F. virginiana*, and *F. chiloensis*, as well as on cultivated strawberries, *F. ananassa*.

1.4.3 SYMPTOMS

Symptoms vary in relation to strain and strawberry cultivar. Mild strains are symptomless in all cultivars (“strawberry latent virus”). Severe strains, in susceptible cultivars, cause distortion and crinkling of the leaves, with leaflets unequal in size and small irregularly shaped chlorotic spots, often associated with the veins (Figure 1.15).

1.4.4 MEANS OF MOVEMENT AND TRANSMISSION

Under natural conditions, SCV is dispersed locally by the strawberry aphid *C. fragaefolii*. Movement also occurs with runners or with propagated material from tissue culture.

1.4.5 PREVENTION AND CONTROL

Propagation of virus-free plants and control of vectors are the essential measures.

1.5 LATENT C VIRUS (SLCV)

The pathogen responsible for strawberry latent C disease has not been isolated or described morphologically, and its affinities are not known. Its normal vector is the aphid *C. fragaefolii*, which is widespread in Europe. The disease is otherwise only graft-transmissible. The organism behaves as a latent virus, normally giving no obvious symptoms on cultivated strawberries except in combination with other virus diseases, such as crinkle, mottle, vein banding, or yellows. It then causes moderate to severe degeneration.

1.5.1 CAUSAL ORGANISM

Rhabdovirus, of *Rhabdoviridae*, the causal agent of the disease, has not been morphologically described, but cross inoculations or natural complexes indicate that it is distinct from known strawberry viruses. In addition, electron microscopy of *F. vesca* (wild Strawberry) showing symptoms of strawberry latent C disease indicated the presence of virus particles belonging to the rhabdovirus group, which were accumulated in the peri-nuclear space and in the nuclei, whereas strawberry crinkle rhabdovirus particles were in the cytoplasm.



FIGURE 1.15 Crinkling of the leaves and unequally sized leaves.

1.5.2 SPECIES AFFECTED BY SLCV

- F. chiloensis* (Chilean strawberry)
- F. vesca* (wild strawberry)
- F. virginiana* (scarlet strawberry (United Kingdom))

1.5.3 SYMPTOMS

The pathogen alone causes no obvious symptoms in commercial strawberry cultivars. In the presence of other viruses, it causes moderate to severe degeneration in the form of extreme stunting, curling, and twisting of the leaves or an intensification of symptoms attributable to the other viruses. The chronic dwarf symptoms range from severe to moderate, but still show an obvious reduction in leaf size.

1.5.4 MEANS OF MOVEMENT AND TRANSMISSION

In the field, the disease is probably transmitted by the aphid vectors. In international trade, infected propagating material, including tissue cultures, is liable to carry the disease; infected strawberry material from the United States has been intercepted in the United Kingdom.

1.5.5 PREVENTION AND CONTROL

As a control method, heat treatment and meristem tip culture, applied separately, are only partly successful in eliminating the pathogen. The main control procedure is based on the use of certified virus-free planting material. The frequency of detection of SLCV in the field appears to be directly related to the presence of nearby sources in the planting. The production of cultivar clones free of SLCV and moderate care in isolation of seedling, selection, and nursery blocks from known sources, followed by the continued replacement of certified fruiting-field stocks, and possibly the use of aphicides, should result in the disappearance of this disease.

1.6 MILD YELLOW EDGE VIRUS

Strawberry mild yellow edge (SMYE) disease is one of the major diseases of strawberries in most parts of the world; however, because of the interaction of cultivars, viruses and virus strains, crop management, and environment, it is difficult to assess the importance of the disease in terms of economic loss. Alone, it is not particularly damaging to most cultivars, but it seldom occurs alone. The complex of the disease with other pathogens, for example, strawberry mottle agent, strawberry crinkle rhabdovirus, strawberry vein banding *caulimovirus*, or strawberry pallidosis agent, can cause severe loss of plant vigor, yield, and fruit quality. It was first reported in *F. vesca*; from California, USA and England; by Horne (1922); Harris (1933).

1.6.1 CAUSAL ORGANISM

Recent investigations have shown that SMYE disease is probably caused by a virus complex consisting of a *Potexvirus* (SMYE-associated *Potexvirus*) as well as a virus originally designated SMYE *Luteovirus*, but which is now recognized as a strain or synonym of soybean dwarf *Luteovirus*.

1.6.2 SPECIES AFFECTED

In nature, both viruses have been found only in *Fragaria spp.* The wild species *F. virginiana*, *F. vesca* and some clones of *F. chiloensis* show symptoms; *F. ovalis* is a symptomless carrier. Most strawberry cultivars are symptomless carriers of the disease.



FIGURE 1.16 Symptoms of SMYE Virus in a strawberry plant.

1.6.3 SYMPTOMS

Cultivated strawberries usually remain symptomless.

If symptoms appear, they generally are:

- Leaflets cupped
- Chlorotic margins
- Vigor reduced
- Chlorotic vein netting
- Necrosis of youngest leaves (Figure 1.16)

1.6.4 MEANS OF MOVEMENT AND TRANSMISSION

It is transmitted by a vector; an insect; *Chaetosiphon fragaraefolii*, *C. thomasi*, *C. thomasi jacobi*; Aphididae. The vector is transmitted in a persistent manner. Virus can help the vector transmission of another virus (SMYE-associated *Potexvirus*); transmitted by grafting; not transmitted by mechanical inoculation; not transmitted by contact between plants (of *F. vesca* clone); not transmitted by seed; not transmitted by pollen.

1.6.5 PREVENTION AND CONTROL

Control of the virus can be achieved by thermotherapy or meristem culture, combined with planting of certified virus-free material. Thermotherapy for SMYE was successful at approximately 50% when the central growing point was excised, and plants were almost completely defoliated during treatment for nine weeks at 38°C. The technique stimulated the development of side crowns, which could then be excised and rooted in sand at normal greenhouse temperatures.³

1.7 MOTTLE DISEASE

Strawberry mottle virus (SMoV), also known as Strawberry Mild Crinkle Virus, is a serious pathogen of strawberries (*Fragaria ananassa*) worldwide and is transmitted by aphids in a semi-persistent manner. Severe strains of SMoV may reduce yield by up to 30% and losses can be up to 80% in mixed infections with other viruses. SMoV occurs in many areas where strawberries are grown.

1.7.1 CAUSAL ORGANISM

The virus belongs to family *Secoviridae* and it has not been assigned with any genus name. It is commonly referred to as SMoV.

1.7.2 SPECIES AFFECTED

- *Fragaria × ananassa*
- *F. virginiana*
- *F. vesca*

1.7.3 SYMPTOMS

Dwarfing of leaves, mottle, vein clearing, and stunting are common symptoms caused by this virus.

1.7.4 MEANS OF MOVEMENT AND TRANSMISSION

It is transmitted by a vector, an insect, *C. fragaraefolii*, *C. thomasi*, *C. minor*, *C. jacobi*, *Aphis gossypii*; Aphididae. The virus is transmitted in a semi-persistent manner. The virus is lost by the vector when it molts; it does not multiply in the vector; is not transmitted congenitally to the progeny of the vector; is transmitted by mechanical inoculation; is transmitted by grafting; is not transmitted by contact between plants; and is not transmitted by seed.

1.7.5 PREVENTION AND CONTROL

- Always buy plants which are certified as virus-free. It is unwise to accept plants from old strawberry beds—these will almost certainly be infected with one or more viruses.
- Destroy and replace plants as soon as yields start to fall, usually after two or three years. Do not use runners from these plants, which will certainly be infected. Instead, buy new certified, virus-free stock
- If possible, avoid replanting strawberries on the same site. Since it is not practical to determine which virus is present on the basis of symptoms, because these are so variable, it is prudent to assume that some of the species spread by soil nematodes may be involved. These will persist in the soil and infect new plants.

1.8 NECROTIC SHOCK VIRUS

For many years, strawberry necrotic shock disease was thought to be caused by a strain of tobacco streak virus (TSV). Tzanetakis, et al. (2004) found that strawberry necrotic shock disease is caused by a different virus and not by a strain of TSV. It was then that the name Strawberry Necrotic Shock Virus (SNSV) was suggested for this virus instead of TSV.

1.8.1 CAUSAL ORGANISM

As mentioned above, the causal organism was once thought to be a strain of TSV, but the further research suggested that it was the SNSV and not TSV.

1.8.2 SPECIES AFFECTED

There are no symptoms seen in commercial cultivars of Strawberry caused by SNSV. Grafted susceptible indicator strawberry plants (*F. vesca*) may show a severe necrotic reaction in new leaves. These symptoms are however temporary, and the new growth appears to be normal and healthy.

1.8.3 SYMPTOMS

Severe necrotic reaction on the leaves is a major symptom observed. Symptoms may also include chlorosis, stunting, and leaf malformation. The commercial cultivars show no symptoms, but there is a visible reduction in the yield and runner production of these cultivars.

1.8.4 MEANS OF MOVEMENT AND TRANSMISSION

Transmission of this virus occurs through seed, pollen, or thrips. This virus has a wide host range, and host plant species near strawberry fields can serve as sources of inoculum.

1.8.5 PREVENTION AND CONTROL

As described, the commercial cultivars show no symptoms of the virus. It becomes very difficult to manage the disease as there are no visible symptoms. So, the most practical way to minimize the risk of infection on commercial fields is to use clean plant material (tissue-cultured, and virus-tested) and to follow best management practices for insect and weed control.

1.9 VEIN BANDING CAULIMOVIRUS

Strawberry Vein Banding Virus (SVBV) is a plant pathogenic virus and a member of the family *Caulimoviridae*. It was first described by Fraizer after a differential aphid transmission to susceptible wild strawberries. He identified suitable virus indicators and demonstrated virus transmission by various aphids, dodder, and grafting. He also established the inability of the virus to transmit via sap.

1.9.1 CAUSAL ORGANISM

The virus, *Caulimovirus*, belonging to family *Caulimoviridae*.

1.9.2 SPECIES AFFECTED

The virus, *Caulimovirus*, is known to occur only on *Fragaria* spp. The main host is *F. vesca* (wild strawberry). Commercial strawberries may also be infected, but diagnostic symptoms are usually only apparent when strawberry latent C “rhabdovirus” is present simultaneously.

1.9.3 SYMPTOMS

1.9.3.1 On *F. vesca*

Symptoms initially appear on the youngest developing leaf; there is an epinasty of midribs and petioles, a tendency for opposite halves of leaflets to be appressed, irregularly wavy leaflet margins, and slight crinkling of the laminae. Usually, the above symptoms are mild and not all present simultaneously. It is not until the affected leaf expands that clearing, followed by yellowish banding of some or all of the veins, becomes visible. Often, the coloration occurs in scattered discontinuous streaks of varying lengths along the main and secondary veins. The second and third leaves formed after onset of symptoms are affected more severely than the first or any subsequent leaf; in older leaves, chlorotic streaks are reduced in number, scattered, and confined to portions of the leaflets. This may be followed by the appearance of a series of apparently healthy leaves and then reappearance of mild or severe symptoms.

1.9.3.2 On Commercial Strawberries

There are no very diagnostic symptoms but, if strawberry latent C disease is also present, the reaction to infection is intermediate to that on *F. vesca*. As affected leaves mature, the vein-banded areas may gradually disappear, or they may become brownish-red or necrotic. Especially on outdoor plants, the veins become discolored, without previous chlorosis. Affected leaflets characteristically exhibit epinasty, mild crinkling, and wavy margins.

1.9.4 MEANS OF MOVEMENT AND TRANSMISSION

In the field, the virus is transmitted by aphid vectors. Because of the ability of certain aphid species to undertake long, high-altitude flights, wide natural dissemination is possible. This is, however, limited by the relatively short persistence of the virus in the vector. In international trade, SVBV is liable to be carried on infected plants and propagating material of strawberries. The following aphids are cited as vectors: *Acyrthosiphon pelargonii*, *Amphorophora rubi*, *Aphis idaei*, *A rubifolii*, *Aulacorthum solani*, *C. fragaefolii*, *C. jacobi*, *C. tetrarhodum*, *C. thomasi*, *Macrosiphum rosae*, *Myzus ascalonicus*, *M. ornatus*, *M. persicae*.

Of these species, *Chaetosiphon* spp. are the most efficient vectors in glasshouse experiments, although other genera are probably important vectors when they occur in large numbers and frequently move from plant to plant. Aphids can acquire and transmit the virus in 30–120 minutes, but persistence in the vector is short, usually less than eight hours (semi-persistent type). There are differences in the efficiency of clonal lines of aphids, and evidence that some species will transmit only certain strains of SVBV. *A. gossypii*, *A. fabae*, *A. solani*, and *Macrosiphum euphorbiae* failed to transmit the virus in a limited number of trials.

The virus is transmissible by grafting and by means of *Cuscuta subinclusa*. Attempts to transmit SVBV mechanically have been unsuccessful. The incubation period in the indicator host varies from two to five weeks depending on the strain.

1.9.5 PREVENTION AND CONTROL

There are no specific control measures. SVBV is highly resistant to inactivation by heat therapy but it can be eliminated from plants by means of meristem tip culture. As a consequence, the use of certified planting material is the best control procedure, and certification schemes for the production of healthy planting material of strawberry are in operation in several countries. Control of aphids with insecticides could reduce the incidence of the disease.

1.10 PHOMOPSIS LEAF BLIGHT OF STRAWBERRY

Phomopsis leaf blight is a common disease of strawberry in the eastern United States. Although the fungus infects leaves early in the growing season, leaf blight symptoms are most apparent on older leaves near or after harvest in Ohio. The economic importance of leaf blight in Ohio appears to be relatively minor; however, incidence of the disease has been increasing. The disease can weaken strawberry plants through the destruction of older foliage. Weakened plants can result in reduced yields the following year. In years highly favorable for disease development, leaf blight can cause defoliation and, in some cases, the death of plants.

Especially in warmer climates, the fungus that causes leaf blight can also cause a fruit rot called soft rot. The first observation of *Phomopsis* fruit rot (soft rot) in Ohio was on plants growing under plastic culture in 1999. Although not common in Ohio, *Phomopsis* fruit rot can result in serious losses.

1.10.1 CAUSAL ORGANISM

Phomopsis leaf blight is majorly caused by the following mentioned fungal organism:

Species	Associated Disease Phase	Economic Importance
<i>Phomopsis obscurans</i>	Leaf blight (Soft rot)	Serious



FIGURE 1.17 Leaf Spot like symptoms.

