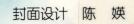
# 植物保护专业英语教程

A Basic Course of Scientific English for Plant Protection

主编 程立生 蔡笃程 副主编 张荣意 骆焱平 王伟 主审 陈鸣芬

欢迎登录:中国农业出版社http://www.ccap.com.cn 全国农业教育教材网http://www.qgnyjc.com





定价: 35.00元

#### 高等院校教材

# 植物保护专业英语教程

# A Basic Course of Scientific English for Plant Protection

主 编 程立生 蔡笃程

副主编 张荣意 骆焱平 王 伟

主 审 陈鸣芬

#### 图书在版编目 (CIP) 数据

植物保护专业英语教程/程立生,蔡笃程主编.— 北京:中国农业出版社,2012.1 高等院校教材 ISBN 978-7-109-16460-4

I. ①植··· Ⅱ. ①程···②蔡··· Ⅲ. ①植物保护-英语-高等学校-教材 Ⅳ. ①H31

中国版本图书馆 CIP 数据核字 (2011) 第 275008 号

中国农业出版社出版 (北京市朝阳区农展馆北路2号) (邮政编码100125) 责任编辑 李国忠

北京中兴印刷有限公司印刷 新华书店北京发行所发行 2012 年 2 月第 1 版 2012 年 2 月北京第 1 次印刷

开本: 787mm×1092mm 1/16 印张: 18.5 字数: 450 干字 定价: 35.00 元

L本版图书出现印刷、装订错误,请向出版社发行部调换)

主 编 程立生 (琼台师范高等专科学校)

蔡笃程 (海南大学)

副主编 张荣意 骆焱平 王 伟 (海南大学)

主 审 陈鸣芬(海南大学)

# 前言

为了进一步贯彻"教育要面向现代化、面向世界、面向未来"的指导思想,适应经济全球化和新的科技革命浪潮对人才培养的需求,教育部于2001年明确提出本科教育要创造条件使用英语等外语进行公共课和专业课教学。各高等院校纷纷采取各种措施鼓励采用双语教学形式开设专业和专业基础课程。

随着科学技术的不断发展,我国各个领域的对外交流日益频繁,对于从事植物保护工作的专业人员而言,既要通过查阅和参考各种主要以英语为载体的文献资料,了解本领域的研究进展和新技术的应用情况,又要经常性地与海外学者或技术人员进行交流。因此植物保护专业的学生在校期间掌握一定的专业英语知识就显得十分重要而迫切。为此,我们组织编写了这本《植物保护专业英语教程》,作为植物保护类专业的本科学生学习专业英语的教材,同时也为相关专业的研究生和技术人员学习专业英语知识提供参考资料。

全书共有20个单元,每个单元由主课文(A)和副课文(B)组成,每篇课文后均附有词汇表和练习,书的末尾附有词汇检索总表、参考文献、常见农药名称的中英文对照和各个单元主课文的练习答案。全书20个单元按照植物保护一级学科所包含的植物病理学、农业昆虫与害虫防治及农药学3个二级学科的顺序排列,所涉及每个二级学科的内容尽可能地体现其学科知识结构的全面性,如与农业昆虫与害虫防治学科相关的内容就包括了昆虫外部形态、内部解剖与生理学、昆虫行为、昆虫与环境的关系、昆虫系统学和害虫治理等方面的内容。全书内容丰富,选材新颖,题材全面,结构科学,通过学习本书内容,学习者不但可以掌握相应的专业英语知识,对其巩固和提高植物保护专业知识也会有很大的帮助。