1.10.2 SYMPTOMS

Leaf blight is caused by the fungus *P. obscurans*. Leaf blight is found most commonly on plants after harvest. The disease is distinctively different from both leaf spot and leaf scorch. However, the young lesions resemble that of Strawberry Leaf Spot (Figure 1.17). The enlarging leaf spots of this disease are round to elliptical or angular and a quarter of an inch to an inch in diameter (Figure 1.18). Spots are initially reddish purple. Later, they develop a darker brown or reddish-brown center surrounded by a light-brown area with a purple border. Similar spots may sometimes develop on the fruit caps. Usually, only one to six lesions develop on a leaflet. Often the infected area becomes V-shaped with the widest part of the “V” at the leaf margin. New lesions appear throughout the summer and fall if weather conditions are favorable. Older leaves become blighted and may die in large numbers. This disease is usually more destructive on slow-growing or weak plants. The same fungus can cause an enlarging, soft, pale-pink rot at the stem end of the fruit. Black specks of pycnidia often develop within the central areas of the older lesions. Initial symptoms on fruit are round, light pink, and water-soaked lesions (Figure 1.19). Frequently, two or more lesions may coalesce into large soft dark brown lesions. Information on resistance to leaf blight in currently used varieties is limited. If growers encounter a high level of disease on certain varieties, these varieties should be avoided.



FIGURE 1.18 *Phomopsis* leaf blight on strawberry.



FIGURE 1.19 *Phomopsis* soft rot.

1.10.3 CAUSE AND DISEASE DEVELOPMENT

This fungus produces conidia in speck-sized, black pycnidia (fungal fruiting bodies) embedded in the centers of older leaf lesions. Conidia ooze out of pycnidia during damp weather when temperatures are high. Conidia are splashed to new leaf tissue where they germinate in the presence of free water to initiate new infections on leaves and fruit. The fungus overwinters on either infected leaves that survive the winter or in dead tissue on old infected leaves.

1.10.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

The disease is spread largely by wind and splashing water from rain or overhead irrigation. The optimum conditions for disease development are temperatures ranging from 26°C–32°C and 72 hours of leaf wetness. Leaf Blight has worldwide distribution but tends to be more severe in cooler climates.

Extended wet periods, particularly in autumn, are the most favorable conditions for the development of disease.

1.10.5 DISEASE CYCLE

P. obscurans overwinters as mycelium and pycnidia on old leaves attached to the plant. Conidia from pycnidia are viable in early spring and are rain splashed onto new leaves. Primary infection may occur early in the growing season, but symptom expression and rapid disease progression do not occur until mid-season.

1.10.6 MANAGEMENT

The disease survives from year to year in infected leaf debris. Management practices include:

- Removal and burial of infected leaves during renovation,
- Use of tolerant varieties,
- Application of an effective and properly timed fungicide, such as Equal 65 WP. Unfortunately, Equal 65 WP applied in cold temperatures and cold weather can injure strawberry leaves.

Unfortunately, *Phomopsis* leaf blight occurs sporadically from year to year. Infection depends on weather conditions leading up to harvest. If we could predict when infection takes place, growers could target fungicide applications more precisely.

In a recent study at Ohio State University, researchers looked at the influence of temperature, leaf wetness duration and leaf age on the infection of strawberry leaves cv. "Honeye" and "Earliglow". Their objective was to develop a prediction model for leaf blight. Disease incidence and severity were most influenced by the age of the leaf. The younger the leaf at the time of infection, the higher the disease observed four weeks later. Leaf wetness duration also significantly influenced the disease. Longer leaf wetness periods resulted in higher levels of the disease. Surprisingly, relationship between infection and temperature on disease severity and incidence was minor.

The results of this study may help to explain the severity of leaf blight observed in 2003. Extended rainy periods in late May and early June were ideal for infection of the new leaves. In addition, overhead irrigation for frost protection resulted in more extended leaf wetness periods. Although August was very warm and dry, September and October were very wet, which resulted in more infections.

High levels of leaf blight may survive this winter and could result in higher disease pressure this coming spring. The disease prediction model developed in Ohio has not been tested in Ontario; however, results suggest that fungicides should be applied to protect new leaves, prior to a leaf wetness event greater than five hours, regardless of temperature. This will be particularly important during next spring and after renovation when plants are producing an abundance of new susceptible leaves.

1.11 GRAY MOLD

Botrytis fruit rot, also called Gray Mold, is a major disease of Strawberries throughout the world. The disease, caused by the fungus *Botrytis cinerea*, is responsible for fruit losses of 50% or more during cool, wet seasons.

In addition to Strawberries, *Botrytis* also causes economic losses for many other crop plants.

The disease affects fruit in the field, resulting in severe pre-harvesting losses. It also affects fruits after harvest, since infections that begin in the field continue to develop during storage and transit at refrigeration.

Fruit turns brown at the calyx end and the fungus produces a gray cotton-like growth on the surface.

1.11.1 CAUSAL ORGANISM

Botrytis fruit rot is majorly caused by a single species of *Botrytis* mentioned below:

Species	Associated Disease Phase	Economic Importance
<i>B. cinerea</i>	Fruit rot	High

1.11.2 SYMPTOMS

Strawberry flowers are highly susceptible to *B. cinerea* and may be blighted directly (Figure 1.20). However, symptoms usually are observed later, on green and ripening fruit. Lesions typically develop on the stem end of the fruit and are often associated with infected stamens or dead petals adhering to the fruit or trapped beneath the calyx (Figure 1.21). Lesions begin as small, firm, light brown spots that enlarge quickly (Figure 1.22). During periods of rainy weather, heavy dews, or high relative humidity, lesions become covered with masses of tan to gray spores (Figure 1.23). Large

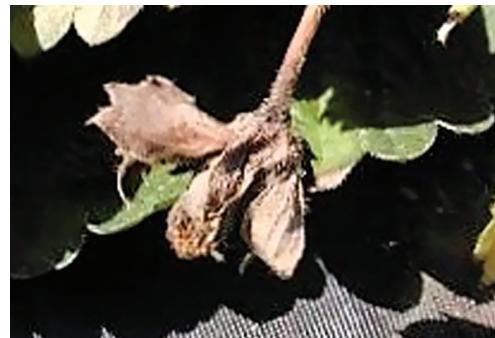


FIGURE 1.20 Flower blighted by *B. cinerea*.

numbers of spores are released as visible gray puffs when infected fruit are disturbed. *Botrytis* may consume and mummify the entire fruit (Figure 1.24).

1.11.3 CAUSE AND DISEASE DEVELOPMENT

B. cinerea is a common colonizer of strawberry foliage in the nursery and is also present on dying vegetation around strawberry fields. After transplanting, spores produced on old dying leaves rapidly colonize new emerging leaves without causing visible symptoms. These conidia are dispersed by air, water, and harvesters to infect flowers during the main bloom period in January and February. Cool to mild temperatures and prolonged leaf wetness promote spore production, germination, and infection of stamens, petals, and other floral parts. Flower infections often progress slowly, with lesions becoming visible on green and ripening fruit two to four weeks after infection. Direct infection of fruit by spores is not considered important in the field or after harvest. However, the pathogen also spreads from diseased fruit to healthy fruit by direct contact (Figure 1.25). As the epidemic progresses, diseased fruit, mummified fruit, and decayed flowers and pedicles become important new sources of inoculum. *Botrytis* fruit rot is especially damaging in annual production systems characterized by prolonged flowering and fruiting cycles. In Florida, the second crop of



FIGURE 1.21 *Botrytis* lesion from colonized petal (arrow).



FIGURE 1.22 *Botrytis* lesion without spores.

fruit that ripens in February and March are more seriously affected than the first crop of fruit that ripen in December and January.

1.11.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Disease development is favored by wet conditions accompanied by temperatures between 5°C and 30°C. Conditions that impede drying of fruit wetted by rain or sprinkler irrigation will encourage *Botrytis* rot.



FIGURE 1.23 *Botrytis* lesion with spores.



FIGURE 1.24 *Botrytis*-mummified fruit.

The gray mold fungus is readily airborne and commonly encountered. Winter carryover is greatest in fields in which there is a large amount of dead plant material, on which the fungus develops. Mild, wet, humid weather is most favorable for infection. Most infections of the fruit result from blossom infections that remain latent in the developing berry, becoming active and causing a rot when the fruit ripens.

1.11.5 DISEASE CYCLE

B. cinerea may colonize and produce conidia on almost any plant debris. It overwinters in strawberry plantings on decayed foliage and fruit from the previous season. Increasing temperatures and moisture in the spring promotes fungal growth and the production of conidia, which are spread by wind and rain to the developing strawberry plants. *Botrytis* conidia are abundant throughout the growing season in most strawberry growing areas.

Strawberries are susceptible to *Botrytis* during bloom and again as fruits ripen. During the blossom blight phase of the disease, the fungus colonizes senescing flower parts, turning the blossoms brown. Blossom infections establish the fungus within the plant and produce inoculums that can spread the fungus to other plants. Cool, wet weather and particularly frost injury favor blossom infections. The fungus can then move into developing fruit and remain quiescent until the fruits start to mature, at which time the rot becomes noticeable.



FIGURE 1.25 Fruit-to-fruit spread of *B. cinerea*.



FIGURE 1.26 Leaf Blight.

Infections may be associated with senescent petals adhering to sepals at the stem end of green or ripe fruit. Infected senescent petals adhering to leaves may also result in leaf blight (Figure 1.26). Abundant gray-brown, fluffy, fungal growth on infected tissue is responsible for the disease's name "Gray Mold".

Fruit infections may be noticeable on green fruits; however, they are most apparent on ripe fruit where abundant sporulation may develop. Fruits touching the ground or in areas where poor air drainage does not allow for rapid drying are most likely to become rotted. When conditions are conducive to abundant blossom infection, the chance of a high level of fruit rot developing at harvest is increased. Figure 1.27 shows the cycle of the disease.

1.11.6 MANAGEMENT

The disease can be managed using the following strategies:

- Chemical control
- Biological control
- Cultural control
- Integrated pest management

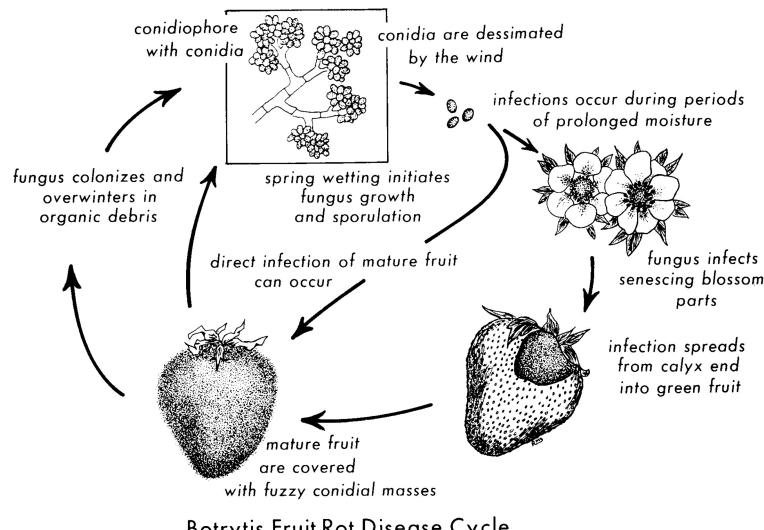


FIGURE 1.27 Disease cycle of *Botrytis* fruit rot on strawberry.

1.11.6.1 Chemical Control

Gray mold control can be aided by applying protective fungicides beginning at or before bloom and continuing until harvest. Where gray mold has been a significant problem before, applications should begin at the white bud stage of flower development. Also, where frost has damaged a planting and a marketable crop remains, great care should be taken to maintain a strict fungicide spray program. In commercial fields in Central Florida, fungicide applications are usually necessary to suppress sporulation and protect flowers from infection. A good disease management program is based on regular applications of a broad-spectrum protective fungicide such as Captan or Thiram. Applications at low rates should begin after overhead irrigation for plant establishment has ended and continued throughout the season. Strawberries bloom from November to March in Florida, but peak blooms occur in November and January/February. Disease incidence is usually low in the first bloom and the regular protectant applications are sufficient to prevent significant early-season losses. During the second peak bloom, fungicides with good activity against *Botrytis* fruit rot can be substituted for protective applications. Captevate®, Elevate®, Pristine®, Scala®, and Switch® are among the most effective fungicides for control of *Botrytis* fruit rot (Table 1.3). The first application should be made at 10% bloom (usually late January). Susceptible cultivars may require up to four applications at weekly intervals to protect flowers throughout the bloom period. Applications are especially critical during periods of mild temperatures and prolonged wetness caused by rains, fogs, or heavy dews. Once this critical period has ended, normal applications of Captan or Thiram can be resumed, usually at high label rates. Applications of protectant fungicides are usually sufficient to control *Botrytis* fruit rot in March when the disease is naturally suppressed by hot weather.

1.11.6.2 Biological Control

Trichoderma isolates are known for their ability to control plant pathogens. It has been shown that various isolates of *Trichoderma*, including *T. harzianum* which isolate T-39 from the commercial biological control product TRICHODEX, were effective in controlling gray mold (*B. cinerea*) in strawberry under controlled and greenhouse conditions. Three selected *Trichoderma* strains, namely T-39, T-161, and T-166, were evaluated in large-scale experiments using different timing application and dosage rates for the reduction of strawberry gray mold. All possible combinations of single, double, or triple mixtures of *Trichoderma* strains, applied at 0.4% and 0.8% concentrations, and at seven- or ten-day intervals were tried. Only a few treatments resulted in significant control of gray mold. Isolates T-39 applied at 0.4% at two-day intervals, T-166 at 0.4%, or T-161 combined with T-39 at 0.4% were as effective as the chemical fungicide fenhexamide. The biocontrol isolates were identified to the respective species *T. harzianum* (T-39), *T. hamatum* (T-105), *T. atroviride* (T-161), and *T. longibrachiatum* (T-166), according to internal transcribed spacer sequence analysis.

1.11.6.3 Cultural Control

Botrytis fruit rot can be controlled by both chemical and cultural measures. Cultural practices include the use of resistant cultivars and the physical removal of infected plant parts (plant sanitation). Although there are no commercial cultivars highly resistant to this disease, “Camarosa”, “Carmine”, and the newly released “FL Radiance” and “FL Elyana” are less susceptible to *Botrytis* fruit rot than “Strawberry Festival”, “Treasure”, and “Sweet Charlie”. The Californian cultivar “Camino Real” has been proven highly susceptible under Florida conditions. Cultivars with large clasping calyces are generally more susceptible because moisture trapped between the calyx and the receptacle promotes the spread of the pathogen from stamens and petals to the developing fruit. Removal of senescing and dying leaves after establishment helps to eliminate a potential source of inoculum. However, studies have shown that leaf pruning modestly reduces disease incidence, but does not increase marketable yield, and is not practical due to the high cost of labor. Yields may even be reduced when pruning includes the removal of partially green leaves. However, the removal

of diseased and culled fruit from the plant canopy during normal harvest operations is considered vital to successful management of *Botrytis* fruit rot.

Certain cultural practices help control gray mold by promoting faster drying of foliage and fruit while other practices reduce exposure to fungal inoculum.

- Select a planting site with good soil drainage and air circulation.
- Expose planting to full sun.
- Orient plant rows toward the prevailing wind.
- Apply appropriate nitrogen levels to prevent excessive foliage from developing.
- Mulch plants with straw to reduce fruit contact with the soil.
- Pick fruit frequently.
- Cull out and remove diseased berries from the planting.
- Handle berries with care to avoid bruising and refrigerate harvested fruit promptly at 0°C–10°C (32°F–50°F).

The following materials are listed in order of usefulness in an IPM Program, considering efficacy. Also, consider the general properties of the fungicide as well as information relating to environmental impact. Not all registered pesticides are listed. Always read the label of the product being used.

1.12 LEATHER ROT OF STRAWBERRY

Leather rot is caused by the soilborne pathogen *Phytophthora cactorum*. The leather-rot pathogen is a fungus-like organism called an oomycete and is not a true fungus. Leather rot has been reported in many regions throughout the United States. In many areas, it is considered a minor disease of little economic importance. However, excessive rainfall during May, June, and July can lead to severe fruit losses and quality reduction. In 1981, many commercial growers in Ohio lost up to 50% of their crop to leather rot. The leather rot pathogen primarily attacks the fruit but may also infect the blossoms. The key control methods are maintaining a good layer of straw mulch between the fruit and the soil, selecting well-drained planting sites, improving water drainage through tiling before planting, or using other methods to improve soil drainage. Avoiding soils that become saturated with water is critical for leather rot control.

1.12.1 CAUSAL ORGANISM

Leather Rot is majorly caused by single species of *Phytophthora* mentioned below:

Species	Associated Disease Phase	Economic Importance
<i>P. cactorum</i>	Fruit rot	Low

1.12.2 SYMPTOMS

The leather rot pathogen can infect berries at any stage of development. When the disease is serious, infection of green fruit is common. On green berries, diseased areas may be dark brown or natural green outlined by a brown margin (Figure 1.28). As the rot spreads, the entire berry becomes brown, maintains a rough texture, and is leathery in appearance. The disease is more difficult to detect on ripe fruit. On fully mature berries, symptoms may range from a little color change to discoloration that is brown to dark purple (Figure 1.29). Generally, infected mature fruit



FIGURE 1.28 Leather rot symptoms on an immature strawberry fruit.

are dull in color and are not shiny or glossy. Infected ripe fruit are usually softer to the touch than healthy fruit. When diseased berries are cut across, a marked darkening of the water-conducting system to each seed can be observed. In later stages of decay, mature fruits also become tough and leathery. Occasionally, a white moldy growth can be observed on the surface of infected fruit. In time, infected fruit dry up to form stiff, shriveled mummies. Berries that are affected by leather rot have a distinctive and very unpleasant odor and taste. Even healthy tissue on a slightly rotted berry is bitter. This presents a special problem to growers in pick-your-own operations. An infected mature berry with little color change may appear normal and be picked and processed with healthy berries. Consumers have complained of bitter-tasting jam or jelly made with berries from fields where leather rot was a problem. Leather rot is most commonly observed in poorly drained areas where there is or has been free-standing water or on berries in direct contact with the soil.

1.12.3 CAUSE AND DISEASE DEVELOPMENT

The pathogen survives the winter as thick-walled resting spores, called oospores, that form within infected fruit as they mummify. These oospores can remain viable in soil for long periods of time. In the spring, oospores germinate in the presence of free water and produce a second type of spore called a sporangium. A third type of spore called a zoospore is produced inside the sporangium. Up to 50 zoospores may be produced inside one sporangium. The zoospores have tails (flagella) and can swim in a film of water. In the presence of free water on the fruit surface, the zoospores germinate and infect the fruit. In later stages of disease development, sporangia are produced on the surface of infected fruit under moist conditions. The disease is spread by splashing or windblown water from rain or overhead irrigation. Sporangia and/or zoospores are carried in water from the surface of the infected fruit to healthy fruit where new infections occur.



FIGURE 1.29 Leather rot symptoms on a mature strawberry fruit. Note the light, off-color area on the fruit.

1.12.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Under the proper environmental conditions, the disease can spread very quickly. A wet period (free water on fruit surface) of two hours is sufficient for infection. The optimum temperatures for infection are between 16°C and 25°C. As the length of the wet period increases, the temperature range at which infection can occur becomes much broader. As infected fruit dry up and mummify, they fall to the ground and lie at or slightly below the soil surface. Oospores formed within the mummified fruit enable the fungus to survive the winter and cause new infections the following year, thus completing the disease cycle.

1.12.5 DISEASE CYCLE

Leather rot is caused by *P. cactorum*, the same organism that causes crown and collar rots of apple and other deciduous fruit trees. This fungus is present in many soils throughout New York.

P. cactorum persists in the soil as thick-walled resting spores (oospores), which can survive in a dormant state for many years. When the soil is moist or wet, some of the oospores in the soil germinate and form structures called sporangia, which are filled with the infection spores of the fungus (zoospores). These microscopic zoospores are released into the soil when it is flooded or puddled and swim to the surface using the tail-like structures that they possess.

A leather rot epidemic can begin when strawberry fruit becomes infected after lying in puddled water containing zoospores or when the puddled water is splashed onto them by rain or sprinklers.

Following initial infection, the leather rot fungus forms additional sporangia on the fruit surface during periods of plentiful rainfall and high relative humidity. The sporangia are spread through the air by wind and rain and cause new infections. Still more sporangia may be produced on newly infected fruit and continue to spread the disease as long as weather conditions remain favorable. The leather rot fungus eventually forms its resting spores (oospores) within the infected fruit, and these spores are returned to the soil when the fruit falls to the ground and decays (Figure 1.30).

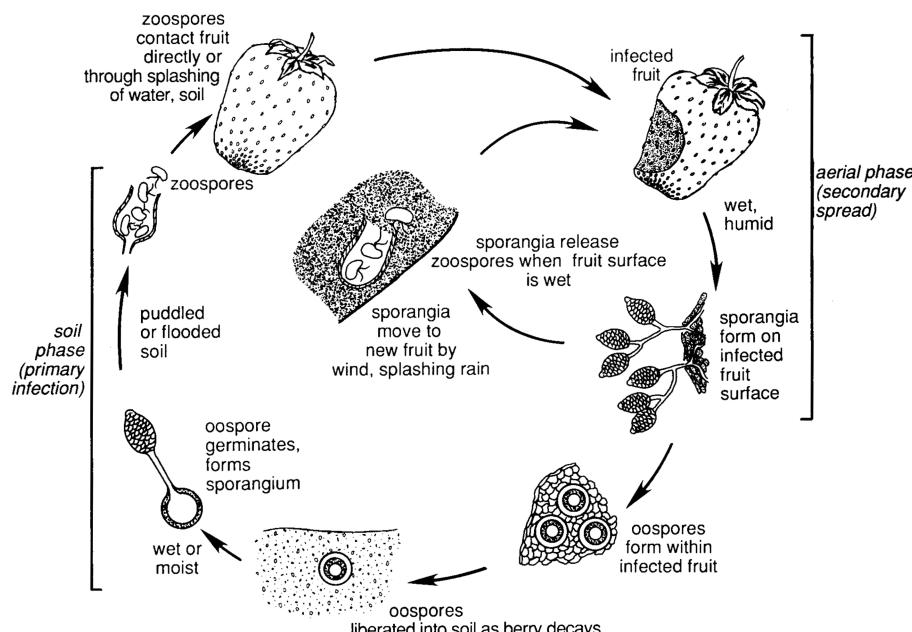


FIGURE 1.30 Leather Rot disease cycle.

1.12.6 MANAGEMENT

Leather rot is not common on annual plantings of strawberries in California because it is usually controlled by preplant fumigation and plastic mulches. Cultural practices play an important role in disease prevention; soil solarization may also provide control. Plantings held for two or three years, however, could be infected by the leather rot pathogen.

1.12.6.1 Chemical Control

Most fungicides currently available for use on strawberries are generally ineffective for controlling leather rot. Although Captan and Thiram are beneficial in suppressing leather rot, they will not provide adequate control if an epidemic develops. Furthermore, the use of these fungicides is severely restricted or prohibited during harvest due to re-entry restrictions or preharvest intervals. Ridomil is registered for use on strawberries for control of red stele and leather rot. Ridomil is very effective for control of leather rot and may be applied in the spring after the ground thaws and before first growth. This early application is recommended primarily for control of red stele but may be beneficial in providing some control of leather rot. A second application is recommended specifically for leather rot and can be made during the growing season at fruit set. Aliette 80% WDG is also registered for use on strawberries and should provide good control of both red stele and leather rot. It can be applied from the initiation of bloom through harvest on a seven- to fourteen-day schedule and has no preharvest restriction.

1.12.6.2 Cultural Control

Ensure that fields are prepared so that they have adequate water drainage. Remove diseased fruit and use plastic mulches. Avoid overhead irrigation; use drip irrigation. Straw mulch has been effective in controlling this disease in the eastern United States.

1.12.6.3 Soil Solarization

This is a unique management strategy to prevent leather rot. In warmer areas of the state, solarization has been shown to be effective for the control of soilborne pathogens and weeds. Solarization is carried out after the beds are formed and can be effective if weather conditions are ideal (30–45 days of hot weather that promotes soil temperatures of at least 50°C). The effectiveness of solarization can be increased by solarizing after incorporating the residue of a cruciferous crop, in particular broccoli or mustards, into the soil or following an application of metam sodium (40 gal/ha).

Table 1.4 outlines the percentages of fungicide effectiveness at disease control: (Table 1.4)

TABLE 1.4
Effect of Fungicides on Control of Strawberry Leather Rot

Treatment and Rate (a.i/ha)	Leather Rot (%) ^a	Marketable Fruit (%) ^b	Total No. of Fruits	Total Yield (kg) ^c	Percent Disease Control
Pyraclostrobin (0.20 kg)	0.5 a ^d	96.8 a	1080 a	10.8 a	99
Azoxystrobin (0.28 kg)	0.4 a	97.8 a	1054 a	10.9 a	99
Phosphorous acid (2.35 kg)	0.8 a	96.8 a	1065 a	10.4 a	98
Mefenoxam (0.56 kg)	0.3 a	97.9 a	1080 a	9.6 a	99
Untreated control	58.1 b	35.9 b	1144 a	7.7 b	—

^a Mean percentage of *Phytophthora cactorum*-infected fruit from three harvest dates (June 3, 7, and 10).

^b Mean percentage of marketable fruit from the above three harvest dates.

^c Total yield from the above three harvest dates for 3 m of crop row per replication.

^d For percentages, the analysis was based on the angular transformation. Numbers followed by the same letter within columns do not differ significantly according to Duncan's modified (Bayesian) LSD test ($P = 0.05$).

1.13 RHIZOPUS FRUIT ROT

Rhizopus fruit rot, or leak, is primarily a postharvest or storage rot, but it may also occur in the field on ripe fruit. The disease is caused by the fungus *Rhizopus* spp.

1.13.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>Rhizopus stolonifer</i> ,	Fruit rot	N/A
<i>Rhizopus nigricans</i> and Other spp.		

1.13.2 SYMPTOMS

Initial infections of *Rhizopus* fruit rot appear as discolored, water-soaked spots on fruit. These lesions enlarge rapidly, releasing enzymes that leave the berry limp, brown, and leaky (Figure 1.31). Under conditions of high relative humidity, the berry rapidly becomes covered with a coat of white mycelium and sporangiophores. The sporangiophores develop black, spherical sporangia, each containing thousands of spores. When disrupted, these sporulating berries release a cloud containing millions of spores. *Rhizopus* and mucor fruit rots closely resemble each other and may be difficult to differentiate in the field.

1.13.3 CAUSE AND DISEASE DEVELOPMENT

The fungus is an excellent saprophyte that lives on and helps break down decaying organic matter. It invades strawberries through wounds and secretes enzymes that degrade and kill the tissue ahead of the actual fungal growth. The fungus is active most of the year in California and survives cold periods as mycelium or spores on organic debris. Spores are airborne. The pathogen has a large host range and is prevalent worldwide.

1.13.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Damaged and overripe fruit exposed to warm temperature and high humidity are the most favored conditions for the disease development.

1.13.5 DISEASE CYCLE

The fungus survives on crop debris and in the soil between seasons. *Rhizopus* can only infect through wounds. Under favorable conditions of high temperature and moisture, sporulation is rapid and abundant. Spores are disseminated by air and by insects.



FIGURE 1.31 Fruit break down caused by *Rhizopus* rot.

1.13.6 MANAGEMENT

Rhizopus stops growing at temperatures below 8°C–10°C (46°F–50°F), so rapid postharvest cooling of fruit is essential for disease control. Field sanitation also is extremely important: do not leave discarded plant refuse or berries in the furrows and be sure to remove all ripe fruit from the field. There are some benefits to the use of protective fungicides, but unless the disease is widespread throughout the field, this pathogen should not cause excessive damage.

1.13.6.1 Cultural Control

Field sanitation is extremely important. Handle fruit with care always. Remove all ripe fruit from the field at harvest. Be sure when fruit is being picked that the entire fruit is removed from the stem, and the fleshy receptacle of the fruit is not left behind as it can serve as a site for invasion by fungus. Cultivars with thick cuticles are less susceptible to *Rhizopus* fruit rot because they are better able to resist infection.

1.13.6.2 Organically Acceptable Methods

Sanitation, cultivar selection, and rapid postharvest cooling are acceptable for use in an organically certified crop.

1.13.6.3 Treatment Decisions

Fungicide treatment is not recommended.

1.14 RED STELE ROOT ROT OF STRAWBERRY

Red stele root rot is a destructive disease in most strawberry producing regions of the world where soils tend to be cool and wet. It is very common in poorly drained soils, particularly during wet spring seasons or those following a rainy autumn. The disease is most destructive in heavy clay soils that are saturated with water during cool weather. Once established in the soil, the fungus remains alive for up to 13 years and possibly longer, regardless of the crop rotation used. Most infections are passed directly from strawberry plant to strawberry plant. Red stele usually does not appear in a new planting until the spring of the first bearing year, from about full bloom to harvest. Minor symptoms of root infection may appear, however, in late fall of the first growing season. Damage increases each year that susceptible cultivars are grown in the infested soil.

Red stele may appear to be well distributed over an entire strawberry field or patch during a cool, wet spring. Normally, however, the disease is most prevalent in lower or poorly drained areas.

1.14.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>Phytophthora fragariae</i>	Root rot	Major

1.14.2 SYMPTOMS

1.14.2.1 Aboveground

Symptoms of red stele rarely occur in the first year of strawberry growth unless plants were severely diseased before planting or if soil conditions were suitable for rapid fungal growth. Usually, red stele is first noticed during bloom of the second year. The symptoms will be most noticeable in low or soil compacted areas of a field where water drainage is poor. Strawberry plants infected with *P. fragariae* will show a general lack of vigor with poor runner growth and small berries. New



FIGURE 1.32 Longitudinal section of a healthy (left) and red stele-infected (right) strawberry root.

leaves may appear bluish-green, while older leaves sometimes turn red, orange, or yellow. The leaves tend to wilt during warm weather or drought stress. Severely diseased plants may collapse prior to fruiting. Although these aboveground symptoms are typical for red stele, they may resemble symptoms caused by other types of root disorders; therefore, roots also need to be examined.

1.14.2.2 Below Ground

To correctly diagnose red stele, strawberry roots should be sampled during early spring and summer up until the time of harvest. Samples taken after harvest are not reliable because infected roots may have already begun to decay. When taking a plant sample, dig rather than pull the plant from the ground. Examine the roots of plants which are just beginning to show signs of wilting. If red stele is present, the roots will appear unbranched and will be lacking feeder roots. This “rat-tail” appearance of the root is a diagnostic trait of red stele. Select a white root with a rotted tip and make a lengthwise cut at the point where diseased root tissue meets healthy tissue. Red stele infected roots will have a reddish-brown core, but the outer tissue will be white. The discoloration will begin at the root tip and move upward, but usually will not move into the crowd (Figures 1.32 and 1.33).

1.14.3 CAUSE AND DISEASE DEVELOPMENT

Often poor drainage leads to general root rot and contributes to red stele root rot. These plants need well-drained soil and will not do well in other soils. Poor drainage stresses plants and roots die from insufficient oxygen. Poor drainage may result from heavy (clay-like) soil, high water table, etc. It is usually a winter problem and not noticed at the time. Such soil may appear well-drained in the summer.



FIGURE 1.33 Red stele infected roots on the left and healthy roots on the right. Note the absence of numerous roots.

Some fungi attack roots that are stressed because of wet soil conditions and cause further root damage. They may be involved in general root rot. Red stele root rot disease is caused by *P. fragariae* and is favored by wet soil.

1.14.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Normally, the disease is prevalent only in the lower or poorly drained areas of the planting; however, it may become fairly well distributed over the entire field, especially during a cool, wet spring. The red stele fungus may become active at a soil temperature of 4°C. However, the optimum soil temperature for growth and disease development is between 13°C and 15.5°C. Under favorable conditions of high soil moisture and cool temperatures, plants will show typical disease symptoms within ten days after infection.

Soil types do not affect the presence or absence of the red stele fungus. It grows in any soil with a pH of 4.0–7.6 but will not grow in an alkaline soil (a pH of 8.0 or above). Heavy clay soils, which retain moisture for long periods of time, provide a conducive environment for the development of the red stele disease because the zoospores can spread greater distances and produce more infection sites.

1.14.5 DISEASE CYCLE

The red stele fungus is spread from one field, or area, to another primarily by the distribution of nursery infected plants. Infection is then spread within the field by moving water, and by soil carried on implements and shoes. Once in the field, thick-walled resting spores (oospores) in infected roots produce large numbers of motile spores (zoospores) that swim about when soil moisture is high, infecting the tips of the young, fleshy roots and destroying their water- and food-conducting tissues. Infection and growth of the fungus in roots reduces the flow of water and nutrients to the developing leaves and fruit causing drought-like symptoms in the plant (Figure 1.34).

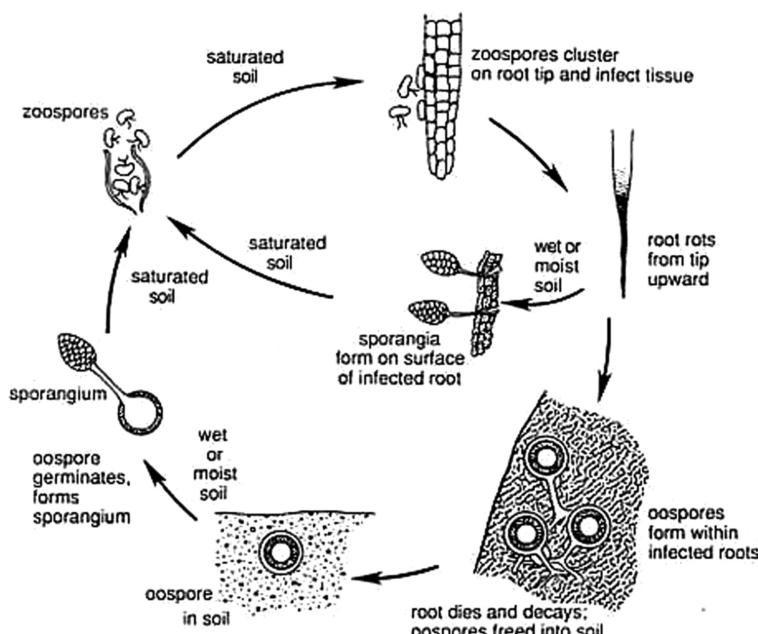


FIGURE 1.34 Disease cycle of red stele root rot on strawberry.

1.14.6 MANAGEMENT

The disease can be managed using the following strategies:

- Chemical control
- Biological control
- Cultural control

1.14.6.1 Chemical Control

Aliette WDG (Fosetyl-Al) and Ridomil Gold 480 EC (metalaxyl-M) are two very effective fungicides registered in Ontario for diseases caused by *Pythium* spp. and *Phytophthora* spp. such as red stele in strawberries. Although both fungicides are effective against root diseases caused by *Phytophthora* spp., they are very different in the way they control these pathogenic fungi and the way they move in plants.

Ridomil was originally targeted to protect crops from foliar diseases; however, it is now widely used for controlling many soilborne diseases as well. Ridomil Gold 480 EC acts on susceptible fungi by inhibiting RNA synthesis. The end result is that Ridomil Gold 480 EC interferes with the development and germination of *Phytophthora* spp.

Ridomil Gold 480 EC is very soluble in water and moves systemically up from roots into stems and then leaves with the transpiration stream of the plant. There is very little downward movement in plants, and therefore, it is important to apply this fungicide as a soil drench for best results against *Phytophthora* root diseases. Ridomil Gold 480 EC can be applied as a soil drench in the fall and the spring for strawberries. In fact, fall is the best time to apply Ridomil Gold 480 EC to control red stele in strawberries. Pay close attention to application timing and always read and follow the label. Ridomil Gold 480 EC should not be applied in the spring to plants bearing strawberries.

Aliette WDG, a phosphonate type of fungicide, on the other hand, is one of the first fungicides developed that can move both up and down in plants. On berries, Aliette is only registered for foliar applications. Aliette can be used as a drench to prevent *Phytophthora* root. Once inside the plant, the active ingredient fosetyl-al is broken down rapidly into phosphorous acid, which is extremely soluble in water and toxic to many *Phytophthora* species. Aliette works in two ways. It acts directly on the invading pathogens to stop their growth and sporangia or spore sack production. It also acts indirectly by stimulating the plant to activate its own defense system, thus helping to prevent future infections from taking place. Plants that have their defense system already activated prior to the invasion by a pathogen can defend much more effectively than plants that do not have their defense system pre-activated.

Regardless of the way these two effective fungicides work, these fungicides should never be used exclusively to control either red stele in strawberries or *Phytophthora* root rot in raspberries, blueberries, or apples. Ridomil Gold 480 EC and Aliette should be alternated with each other and be included as part of an integrated disease management system to reduce the potential of resistance developing.

1.14.6.2 Cultural Control

Since significant production and movement of infective zoospores occurs only during periods when the soil is completely saturated, the key to control is drainage. Strawberries should not be planted in low-lying or heavy soils where water accumulates or is slow to drain. On marginal soils, planting strawberries on beds raised at least ten inches high will bring much of the root system above the zone of greatest pathogen activity and the severity of red stele root rot should be significantly reduced.

The only practical method of controlling red stele is to grow certified, disease-free plants of resistant cultivars. Only resistant varieties should be planted in a field where red stele is known to have caused losses within the last five to ten years. Resistant cultivars include Darrow, Delite, Earliglow, Guardian, Midway, Pathfinder, Redchief, Redglow, Sparkel (Paymaster), Stelemaster, Sunrise, and

Surecrop. All of these cultivars are adapted to conditions in Illinois. However, not all are resistant in all infested soils because different races or strains of the fungus occur. These races vary in their ability to infect the different cultivars. A cultivar that is resistant to red stele in one area may be susceptible in another. Several races of *P. fragariae* have been found in Illinois. Earliglow, Guardian, Redchief, Sunrise, and Surecrop are the only resistant cultivars presently suggested for use in Illinois. They are resistant to three or more races of the fungus. Even these cultivars should be rotated with other crops to reduce the chance that a new, more virulent race of the fungus may appear) one that could attack resistant cultivars. Always plant small “trial” plots of new varieties to test them for resistance to red stele on your farm and to evaluate their performance before you make extensive plantings.

Whenever possible, select a planting site that has never had red stele, has good to excellent drainage, and is located where water from nearby land will not drain through it. Avoid low, wet spots.

If possible, use your own tools and machinery for setting out a strawberry field and carrying out general cultural practices. If you borrow equipment, be sure to clean off the soil and plant debris thoroughly before using it.

Soil fumigation with soil sterilants and/or pesticide applications may be helpful in situations where resistant varieties are not available or are not adapted. Extreme care should be taken not to reinfest a fumigated field by using contaminated equipment or plants. Soil fumigation should be the last resort in controlling the red stele disease. The first step is to use resistant varieties and a selection of well-drained planting sites.

It is important to minimize the chance of introducing the red stele fungus into a field where it does not already exist. Buy nursery stock only from a reputable supplier, and take care not to transfer soil on farm implements from an infested field into a clean one. New fungicides active against red stele also help in controlling this disease but are most effective when used in combination with good soil—water management practices.

1.14.6.3 Biological Control

After *in vitro* screening of more than 100 bacterial isolates from the rhizosphere on their antagonistic effect against *P. fragariae* var. *fragariae*, the causal agent of red stele disease of strawberry, three bacteria out of different genera *Raoultella terrigena* (G-584), *Bacillus amyloliquefaciens* (G-V1), and *Pseudomonas fluorescens* (2R1-7) were found with the highest inhibitory effect on the mycelial growth of both *Phytophthora* spp. For the management of the fungal disease, the antagonistic bacteria were further evaluated under greenhouse and field conditions. In the greenhouse all three bacteria were significantly effective in reducing red core, exhibited a similar level of control as the chemical fungicide Aliette of up to 59%. In field trials conducted at different locations in Germany under artificially and naturally infested soil conditions in two seasons, 2003–2005, different levels of biocontrol were performed by the tested bacteria. In trial during the first season under artificial conditions, the three rhizobacteria showed a significant control of up to 45% against the disease and in the next season, only *B. amyloliquefaciens* was effective against red stele. Under natural conditions, a significant effect of 37.5% was observed from a mixture of *R. terrigena* and *B. amyloliquefaciens* in the first season, and in the second season *R. terrigena* showed a significant effect of 45.1% in the northern part of Germany. In the south, *R. terrigena* and *B. amyloliquefaciens* were significantly efficient up to 51.5% and the overall effects were similar to Aliette.

1.15 PHYTOPHTHORA CROWN ROT

Phytophthora crown and root rot caused by *P. cactorum* is a disease of long-standing importance in strawberry. It is responsible for sporadic but serious production losses. Pre-plant soil fumigation, improved cultural practices, and systemic oomycete fungicides have helped to minimize the losses, but the pathogen's ability to survive indefinitely in soil and its capacity for rapid reproduction have prevented its eradication from strawberry production systems. The pathogen causes loss primarily by killing plants, but it also can reduce growth and yield through sub-lethal infections.

1.15.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>P. cactorum</i>	Crown rot and root rot	N/A

1.15.2 SYMPTOMS

Symptoms of disease caused by *P. cactorum* vary with the stage in the production system and the time of year. Early in the season, either at nurseries or fruiting fields, infected plants may exhibit stunting. As weather warms, the most notable symptom of infection, at least on susceptible cultivars, is plant collapse (Figure 1.35) associated with crown rot (Figure 1.36). However, it is difficult to reliably distinguish crown necrosis caused by *P. cactorum* from that induced by *C. acutatum* or other pathogens, especially in the later stages of disease. Furthermore, in the early stages after infection, crown rot caused by *P. cactorum* may be limited to outer regions or sectors of the plant crown. Diagnostic tests are required to determine with certainty which pathogen or pathogens are associated with the problem. At nurseries, *P. cactorum* causes runner lesions in addition to crown and root rot. Many of the roots of daughter plants infected by *P. cactorum* exhibit regions of dark necrosis, which may be limited to the outer (cortex) or extend into the inner (stele) portions of the root. The pathogen also can be carried on nursery stock, lacking clear symptoms of disease.

1.15.3 CAUSE AND DISEASE DEVELOPMENT

Of the *Phytophthora* species involved, *P. cactorum* is the most common; the others are much less prevalent on strawberry. *Phytophthora* is soilborne. When the soil becomes saturated with water, the pathogen can produce and release zoospores, which swim through water-filled pores to infect plant tissue. *Phytophthora* species also produce resilient spores (chlamydospores, oospores) that enable them to survive in soil for long periods without a host or under adverse conditions.

1.15.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Infections can occur during cool to moderate temperatures, which are typical throughout coastal fruit-production cycles.



FIGURE 1.35 Symptoms of *Phytophthora* crown and root rot caused by *Phytophthora cactorum*. Typical “plant collapse” in a commercial fruiting field.



FIGURE 1.36 Symptoms of *Phytophthora* crown and root rot caused by *P. cactorum*. Magnified crown rot.

1.15.5 DISEASE CYCLE

The most important propagule for this pathogen is zoospores, which originate from hyphae or germinating oospores and sporangia. In many cases, this pathogen may enter a field through infected transplants. Infection by *P. cactorum* usually occurs during warm periods with prolonged wetness.

Motile zoospores are released from sporangia during saturated soil conditions and enter through wounds. Once the zoospore reaches a host, it infects, and developing hyphae of the fungus colonize the host (Figure 1.37).

Disease expression is influenced by time of planting and environmental conditions. Plantings established in fall may have wilted plants soon after planting but it is possible the disease will not be expressed until the following spring after the pathogen has resumed activity.

1.15.6 MANAGEMENT

The key to effective management of disease caused by *P. cactorum* is integrated prevention. No single disease control measure is completely effective for management of *Phytophthora* crown rot in strawberry cultivars highly susceptible to the pathogen, but combined approaches can be very effective. Effective pre-plant soil fumigation helps to insure production of clean nursery stock and minimize the risk of infection in fruiting fields. Pre-plant fumigation with mixtures of methyl bromide and chloropicrin (MB: CP) typically kill all or most inoculum of the pathogen at soil depths in the surface 2–3 ft of soil. Trials indicated that CP or mixtures of 1,3-dichloropropene (1,3-D) and CP can approach the effectiveness of MB: CP, but only at rates of at least 300 lb/A. With drip applications, drip-line placement and water application amount during the fumigation can critically influence effectiveness of CP and 1,3-D: CP for control of *P. cactorum* in soil. Because these fumigants have less diffusion potential than MB, uniform wetting of the bed is essential for effective control of the inoculum. Virtually impermeable film (VIF) properly applied over plant beds can significantly reduce fumigant emissions to the atmosphere and improve control of weed seeds

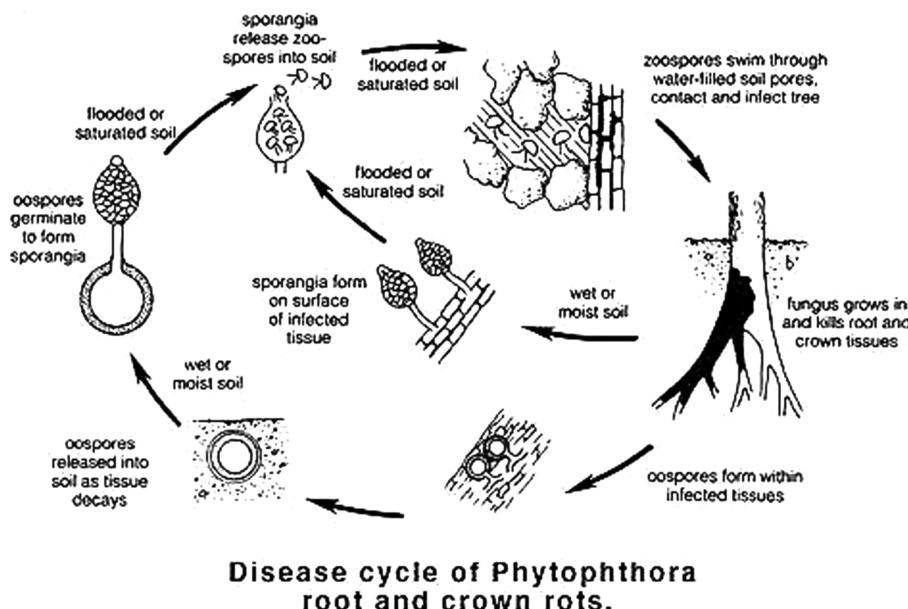


FIGURE 1.37 Disease cycle of *P. cactorum*.

and pathogen inoculum near the soil surface; however, control of *P. cactorum* and other soilborne inoculum at soil depths of 1 ft or more was not significantly improved by VIF (Table 1.5).

1.16 RHIZOCTONIA ROOT ROT

Rhizoctonia Root Rot, also known as black root rot of strawberries is a very complex and serious disease that has been reported in strawberry fields around the world. Several pathogenic organisms have been associated with the disease; however, the soilborne fungus *Rhizoctonia fragariae* is probably the most frequently isolated pathogen from strawberry roots exhibiting symptoms of black root rot.

1.16.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>R. fragariae</i>	Black root rot	Major

1.16.2 SYMPTOMS

Affected plants may be scattered throughout a strawberry planting or grouped in one or more parts of it. Plants with black root rot are less vigorous than normal plants and produce fewer runners. Individual or groups of leaves may wilt, discolor, and die. Entire plants may die when black root rot is severe. Affected plants should be carefully dug up (not pulled) and their root systems examined. Plants with black root rot will exhibit 1 or more of the following root symptoms:

- Root system smaller than in normal plants.
- Main root with lesions—these are darker than the rest of the root.

TABLE 1.5

Effects of Treatments with Aliette or Ridomil on Productivity of Two Strawberry Cultivars in Non-Infested Soil and Soil Infested with *Phytophthora cactoruma*^a

Strawberry Cultivar	Soil Treatment ^b	Chemical Treatment Program ^c	Marketable Yield (Total Grams per Plant)
Diamante	Infestation w/ <i>P. cactorum</i>	• Aliette plant dip and spray	1031
		• Water control plant dip and spray	572
		• Ridomil soil drench	1163
		• Water control soil drench	659
	Non-infested control	• Aliette plant dip and spray	1113
		• Water control plant dip and spray	1097
		• Ridomil soil drench	1172
		• Water control soil drench	1128
Aromas	Infestation w/ <i>P. cactorum</i>	• Aliette plant dip and spray	1388
		• Water control plant dip and spray	938
		• Ridomil soil drench	1400
		• Water control soil drench	891
	Non-infested control	• Aliette plant dip and spray	1481
		• Water control plant dip and spray	1250
		• Ridomil soil drench	1463
		• Water control soil drench	1384

^a From a field trial at Monterey Bay Academy in 2001/02.

^b After pre-plant fumigation with methyl bromide-chloropicrin mixture, the soil treatments were applied to each planting hole in 100 ml of V8 juicecoat- vermiculite medium that was either permeated with *P. cactorum* (the infestation treatment) or sterile (the non-infested control).

^c The pre-plant dip and spray treatments with Aliette were applied at maximum label rates; one pre-plant dip and five foliar sprays were applied over the growing season. The drench program with Ridomil simulated drip chemigation with the material; the maximum label rate was used, with one treatment applied at planting and two more applied during the growing season.

- Feeder roots are lacking.
- Feeder roots with dark zones or lesions.
- All or part (usually the tip) of main roots killed. A cross-section of a dead root shows it is blackened throughout (Figures 1.38 and 1.39).

1.16.3 CAUSE AND DISEASE DEVELOPMENT

One or more of the following factors may be involved in black root rot problems: soil fungi (e.g., *Rhizoctonia*, *Fusarium*, *Pythium*) nematodes (microscopic round worms), winter injury, fertilizer burn, drought, excess salts, herbicide injury, wet soils, or pH imbalance. In some cases, environmental factors may predispose plants to an attack by the root rotting fungi.

1.16.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Wet soils, excess salt conditions of the soil, pH imbalance, and winter injury favor the development of the disease.



FIGURE 1.38 Strawberry plant dying from black root rot.

1.16.5 MANAGEMENT

There are no cures or guaranteed controls for black root rot. Control measures center around proper planting and care of strawberry plants. The following strategies can be followed:

- Use only healthy, white-rooted strawberries when planting.
- Plant in well-drained soils.
- Maintain plant vigor with adequate fertilization and cultivation.
- Irrigate strawberries during dry periods.
- Fumigation may be practical for commercial plantings. It is not recommended for home plantings.



FIGURE 1.39 Black root rot developing on primary and feeder roots of strawberry.

1.17 CHARCOAL ROT OF STRAWBERRY

Charcoal rot, caused by *Macrophomina phaseolina*, is a relatively new disease in Florida. This disease was first observed in December 2001, when collapsed and dying strawberry plants from a commercial field were submitted to our diagnostic clinic. During the 2003–2004 season, *M. phaseolina* was isolated from dying strawberry plants from the original field and two additional farms. Since then, a few additional samples are received in our diagnostic clinic every season. Affected plants are often found along field margins or other areas that were inadequately fumigated with methyl bromide. Charcoal rot has also been reported on strawberry in France, India, and Illinois.

1.17.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>M. phaseolina</i>	Wilt and Crown Rot	Minor

1.17.2 SYMPTOMS

Symptoms caused by *M. phaseolina* are similar to those caused by other crown-rot pathogens such as *Colletotrichum* and *Phytophthora* species. Plants initially show signs of water stress and subsequently collapse (Figure 1.40). Cutting the crowns of affected plants reveals reddish-brown necrotic areas on the margins and along the woody vascular ring (Figure 1.41). To confirm a diagnosis, a sample must be submitted to a Diagnostic Clinic and the pathogen must be isolated from the diseased crowns and identified.

1.17.3 CAUSE AND DISEASE DEVELOPMENT

Very little is known regarding this disease on strawberries. *M. phaseolina* is a common soilborne pathogen in many warm areas of the world and has a very broad host range. Many vegetable crops planted as second crops after strawberry such as squash, cantaloupe, and peppers, legumes, and others are susceptible. Those infections may increase inoculum levels of *M. phaseolina* in the soil in the off-season for strawberries.

1.17.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

In general, high temperatures and low soil moisture favor infection and disease development.



FIGURE 1.40 Plant wilt symptom of charcoal rot.



FIGURE 1.41 Internal crown symptoms of charcoal rot.

1.17.5 MANAGEMENT

No fungicides are labeled for control of charcoal rot on strawberries. Topsin M® is labeled for control of charcoal rot on other crops. Preliminary results with Topsin M® have shown that application of this product may delay the onset of symptoms. Studies are currently being conducted to determine if cultivars differ in susceptibility to charcoal rot. This disease may be an emerging threat as the Florida strawberry industry makes the transition from methyl bromide to other fumigants.

1.18 GNOMONIA FRUIT ROT AND LEAF BLOTCH

Gnomonia fruit rot, leaf blotch, and stem-end rot caused by *Gnomonia comari* P. Karst. (anamorph, *Zythia fragariae* Laibach) were observed in a strawberry fruit production field at Watsonville, CA, in 1996. *Z. fragariae* has been known for years to attack leaves and cause leaf blotch but this is the first time that the perfect stage, *G. comari*, was identified and documented to infect fruits and cause stem-end rot in California.

1.18.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>G. comari</i>	Fruit Rot, Leaf Blotch, Stem-End Rot	Serious

1.18.2 SYMPTOMS

Plants become infected between flowering and harvesting. Figure 1.42 shows how the fruiting bodies of the fungus develop on trash and from there the spores are produced that infect the next crop. Figure 1.43 shows very early symptoms of the disease on leaves. It is important to start controlling the disease at this stage. The fungus first infects the calyx (see Figure 1.44), and disease spreads into the fruit as a rot. Both green and ripe fruit may be infected. Infected fruit ripens early and turns pale red to brown. They remain firm but are often invaded by other fruit rots such as gray mold (Figure 1.45).



FIGURE 1.42 Black “dots” are the fruiting bodies of the fungus on strawberry residue. Spores infect the next crop.



FIGURE 1.43 Early symptoms of infection on a leaf (small lesions on the underside).



FIGURE 1.44 The start of stem-end rot in fruit caused by *Gnomoniopsis* (on the tips of the calyx).

1.18.3 CAUSE AND DISEASE DEVELOPMENT

Trash from previous and current strawberry crops left in the soil is the source of infection. Planting material can carry the spores systemically, but levels are usually quite low, and it is unlikely that runners are a significant source of infection (Figure 1.46).



FIGURE 1.45 Leaf symptoms of *Gnomoniopsis*.

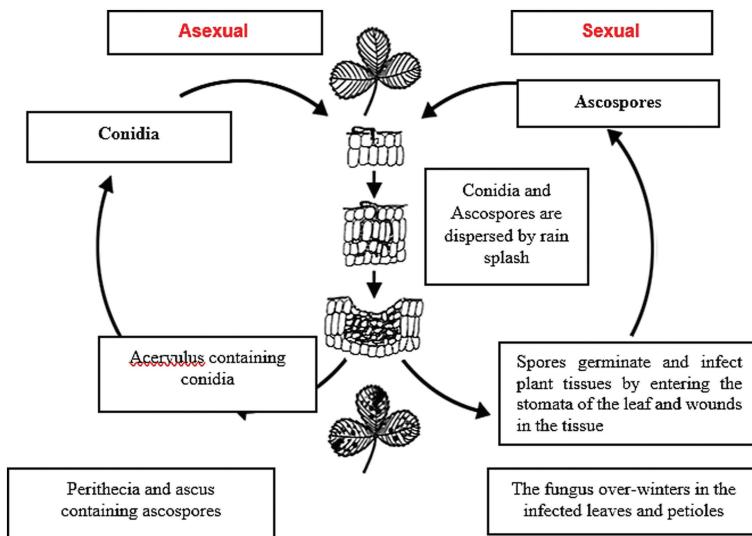


FIGURE 1.46 Life cycle of *G. comari*.

1.19 LEAF SPOT OF STRAWBERRY

Leaf spot is one of the most common and widespread diseases of strawberry. *M. fragariae* (asexual stage *Ramularia tulasnei* Sacc.) is also the cause of black seed disease on strawberry fruit, which occurs occasionally in North America where *Mycosphaerella* leaf spot is present. Prior to the development of resistant cultivars and improved control programs, leaf spot was the most economically important strawberry disease.

1.19.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>M. fragariae</i>	Leaf Spot. Also Affects the Petioles, Stolons, and Fruits	Low

1.19.2 SYMPTOMS

1.19.2.1 Leaves

Leaf symptoms vary with strawberry cultivar, the strain of the fungus causing disease, and environmental conditions. Leaf lesions or “spots” are small and round (3–8 mm diameter), dark purple to reddish in color, and are found on the upper leaf surfaces (Figure 1.47). The center of the spots becomes tan to gray to almost white over time, while the broad margins remain dark purple. Lesion centers on younger leaves stay light brown, with a definite reddish purple to rusty brown margin. Numerous spots may coalesce and cause the death of the leaf. Large, spreading lesions that involve large portions of the leaflet are formed on some highly susceptible cultivars; the centers of which remain light brown. In warm humid weather, typical solid rusty brown lesions without purple borders or light-colored centers may form on young leaves. Lesions are evident on the undersurface of the leaf but are less intense in color, appearing as indistinct tan or bluish areas.

1.19.2.2 Leaf Stems (Petioles), Runners, Fruit Stalks (Pedicels), Berry Caps (Calyxes)

Symptoms are almost identical to those on leaves, except for fruit. Only young tender plant parts are infected by this pathogen (Figure 1.48).

1.19.2.3 Fruit

Superficial black spots (6 mm in diameter) form on ripe berries under moist conditions. These spots surround groups of seeds (achenes) on the fruit surface. The surrounding tissue becomes brownish black, hard, and leathery. The pulp beneath the infected area also becomes discolored; however, no general decay of the infected berry occurs. Usually, only one or two spots occur on a berry, but some may have as many as eight to ten “black-seeds” (Figure 1.49). Symptoms are most conspicuous on white, unripe fruit and on ripe fruit of light-colored cultivars. Economic losses in this case are due to the unattractiveness of “black seed” spots on fruit, rather than fruit rot.



FIGURE 1.47 Typical foliar symptoms of leaf spot on strawberry leaves.



FIGURE 1.48 *M. fragariae*, pathogen of strawberry, leaf spot on sepals.

1.19.3 CAUSE AND DISEASE DEVELOPMENT

M. fragariae overwinters in lesions in old leaves and produces its first spores in about mid-May. The spores fall on other leaves and germinate when it rains. After an incubation period ranging from 15 to 30 days, new spots appear and produce new spores that infect other young leaflets. This cycle can be repeated several times during a single growing season. Strawberry leaf spot is spread by water. During rainfall events or spray irrigation, the water droplets that make contact with the leaves tear spores away from the lesions and project them onto new leaves. Unlike several other species of fungi, the spores are not transported by the wind, limiting their propagation. Heavy, frequent downpours can, however, result in epidemic outbreaks of the disease.

1.19.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Leaf spot may reach economic threshold levels, provided young leaves and inoculum are present, under conditions of high temperatures and long periods of leaf wetness. Research results show most severe infection of young leaves to occur during periods of leaf wetness from 12 to 96 hours when temperatures fall in the range of 15°C–20°C. This data suggests fungicide treatments should be applied in early spring and after renovation of plantings if inoculum was present.

1.19.5 DISEASE CYCLE

In the south, perithecia and sclerotia are absent. Conidia are produced in small dark fruiting bodies (pseudothecia) within leaf lesions and serve as inoculum. In this instance, infection is a continuous



FIGURE 1.49 Black seed symptoms on strawberry.

process with older lesions producing conidia to infect young leaves during each season. Conidia landing on leaf surfaces produce germ tubes which penetrate through natural leaf openings (stomata) on the upper and lower surfaces of leaves. New conidia are produced on clusters (fascicles) of conidiophores which grow out through stomata. These are carried to new leaves by rain splash, and the disease cycle begins again (Figure 1.50).

In northern growing regions, the life cycle is somewhat different. Three sources of primary inoculum may be present: conidia overwintering on living leaves, conidia from overwintering sclerotia, and ascospores. Abundant conidia, produced in early summer on lesions on both upper and lower leaf surfaces and lesions on other plant parts, are spread primarily by water splash. High rainfall can lead to disease of epidemic proportions. Sclerotia are produced profusely during the winter on dead infected leaves. These may also produce abundant conidia in the spring. Conidia also develop on occasion from the bases (apices) of perithecia. Perithecia are produced primarily on upper surfaces of overwintered leaves. From these, perithecia are wind disseminated. It is not known if these serve as an important source of primary inoculum, but they are most probably a means by which genetically different strains of the fungus may travel long distances. *M. fragariae* establishes in the stigma at the time of flowering and then grows to the achene. From there it infects surrounding berry (receptacle) tissue. Conidia produced in leaf infections are probably the primary inoculum source for fruit infections.

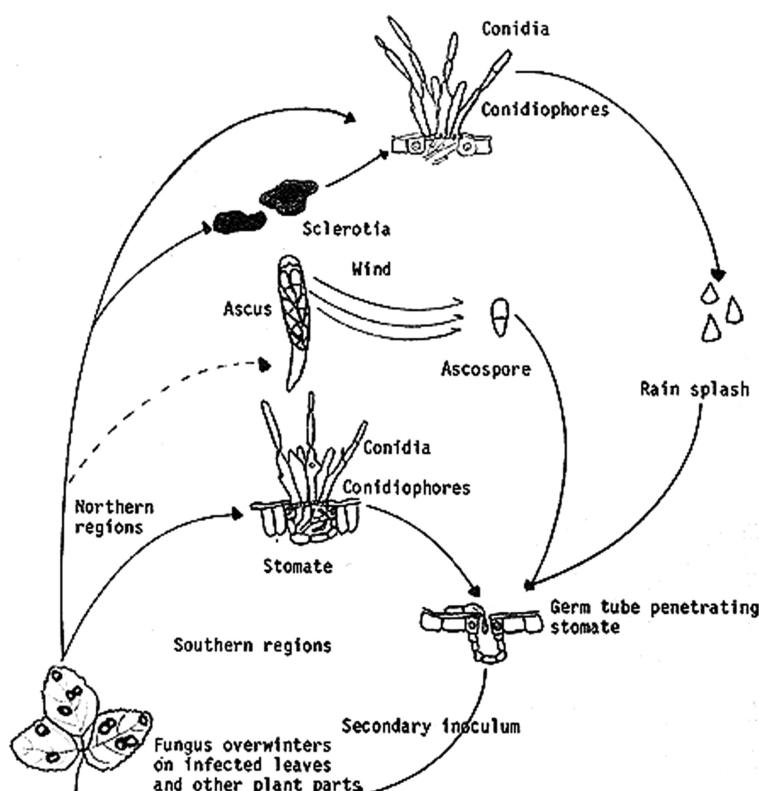


FIGURE 1.50 Disease cycle of *M. fragariae*.

1.19.6 MANAGEMENT

1.19.6.1 Chemical Control

Keep varying the fungicides used to prevent or promote the development of resistance. Bear in mind that too many applications of any pesticide can lead to the development of resistance. Captan, Folpet, Dodine, and copper are a few of the fungicides recommended by Réseau d'avertissements phytosanitaires (RAP). It is recommended that copper be applied (in the form of tribasic copper sulfate) during the year of harvest. It is important not to abuse this metal since it accumulates in the soil and could reach toxic concentrations.

1.19.6.2 Cultural Control

1.19.6.2.1 Scouting for the Disease

Despite all precautions, it is impossible never to have problems with leaf spot. The provisional scouting method for leaf spot approved by the RAP consists in observing 100 leaflets at random (one leaflet per plant) twice—once at the end of September of the planting year and again during flowering in the first growing season. In the spring, treatment is not recommended if less than 25% of the old leaves have symptoms the previous fall, and, during the growing season, if less than 10% of the new leaflets are infected at flowering.

1.19.6.2.2 Umbrella Effect

If the scouting results indicate that control measures are required, preventive measures should be taken. The threshold limit value for leaf spot is higher than that for gray mold rot, which attacks the fruit directly. However, recent Quebec studies reveal that owing to the method of infection of *M. fragariae*, the best strategy is to obtain the “umbrella effect”. The “umbrella” strategy is based on the fact that only young strawberry leaflets are susceptible to the disease. It is important, therefore, to protect the young foliage through fungicide applications during the rapid growth period of strawberries. The foliage must then be protected until the plant has formed a few leaves and these leaves have passed the stage at which they are susceptible to the disease. The leaves at the very top of the plant, which are free of disease, protect the new leaves, hence the name “umbrella”. This prevents spores on infected leaves from contaminating the smaller leaves located below by dripping rainwater. When the crop is mowed, the same approach must be taken, i.e., to treat until the umbrella effect is obtained.

1.19.6.3 Preventive Treatment

M. fragariae requires water to produce infection. When treatment is necessary, it is highly recommended that preventive treatment be carried out when weather forecasts call for rain within 24 hours.

The following materials are listed in order of usefulness in an IPM Program, considering efficacy. Also, consider the general properties of the fungicide as well as information relating to environmental impact. Not all registered pesticides are listed. Always read the label of the product being used (Table 1.6).

1.20 BACTERIAL LEAF SPOT OF STRAWBERRY

Bacterial leaf spot, also known as Angular Leaf Spot is a bacterial disease caused by *Xanthomonas fragariae*, a pathogen highly specific to both the wild and the cultivated strawberry, *F. ananassa*. Bacterial angular leaf spot disease on strawberries has been increasing in importance to strawberry producers in recent years because it is spread by infected but asymptomatic strawberry plantlets used in annual row culture systems. Until now there have been no control methods for the disease

TABLE 1.6
Preventive Treatment

Common Name (Trade Name)	Mode of Action Group Name (Number) ^a	Amount/ Acre ^a	R.E.I. ^b (Hours)	P.H.I. ^b (Days)
Chlorothalonil (BravoWeatherStik)	Chloronitrile (M5)	1.5 pt	12	N.A.
Myclobutanil (Rally) 40W	Demethylation inhibitor (3)	2.5–5 oz	24	0
Triflumizole (Procure) 50WS	Demethylation inhibitor (3)	4–8 oz	12	1

N.A. Not applicable.

Group numbers are assigned by the Fungicide Resistance Action Committee (FRAC) according to different modes of actions (for more information, see <http://www.frac.info/>). Fungicides with a different group number are suitable to alternate in a resistance management program. In California, make no more than one application of fungicides with mode of action Group numbers 1, 4, 9, 11, or 17 before rotating to a fungicide with a different mode of action Group number; for fungicides with other Group numbers, make no more than two consecutive applications before rotating to fungicide with a different mode of action Group number.

^a Apply all materials in 200-gal water/acre to ensure adequate coverage.

^b Restricted entry interval (R.E.I.) is the number of hours (unless otherwise noted) from treatment until the treated area can be safely entered without protective clothing. Preharvest interval (P.H.I.) is the number of days from treatment to harvest. In some cases, the REI exceeds the PHI. The longer of two intervals is the minimum time that must elapse before harvest.

and no resistant varieties. Four genetically distinct strains of the pathogen, *X. fragariae*, have been identified and used to screen a collection of 81 strawberry accessions for resistance to the pathogen.

1.20.1 CAUSAL ORGANISM

Species	Associated Disease Phase	Economic Importance
<i>X. fragariae</i>	Leaf Spot	High

1.20.2 SYMPTOMS

Infection first appears as minute, water-soaked spots on the lower surface of leaves. The lesions enlarge to form translucent, angular spots that are delineated by small veins and often exude a viscous ooze of bacteria and bacterial exudates, which appear as a whitish and scaly film after drying. As the disease progresses, lesions coalesce and reddish-brown spots, which later become necrotic, appear on the upper surface of the leaves. A chlorotic halo usually surrounds the infected area. *X. fragariae*, the causal agent of ALS, is a slow-growing, gram-negative bacterium that produces water-soaked lesions on the lower leaf surfaces (Figure 1.51). The bacteria enter the leaf through the stomata (tiny pores that are most abundant on the lower surface of the leaf). Lesions begin as small and irregular spots on the undersurface of the leaflets. When moisture is high on the leaves, lesions ooze sticky droplets of bacteria. As the disease develops, lesions enlarge and coalesce to form reddish-brown spots, which later become necrotic (Figure 1.52).

A practical way to recognize the disease is to place the leaves against a source of background light where the translucent spots can be seen (Figure 1.53). The tissue with older damage eventually dies and dries up, giving leaves a ragged appearance.

During severe epidemics, the pathogen can also cause lesions on the calyx of fruit that are identical to foliar lesions (Figure 1.54). When severe, these calyxes can dry up and make the fruit unmarketable.



FIGURE 1.51 Water-soaked lesions of angular leaf spot.



FIGURE 1.52 Reddish-brown spots of angular leaf spot.



FIGURE 1.53 Translucent spots of angular leaf spot.



FIGURE 1.54 Water-soaked lesions of angular leaf spot on the calyx.

1.20.3 CAUSE AND DISEASE DEVELOPMENT

The primary source of inoculum in a new field is contaminated transplants. Secondary inoculum comes from bacteria that exude from lesions under high moisture conditions. Bacteria can survive on dry infested leaves and tissue buried in the soil for up to 1 year. The pathogen can be spread easily by harvesting operations when wet and cool conditions favor the production of bacterial exudate. The pathogen also can be dispersed by rain and overhead sprinkler irrigation. If the disease invades the vascular system of the plant, the disease will be difficult to control. Affected plants may wilt and die.

1.20.4 FAVORABLE CONDITIONS OF DISEASE DEVELOPMENT

Not much is known in this respect, and more research is underway to determine which conditions are most favorable for disease development and spread. Some report moderate to low daytime temperatures and nighttime temperatures below freezing are needed. Most researchers agree high humidity is also a key factor. The development of the disease seems favor by warm days (20°C) and cold nights (-2°C – 4°C).

The primary source of inoculum in a new field is contaminated transplants. Secondary inoculum comes from bacteria that exude from lesions under high moisture conditions. Bacteria can survive on dry infested leaves and tissue buried in the soil for up to one year.

The pathogen can be spread easily by harvesting operations when wet and cool conditions favor the production of bacterial exudate. The pathogen also can be dispersed by rain and overhead sprinkler irrigation.

1.20.5 DISEASE CYCLE

Inoculum for primary spring infections in new growth comes primarily from infected transplants or systemically infected overwintered plants and dead leaves. This bacterium is resistant to adverse conditions such as desiccation and can survive for long periods in dry leaf debris or buried leaves in soil.

Bacteria exuded from the undersides of leaves under high moisture conditions serve as the secondary source of inoculum in plantings. Angular leaf spot (ALS) bacteria are carried from plant to plant by splashing water from rain or overhead irrigation, as well as harvesting operations. The motile bacterial cells may enter the plant through drops of dew, guttation droplets, and rain or irrigation water.

1.20.6 MANAGEMENT

The best way to control ALS is to use pathogen-free transplants. Since this is not always possible, growers should avoid harvesting and moving equipment through infested fields when the plants are wet. Minimizing the use of overhead sprinklers during plant establishment and for freeze protection also reduces the spread of the disease. The use of surfactant-type spray adjuvants should also be avoided when ALS is a threat since these products often help bacteria penetrate through the stomata and may enhance disease development.

Copper-based products can provide effective control of the disease in some instances, but low rates of copper should be used since phytotoxicity (reddening of older leaves, slow plant growth, and yield decrease) has been documented with repeated sprays. Many copper products are labeled for ALS control on strawberry, such as copper hydroxide, copper oxychloride, basic copper sulfate, cuprous oxide, and various other copper compounds. These active ingredients suppress ALS, but it is important to apply the correct amount. Trial results have shown that preventive, weekly applications of copper fungicides at 0.3 lb of *metallic copper* per acre were effective in reducing disease symptoms without causing phytotoxicity on the plants. However, trial results have also shown that when disease pressure is low to moderate, the use of copper sprays did not significantly increase yield. Copper products can increase yield and decrease the possibility of fruit rejection only when environmental conditions are highly favorable for infection and spread.

Many other products have been tested over the years in the search for an alternative to copper. Actigard®, a plant-resistant activator manufactured by Syngenta, has been shown to suppress ALS. Actigard® is used to control bacterial spot disease on tomatoes in Florida, but it is not currently approved for use on strawberry.

1.21 THE DAGGER NEMATODE OF STRAWBERRY

The Dagger Nematode, also known as American Dagger Nematode, is one of many species of the genus *Xiphinema*. The common name “Dagger Nematode” applies to all species of the genus. *X. americanum* was first described in 1913 by N. A. Cobb, who had recovered it from the roots of corn, grass and citrus found growing on both the “Atlantic and Pacific slopes of the United States”. Found in both agricultural and forest soils, *X. americanum* has been referred to as the most destructive plant-parasitic nematode in America. It has been reported that many nematodes identified as *X. americanum* from various parts of the world are probably a number of different closely related species.

1.21.1 DISTRIBUTION OF THE CAUSAL ORGANISM

X. americanum has a worldwide distribution. It has been found in Canada, the United States, Mexico, Central and South America, and in the Caribbean Islands; has also been recorded from Africa, Japan, India, parts of Europe, Australia, U.S.S.R., and Pakistan.

1.21.2 SYMPTOMS

The symptoms that plants exhibit in response to the pathogenicity of *X. americanum* are similar to those of other migratory ectoparasitic nematodes of roots. It is common to see poor growth and

Strawberry

- 1 Ellis, M. A. , and Erincik, O. , 2016. Anthracnose of Strawberry, Ohio: Department of Plant Pathology, The Ohio State University. <https://ohioline.osu.edu/factsheet/plpath-fru-16> (Apr 15, 2016).
- 2 Ellis, M. , 2012. Fungicides for Strawberry Disease Control, Wooster OH: Department of Plant Pathology, The Ohio State University OARDC.
- 3 Converse, R. H. , Martin, R. R. , and Spiegel, S. , 1987. Strawberry mild yellow-edge. In: Virus Diseases of Small Fruits, Ed. by Converse, R. H. , Agriculture Handbook No. 631, Washington DC, USA: US Department of Agriculture, pp. 25-9.4.Tzanetakis I. E., Mackey I. C., Martin R. R., 2004. Strawberry necrotic shock virus is a distinct virus and not a strain of Tobacco streak virus. *Arch Virol.* 2004 Oct, 149(10): 2001–11.
- 5 Ries, S. M. , 1996. Reports on Plant Diseases: Verticillium Wilt of Strawberry, IL: University of Illinois. <http://ipm.illinois.edu/diseases/series700/rpd707>, June 1996.
- 6 University of Illinois Extension, 1999. College of Agricultural, Consumer and Environmental Sciences , Reports on Plant Disease, RPD No. 1103, May 1999.

Tomato

- 1 Miller S., and Huang R. , Bacterial Canker of Tomato. https://www.lincolnu.edu/c/document_library/get_file?uuid=585aefcc-b271-46d3-878e-0ab895232094&groupId=145912.
- 2 Sikora E. J. , 2011. Virus Diseases of Tomato, Extension Plant Pathologist, Associate Professor, Plant Pathology, Auburn University: Auburn, AL, June 2011.
- 3 Nelson S. C. , 2008. Late Blight of Tomato (*Phytophthora infestans*), Honolulu, Hawai'i: Department of Plant and Environmental Protection Sciences, CTAHR University of Hawai'i at Mānoa, August 2008.
- 4 Reddy P. P. , 2014. Biointensive Integrated Pest Management in Horticultural Ecosystems, Springer Publications: New York, NY, pp. 83.
- 5 Smith, I. M. , Dunez J. , Phillips D. H. , Lelliott R. A. , and Archer S. A. , eds. 1988. European Handbook of Plant Diseases, Oxford: Blackwell Scientific Publications, pp. 583.
- 6 Agrios, G. N. , 1988. Plant Pathology, 3rded. New York: Academic Press, Inc., pp. 803.
- 7 McGovern, R. J. , and Datnoff L. E. , 1992. Fusarium crown and root rot: Reevaluation of management strategies. Proceedings of the Florida Tomato Institute, (FTI'1992), Gainesville, FL: University of Florida-IFAS, pp. 75–86.
- 8 Ozbay N. , and Newman S. E. , 2004. Fusarium crown and root rot of tomato and control methods. *Plant Pathology Journal*, 3: 9–18.
- 9 Aycoc k R., 1966. Stem rot and other diseases caused by *Sclerotium rolfsii*. *Tech. Bull.*, 174. Raleigh, NC: North Carolina State University Agricultural Experiment Station.
- 10 Leeper, P. W., Phatak, S. C., and George, B. F., 1992. Southern blight-resistant tomato breeding lines: 5635M, 5707M, 5719M, 5737M, 5876M, and 5913M. *Hortscience* 7: 475–478.
- 11 Smith, I. M. , Dunez J. , Phillips D. H. , Lelliott R. A., and Archer S. A., eds. European handbook of plant diseases, Oxford, United Kingdom: Blackwell Scientific Publications. pp., 583 1988.
- 12 Louter, J. H., and Edgington, L. V., 1985. Cross protection of greenhouse tomato against fusarium crown and root rot. *J. Plant Pathol.*, 7: 445–446.

Citrus

- 1 Dewdney, M. M. , and Timmer, L. W. 2009. Alternaria Brown Spot. University of Florida, 2010. http://gardener.wikia.com/wiki/Alternaria_brown_spot
- 2 Dewdney, M. , UF/IAF Citrus Extension, University of Florida. http://www.crec.ifas.ufl.edu/extension/black_spot/citrus_black_spot.shtml
- 3 Menge, J. A. , and Ohr H. D. , Plant Pathology, UC Riverside, UC IPM Pest Management Guidelines: Citrus, UC ANR Publication 3441. <http://ipm.ucanr.edu>
- 4 Adaskaveg, J. E. , Management of Citrus Brown Rot, Department of Plant Pathology, University of California Riverside, C. A. <http://www.calcitrusquality.org/wp-content/uploads/..Citrus-Brown-Rot-JA-9-29-11.pdf>
- 5 Based on blog written by Karen Harty, Florida Master Gardener and Citrus Advisor. <https://growagardener.blogspot.com/2014/01>
- 6 Brlansky, R. H. , Damsteegt, V. D. , and Hartung, J. S. 2002. Transmission of the citrus variegated chlorosis bacterium *Xylella fastidiosa* with the sharpshooter *Oncometopia nigricans*. *Plant Dis.* 86:1237–1239. <https://pubag.nal.usda.gov/pubag/downloadPDF.xhtml?id=741&content=PDF>

Apple

- 1 Peter, K. A. , PH.D., "Apple Disease - Alternaria Leaf Blotch", Pennsylvania State University Extension, October 17, 2017. <https://extension.psu.edu/apple-disease-alternaria-leaf-blotch>
- 2 Central Science Laboratory , Sand Hutton, York, "Alternaria Blotches on Apple and Pear EC Listed Diseases". <https://www.adlib.ac.uk/resources/000/193/618/alternaria-defra.pdf>
- 3 Biggs, A. R. , "Apple Scab", West Virginia University, August 14, 2013. <http://articles.extension.org/pages/66202/apple-scab>
- 4 Steiner, P. W. , University of Maryland and Biggs A. R., West Virginia University, "Fire Blight", 1998. http://www.caf.wvu.edu/Kearneysville/disease_month/fireblight.html
- 5 Ellis, M. A. , "Sooty Blotch and Fly Speck of Apple", Department of Plant Pathology, Ohio State University Extension, April 13, 2016. <https://ohioline.osu.edu/factsheet/plpath-fru-41>
- 6 Grimova, Lenka , Winkowska, Lucie , Konrady, Michal and Rysanek, Pavel , "Apple Mosaic Virus", *Phytopathologia Mediterranea* (2016) 55, 1, pp. 1–19.
- 7 Zwet, T. van der , Yoder, K. S. , and Biggs, A. R. , "Blister Spot of Apple", Mid-Atlantic Orchard Monitoring Guide (NRAES-75), August 30, 2011. <http://articles.extension.org/pages/60624/blister-spot-of-apple>
- 8 Home Orchard Society , "Bitter Pit: Cause And Control". <http://www.homeorchardsociety.org/growfruit/trees/bitter-pit-cause-and-control.9.de>
- Sequeira, O.A. and Posnette, A.F. (1969). Commonw. Bur. Hort. Pl. Crops. Tech. Commun. No. 30, Suppl. 2/3/4, 76a.
- 10 Mink, G. I., and Shay, J. R., 1962. Latent viruses of apple. *Purdue Agric. Exp. Stn. Res. Bull.* 756.

Banana

- 1 Proceedings of the workshop on review of the strategy for the "Management of banana Xanthomonas wilt", Bioversity International pp. 84–87, 2009
- 2 Mwangi, M. , "Responding to Banana Xanthomonas Wilt Amidst Multiple Pathogens and Pests", Crop Crisis Control Project, 2007
- 3 Gaur, R. K. , Hohn, T., and Sharma, P. , "Plant Virus–Host Interaction Molecular Approaches and Viral Evolution", Cambridge: Academic Press, 2014.
- 4 Lockhart, B. E. , "Banana Streak Badnavirus Infection in Musa: Epidemiology, Diagnosis and Control", Department of Plant Pathology, St. Paul, USA: University of Minnesota.
- 5 Jeyakumar, P. , Paper on "Physiological Disorders in Fruit Crops", Assistant Professor (Crop Physiology), Department of Pomology Horticultural College and Research Institute Tamil Nadu Agricultural University, Coimbatore, India.
- 6 Viljoen, A. , Mahuku, G. , Massawe, C. , et al., "Banana Pests and Diseases Field Guide for Disease Diagnostics and Data Collection", International Institute of Tropical Agriculture, 2016.
- 7 Richard, B. , and David, C. , "Scientific Opinion of the Panel on Plant Health", *The EFSA Journal* 652, 5–21, 2008.

Pepper

- 1 Information referenced from Pessl Instruments "Anthracnose Fruit Rot, Colletotrichum". http://docs.metas.at/Anthracnose+Fruit+Rot%2C+Colletotrichum?structure=Disease+model_en
- 2 Ritchie , D.F. 2000. "Bacterial spot of pepper and tomato", The Plant Health Instructor. <https://www.apsnet.org/edcenter/intropp/lessons/prokaryotes/Pages/Bacterialspot.aspx>
- 3 Sun X. , Nielsen, M. C. and Miller, J. W. , 2002, "Bacterial Spot of Tomato and Pepper", Plant Pathology Circular No. 129 (Revised).
- 4 Cerkauskas R. , 2004, "Cercospora Leaf Spot", AVRDC – The World Vegetable Center.
- 5 Xie C. and Vallad, G. , 2009, "Integrated Management of Southern Blight in Vegetable Production", University of Florida IFAS Extension. <http://edis.ifas.ufl.edu/pp272>
- 6 Bhat, R. G. , Smith, R. F. , Koike, S. T. , Wu, B. M. , and Subbarao, K. V. 2003. "Characterization of *Verticillium dahliae* isolates and wilt epidemics of pepper", *APS Journal*. <https://apsjournals.apsnet.org/doi/pdf/10.1094/PDIS.2003.87.7.789>

Potato

- 1 Inglis, D. A. , Schroeder, B. K. , 2011, "Bacterial Soft Rot and Lenticel Spot on Potato Tubers", Washington State University Extension.
- 2 Elphinstone, J. G. , 1987, "Soft Rot and Blackleg of Potato", Technical Information Bulletin 21, International Potato Center (CIP).
- 3 Martin, C. and French, E. R. , "Bacterial Wilt of Potato", Technical Information Bulletin 13, International Potato Center (CIP).
- 4 Syngenta group company , 2007, "Management of Pink Rot (*Phytophthora erythroseptica*)".
- 5 Zotarelli, L. , Hutchinson, C. , Byrd, S. , Gergela, D. , and Rowland, D. L. , 2003, "Potato Physiological Disorders - Brown Center and Hollow Heart", UF/IFAS Extension.
- 6 "Late Blight of Potato and Tomato", Information referenced from Ohio State University as seen on:
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC10359.html>
- 7 Wharton, P. , Kirk, W. , Berry, D. , and Snapp, S. , "Potato Diseases: Rhizoctonia Stem Canker and Black Scurf", Michigan State University Extension.
http://msue.anr.msu.edu/resources/potato_diseases_rhizoctonia_stem_canker_and_black_scurf_e2994

Onion

- 1 Jones, D. G. , 1998, "The Epidemiology of Plant Diseases", pp. 410–415, Springer Science Business Media, B. V.
- 2 Ehn, B. , Ferry, A. , Turini, T. , and Crowe, F. , 2012, "White Rot of Onion and Garlic: Symptoms and Controls".
- 3 "Pink Root on Onions", 2012, New Mexico State University Extension on Plant Pathology, U.S. Department of Agriculture cooperating.
- 4 Michailides, T. J. , "Pest, disease and physiological disorders management: Above Ground Fungal Diseases", University of California Davis Fruit and Nut Information, pp 214–232.
- 5 Yohalem, DS , Nielsen, K , Nicolaisen, M. , "Taxonomic and Nomenclatural Classification of the Onion Neck Rotting Botrytis Species", Mycotaxon 85: 175–182, 2003.
- 6 Trujillo, E.C., 1963. Fusarium yellows and rhizome rot of common ginger. Phytopath. 53: 1370–1371.
- 7 Sharma, S.K. and Dohroo, N.P. , 1990. Occurrence and distribution of fungi causing ginger yellows in Himachal Pradesh. Pl. Dis. Res., 5(2): 200–202.
- 8 Prachi, T.R. , Sharma, T. , and Singh, B.M., 2001. In vitro and in vivo phytotoxic effect of culture filtrates of *Fusarium oxysporum* f. sp. *zingiberi* on *Zingiber officinale*. Advances in Horticultural Sciences 14: 52–58.
- 9 Behera, S., Santra, S., Chattopadhyay, S., Das, S., and Maity, T. K., 2013. "Variation In Onion Varieties For Reaction To Natural Infection Of *Alternaria Porri* (Ellis) Ciff. And *Stemphylium Vesicarium* (Wallr.)". The Bioscan. 8(3): 759–761.

Chili

- 1 Than, P. P. , Prihastuti, H. , Phoulivong S. , 2008, "Chilli anthracnose disease caused by *Colletotrichum* species", Journal of Zhejiang University Science B pp. 764–778.
- 2 Sarah, E. , Perfect, H. , Hughes, R. , 1999, "Colletotrichum: A Model Genus for Studies on Pathology and Fungal–Plant Interactions", School of Biological Sciences, University of Birmingham, Birmingham, United Kingdom.
- 3 TNAU Agritech Portal , "Bacterial leaf spot: *Xanthomonas campestris* pv. *vesicatoria*"
http://agritech.tnau.ac.in/crop_protection/chilli_diseases_4.html
- 4 Boucher T. J. , 2012, "Managing Bacterial Leaf Spot in Pepper", IPM, University of Connecticut.
<http://ipm.uconn.edu/documents/raw2/Managing%20Bacterial%20Leaf%20Spot%20in%20Pepper/Managing%20Bacterial%20Leaf%20Spot%20in%20Pepper.php?display=print>
- 5 Report on Plant Disease , 2001, "Phytophthora Blight Of Pepper", Department of Crop Sciences, University of Illinois at Urbana Champaign.
- 6 Report on Plant Disease , 1997, "Verticillium Wilt Disease", Department of Crop Sciences, University of Illinois at Urbana Champaign. International Journal of Systematic Bacteriology, Vol. 45, No. 3, pp. 472–489, 1995.

Cucurbits

- 1 Sikora, E. J. , 2011. Common Diseases Of Cucurbits, Extension Plant Pathologist, Auburn University.
- 2 Palenchar, J. , Treadwell, D. D. , and Datnoff, L. E. , 2009. Cucumber Anthracnose in Florida, University of Florida IFAS Extension.
- 3 Report on Plant Diseases , 2012. Angular Leaf Spots of Cucurbits, Department of Crop Sciences, University of Illinois at Urbana Champaign.
- 4 Gubler, W. D. , 2009. Cucurbits: Charcoal Rot, Plant Pathology, UC Davis.
- 5 Report on Plant Diseases , 1993. Root Knot Nematodes, Department of Crop Sciences, University of Illinois at Urbana Champaign.
- 6 Nuñez-Palenius, H. G. , Hopkins, D. , and Cantliffe, D. J. , Powdery Mildew of Cucurbits in Florida, University of Florida IFAS Extension.
- 7 Wick, R. L. , 2013. Root Diseases of Greenhouse Crops, University of Massachusetts Amherst.
- 8 Zitter, T. A. , Scab, Department of Plant Pathology, Cornell University.
- 10 Mossler, M. A. , Nesheim, O. N. 2005. Florida Crop/Pest Management Profile: Squash. Electronic Data Information Source of UF/IFAS Extension (EDIS). CIR 1265. February, 3, 2005. <http://edis.ifas.ufl.edu/>.
- 11 Dik, A. , Albajes. R. , 1999. Principles of epidemiology, population biology, damage relationships and integrated control of diseases and pests. In Albajes R . In L. Gullino , J. van Lenteren , Y. Elad , (eds.), Integrated pest and disease management in greenhouse crops. Dordrecht, The Netherlands: Kluwer Academic Publishers: 69–81.
- 12 Konstantinidou-Doltsinis, S. , Schmitt, A. 1998. Impact of treatment with plant extracts from Reynoutria sachalinensis (F Schmidt) Nakai on intensity of powdery mildew severity and yield in cucumber under high disease pressure. *Crop Protection* 17: 649–656.
- 13 Brown, J. , 2002. Comparative genetics of avirulence and fungicide resistance in the powdery mildew fungi. In Bélanger, R., W. R. Bushnell, A. J., Dik, T. L. W. Carver, (ed.), *The Powdery Mildews. A Comprehensive Treatise*. St. Paul, MI: APS Press: 56-65.
- 14 Paulitz, T. C. , Belanger, R. R. , 2001. Biological control in greenhouse systems. *Annual Review of Phytopathology* 39: 103–133.

Ginger

- 1 Nelson, S. , 2013. Bacterial Wilt of Edible Ginger in Hawaii, University of Hawaii, <https://cms.ctahr.hawaii.edu/gingerwilt>.
- 2 Trujillo, E. E. , 1964. Diseases of Ginger, Hawaii Agricultural Experiment Station, University of Hawaii, Circular 62, 6.
- 3 Belgrave, A. , 2007. Biological Control of *Fusarium oxysporum* f.sp. *Cubense* Using Non-Pathogenic *F. oxysporum* Endophytes, University of Pretoria, 132–134.
- 4 Ravindran, P. N. , and Babu, K. N. , 2004. *Ginger: The Genus Zingiber*, CRC Press, 321.
- 5 Kim, C. H. , and Yang, S. S. , Effects of Soil Disinfection, Fungicide Application, and Narrow Ridge Cultivation on Development of Ginger Rhizome Rot Caused by *Pythium myriotylum* in Fields, Plant Pathology Division, National Institute of Agricultural Science & Technology, Suwon, 129–135.
- 6 Ram, D. , Kusum, M. , Lodha, B. C. , Webster, J. , and Mathur, K. , 2000. Evaluation of resident biocontrol agents as seed treatments against ginger rhizome rot. *Indian Phytopath*, 53(4): 450–454.
- 7 Setty, T. A. S. , Guruprasad, T. R. , Mohan, E. and Reddy, M. N. N. , 1995. Susceptibility of ginger cultivars to rhizome rot at west coast conditions. *Environ. Ecol.*, 13: 242–244.
- 8 Huqa, M. I. , and Nowsher Ali Khanb, A. Z. M. , 2007. Efficacy in-vivo of different fungicides in controlling *Stemphylium* blight of lentil during 1998–2001. *Bangladesh J. Sci. Ind. Res.* 42(1), 89–96.
- 9 Yabuuchi, E. , Kosako, Y. , Yano, I. , Hotta, H. , and Nishiuchi, Y. , 1995. Transfer of two *Burkholderia* and an *Alcaligenes* species to *Ralstonia* gen. nov.: proposal of *Ralstonia pickettii* (Ralston, Palleroni and Douderoff 1973) comb.nov., *Ralstonia solanacearum* (Smith 1896) comb. nov. & *Ralstonia eutropha* (Davis 1969) comb. nov. *Microbiology and Immunology* 39, 897–904.
- 10 Prachi, T. R. , Sharma, T. , and Singh, B. M. , 2001. In vitro and in vivo phytotoxic effect of culture filtrates of *Fusarium oxysporum* f. sp. *zingiberi* on *Zingiber officinale*. *Advances in Horticultural Sciences* 14(2): 52–58.
- 11 Sohi, H. S. , Sharma, S. L. , and Verma, B. R. , 1973. Chemical control of *Phyllosticta* leaf spot of ginger (*Zingiber officinale*). *Pesticides* 7: 21–22.
- 12 Singh, A. K. , 2011. Management of rhizome rot caused by *Pythium*, *Fusarium* and *Ralstonia* spp. in ginger (*Zingiber officinale*) under natural field conditions. *Indian J. Agric. Sci.* 81: 268–270.

13 Setty, T. A. S. , Guruprasad, T. R. , Mohan, E. and Reddy, M. N. N. , 1995. Susceptibility of ginger cultivars to rhizome rot at west coast conditions. Environ. Ecol. 13: 242–244.

Maize

- 1 Lipps, P. E. , and Mills, D. R. , 2001. Anthracnose Leaf Blight and Stalk Rot of Corn, The Ohio State University Extension Bulletin 802.
- 2 Jackson, T. A. , 2014. Rust Diseases of Corn in Nebraska, University of Nebraska – Lincoln Extension, 4.
- 3 Report on Plant Diseases , 1990. Corn Smuts, Department of Crop Sciences, University of Illinois at Urbana Champaign. <http://ipm.illinois.edu/diseases/series200/rpd203>.
- 4 Crous, P. W. , Groenewald, J. Z. , Groenewald, M. , Caldwell, P. , Braun, U. , and Harrington, T. C. , 2006., Species of Cercospora associated with grey leaf spot of maize. Stud Mycol. 55: 189–197.
- 5 Mishra, S. R. , 2014, Virus and Plant Diseases, Discovery Publishing House, 141–142.
- 6 Singh, R. , and Srivastava, R.P. , 2012, Southern Corn Leaf Blight- An Important Disease of Maize: An Extension Fact Sheet. Indian Research Journal of Extension Education Special Issue (Volume I), pp. 334–337.
- 7 Williams, K. M. , 2014, Characterization of an RTX-Like Toxin and an Alpha-2-Macroglobulin in *Pantoea stewartii* subsp. *stewartii*, Causal Agent of Stewart's Wilt of Sweet Corn. UC Riverside Electronic Theses and Dissertations.
- 8 Gay, J. P. , and Cassini, R. , 1973. Possibilities of control of maize diseases by fungicide treatments during growth. Phytiatrie Phytopharmacie, 22(1): 19–26.
- 9 Pronczuk, M , Bojanowski, J , and Warzecha, R , 1996. Preliminary evaluation of effectiveness of fungicides in protecting maize plants against diseases. Biuletyn Instytutu Hodowli i Aklimatyzacji RoSlin, No. 197:151–155; 6 ref.
- 10 Arny, D. C. , Smallej, E. B. , Ullstrup, A. J. , Worf, G. L. , and Ahrens, R. W. , 1971. Eyespot of maize, a disease new to North America. Phytopathology, 61: 54–57.
- 11 Reifsneider, F.J.B. , Arny, D.C. , 1983. Yield loss of maize caused by *Kabatiella zeae*. Phytopathology, 73(4):607–609.
- 12 HYP3 , 2005. Eyespot of maize. HYP3 on line.
<http://www.inra.fr/Internet/Produits/HYP3/pathogene/6kabzea.html>.

Grape

- 1 Report on Integrated Pest Management, Michigan State University.
http://www.ipm.msu.edu/grape_diseases/anthracnose
- 2 Hed, B. , 2017. 2017 Summer Disease Management Review, Penn State Extension.
<https://psuwineandgrapes.wordpress.com/category/viticulture-2/canopy-management>
- 3 Wayne, F. W. , 2003. Black Rot of Grapes, Cornell University, NYAES.
- 4 Ellis, M. A. , 2016. Downy Mildew of Grape, Ohio State University Extension.
<https://ohioline.osu.edu/factsheet/plpath-fru-33>
- 5 Borkar, S. G. , and Yumlembam, R. A. , 2017. Bacterial Diseases of Crop Plants, CRC Press.
- 6 Ellis, M. A. , 2016. Eutypa Dieback Of Grape, Ohio State University Extension.
<https://ohioline.osu.edu/factsheet/plpath-fru-11>
- 7 Agrios, G. N. , Transmission of Plant Diseases By Insects, University of Florida.
- 8 Constable, F. , and Rodoni, B. , 2014. Grapevine Leafroll-Associated Viruses, Department of Environment and Primary Industries (DEPI), Victoria.
- 9 Halleen, F. , Fourie, P. H. , and Lombard, P. J. , 2010. Protection of Grapevine Pruning Wounds against *Eutypa lata* by Biological and Chemical Methods, Article in South African Journal for Enology and Viticulture.
- 10 Tefera, M. , Robert, R. M. and Rayapati, A. N. , July 9–11, 2008. The Occurrence of Grapevine Fanleaf Virus in Washington State Vineyards, Proceedings of the 2nd Annual National Viticulture Research Conference University of California, Davis.
- 11 Jones, L. R. & Grout, A. J. 1897. *Alternaria fasciculata* Bull. Torrey bot. Club 24: 257.
- 12 Ophel, K. , Allen, K. , 1990, *Agrobacterium vitis* sp. nov. for Strains of *Agrobacterium biovar 3* from Grapevines, International Journal of Systematic and Evolutionary Microbiology 40: 236–241.
- 13 Carter, M. V. , 1991. The Status of *Eutypa lata* as a Pathogen. Kew, UK: International Mycological Institute, Phytopathological Papers no. 32.
- 14 Munkvold G.P. , Marois J.J. , 1993b. Efficacy of natural epiphytes and colonizers of grapevine pruning wounds for biological control of eutypa dieback. Phytopathology 83, 624–629.

- 15 John, S. , Wicks, T. J. , Hunt, J. S. , Lorimer, M. F. , Oakey, H. , Scott, E. S. , 2005. Protection of grapevine pruning wounds from infection by *Eutypa lata* using *Trichoderma harzianum* and *Fusarium lateritium*. *Australasian Plant Pathology* 34, 569–575.
- 16 Andret-Link, P. , Laporte, C. , Valat, L. , Ritzenthaler, C. , Demangeat, G. , Vigne, E. , Laval, V. , et al., 2004. Grapevine fanleaf virus: Still a major threat to the grapevine industry. *J. Plant Pathol.* 86(3), 183–195.
- 17 Martelli, G. P. , Savino, V. , 1990. Fanleaf degeneration. In: Pearson, R. C. and Goheen, A. (eds.) *Compendium of grape diseases.*, St Paul, MN: APS Pres, 48–49.
- 18 Hewitt, W.B. , Raski, D.J. , and Goheen, A.C. , 1958. Nematode vector of sail-borne fanleaf virus of grapevines. *Phytopathology*, 48: 586-595.
- 19 Cohn, E. , Tanne, E. , and Nitzany, F. E. , 1970. *Xiphinema italiae*, a new vector of grapevine fanleaf virus. *Phytopathology*, 60: 181–182.
- 20 Graniti, A. & Martelli, G. P. 1966. Further observations on legno riccio rugose wood, a graft transmissible stem pitting of grapevine. *Proc. Int. Conf: Virus Vector Perennial Hosts and Vitis*, 1965, p. 168–179. Div. Agric. Sci., Univ. Calif., Davis.

Sugarcane

- 1 TNAU Agritech Portal , “Organic Farming: Disease Management of Sugar”.
http://agritech.tnau.ac.in/org_farm/orgfarm_agridiseases.html
- 2 Majumder, D. , 2012, “Wilt disease of sugarcane”, IITK Agropedia. <http://agropedia.iitk.ac.in/content/wilt-sugarcane>
- 3 Rott, P. , and Comstock, J. C. , 2002, “Sugarcane Smut Disease”, University of Florida, IFAS Extension.
- 4 Comstock, J. C. , Sandhu, H. S. , and Odero, D. C. , 2015, “Sugarcane Yellow Leaf Disease”, University of Florida IFAS Extension.
- 5 La Granja Agricultural Research and Extension Center (LGAREC), 2012, “*Pellicularia sasakii* (Shirai) ITO”. <https://www.bar.gov.ph/index.php/biofuels-home/bioethanol/sugarcane/sugarcane-diseases/1544-banded-6>
- 6 Nainwal, K. , 2009, “Eye Spot Disease in Sugarcane”, IITK Agropedia.
- 7 Lehrer, A. T. , Schenck, S. , Yan, S.-L. , and Komor, E. 23 July 2007, “Movement of aphidtransmitted Sugarcane yellow leaf virus (ScYLV) within and between sugarcane plants”, *Plant Pathology* 56, 711–717.
- 8 Went, F. A. F. C. 1893. Het Rood Snot. *Arch. Java Suikerindus.* 1: 265-282.
- 9 Butler, E. J. , 1906. Fungus Diseases of Sugar-cane in Bengal. *India Dept. Agr. Mem., Bot. Ser.* 1 (3): 2–24.
- 10 Miller, W. A., Rasochova, L., 1997. Barley yellow dwarf virus. *Annual Review of Phytopathology* 35 : 167–90.
- 11 Rassaby, L., Girard, J. C., Lemaire, O., Costet, L. , Irey, M. S., Kodja, H. , Lockhart, B. E. L., Rott, P., 2004. Spread of Sugarcane yellow leaf virus in sugarcane plants and fields on the island of Réunion. *Plant Pathol.* 53: 117–125
- 12 Rangaswami, G. 1960a. Studies on two bacterial diseases of sugarcane. *Current Science* 29: 318–319
- 13 Rangaswami, G. 1960b. Further studies on bacterial gummosis and red stripe disease of sugarcane *Journal of Annamalai University* 22: 135–50.
- 14 Yadav, M. K., Dhakad. P. K. , Yadav. S. K. Sushreeta, N. , and Ram, C. , 2016. A Treatise on Sugarcane Diseases in North India. *Advances in Life Sciences* 5(11), 4366–4367.

Guava

- 1 Merida, M. and Palmateer, A. J. , 2006, “2013 Florida Plant Disease Management Guide: Guava (*Psidium guajava*)”, University of Florida IFAS Extension.
- 2 Tabua, E. , “Plant Diseases and Their Management”, Lecture at Florida National University.
- 3 Prakash, O. , “IPM Schedule for Guava Pests”, National Horticulture Mission Ministry of Agriculture Extension Bulletin No. 2.