本书的出版得到了海南大学重点学科经费的资助。

由于时间仓促,加之编者水平有限,书中错漏之处在所难免,恳请同行专家和师生批评指正。

编 者 2011年8月

# **Contents**

## 前言

| Unit I (A)  | The Concept of Disease in Flants                        | 1   |
|-------------|---|-----|
| Unit 1 (B)  | Molecular Plant Pathology                               | 5   |
| Unit 2 (A)  | Genes and Disease                                       | 11  |
| Unit 2 (B)  | The Gene-for-Gene Concept                               | 15  |
| Unit 3 (A)  | Systemic Acquired Resistance                            | 19  |
| Unit 3 (B)  | Defense Through RNA Silencing by Pathogen-derived Genes | 27  |
| Unit 4 (A)  | Integrated Control of Plant Diseases                    | 30  |
| Unit 4 (B)  | New Tools in Epidemiology                               | 37  |
| Unit 5 (A)  | Characteristics of Plant Pathogenic Fungi               | 4]  |
| Unit 5 (B)  | Isolating the Pathogen                                  | 48  |
| Unit 6 (A)  | Characteristics of Plant Pathogenic Bacteria            | 51  |
| Unit 6 (B)  | Phytoplasmas and Spiroplasmas                           | 57  |
| Unit 7 (A)  | Detection and Identification of Plant Viruses           | 61  |
| Unit 7 (B)  | Virus Infection and Virus Synthesis                     | 66  |
| Unit 8 (A)  | Characteristics of Plant Pathogenic Nematodes           | 69  |
| Unit 8 (B)  | Root-knot Nematodes: Meloidogyne spp                    | 74  |
| Unit 9 (A)  | Insect Biodiversity and Conservation                    | 78  |
| Unit 9 (B)  | Reasons for the Studying of Insects                     | 88  |
| Unit 10 (A) | External Structure of Insects                           | 94  |
| Unit 10 (B) |   | 104 |
| Unit 11 (A) | Embryonic and Postembryonic Development of Insects      | 11  |
| Unit 11 (B) | Specialization of Insects                               | 12  |
| Unit 12 (A) |   |     |
| Unit 12 (B) |   |     |
| Unit 13 (A) | Environment Factors Affecting Development of Insects    | 14  |
| Unit 13 (B) | Environment and Insects ·····                           | 15  |
| Unit 14 (A) | Insect Systematics: Phylogeny and Classification        | 15  |
| Unit 14 (B) | Naming, Describing and Classification of Insects        | 16  |
| Unit 15 (A) | Detrimental Insects                                     | 17  |
| Unit 15 (B) | Beneficial Insects                                      | 18  |
| Unit 16 (A) | Integrated Pest Management                              | 18  |
|             |   |     |

### 植物保护专业英语教程 A Basic Course of Scientific English for Plant Protection

| Unit 16 (B)                                      | Reasons for Insects Becoming Pests and Assesment of Pest Status ······· | 197 |  |  |
|--|---|-----|--|--|
| Unit 17 (A)                                      | What Is a Pesticide?  | 202 |  |  |
| Unit 17 (B)                                      | Generic Terms, Trade Names, and Chemical Names                          | 208 |  |  |
| Unit 18 (A)                                      | Agriculture Formulations  | 211 |  |  |
| Unit 18 (B)                                      | The Formulations of Pesticides ·····                                    | 219 |  |  |
| Unit 19 (A)                                      | Chemical Control in USA   | 223 |  |  |
| Unit 19 (B)                                      | Insecticides  | 231 |  |  |
| Unit 20 (A)                                      | Herbicides ·····  | 234 |  |  |
| Unit 20 (B)                                      | Mode of Action ·····  | 241 |  |  |
| Glossary   |   |     |  |  |
| English-Chinese Common Names of Pesticides ····· |   |     |  |  |
| Key to Exercises                                 |   |     |  |  |
|  |   |     |  |  |
| References ·····                                 |   |     |  |  |

# Unit 1 (A) The Concept of Disease in Plants

Because it is not known whether plants feel pain or discomfort and because, in any case, plants do not speak or otherwise communicate with us, it is difficult to pinpoint exactly when a plant is diseased. It is accepted that a plant is healthy, or normal, when it can carry out its physiological functions to the best of its genetic potential. The meristematic (cambium) cells of a healthy plant divide and differentiate as needed, and different types of specialized cells absorb water and nutrients from the soil; translocate these to all plant parts; carry on photosynthesis, translocate, metabolize, or store the photosynthetic products; and produce seed or other reproductive organs for survival and multiplication. When the ability of the cells of a plant or plant part to carry out one or more of these essential functions is interfered with by either a pathogenic organism or an adverse environmental factor, the activities of the cells are disrupted, altered, or inhibited, the cells malfunction or die, and the plant becomes diseased. At first, the affliction is localized to one or a few cells and is invisible. Soon, however, the reaction becomes more widespread and affected plant parts develop changes visible to the naked eye. These visible changes are the symptoms of the disease. The visible or otherwise measurable adverse changes in a plant, produced in reaction to infection by an organism or to an unfavorable environmental factor, are a measure of the amount of disease in the plant. Disease in plants, then, can be defined as the series of invisible and visible responses of plant cells and tissues to a pathogenic organism or environmental factor that result in adverse changes in the form, function, or integrity of the plant and may lead to partial impairment or death of plant parts or of the entire plant.

The kinds of cells and tissues that become affected determine the type of physiological function that will be disrupted first. For example, infection of roots may cause roots to rot and make them unable to absorb water and nutrients from the soil; infection of xylem vessels, as happens on vascular wilts and in some cankers, interferes with the translocation of water and minerals to the crown of the plant; infection of the foliage, as happens in leaf spots, blights, rusts, mildews, mosaics, and so on, interferes with photosynthesis; infection of phloem cells in the veins of leaves and in the bark of stems and shoots, as happens in cankers and in diseases caused by viruses, mollicutes, and protozoa, interferes with the downward translocation of photosynthetic products; and infection of flowers and fruits interferes with reproduction. Although infected cells in most diseases are weakened or die, in some diseases, e. g., in crown gall, infected cells are induced to divide much faster (hyperplasia) or to enlarge a great deal more (hypertrophy) than normal cells and to produce abnormal amorphous overgrowths (tumors) or abnormal organs.

Pathogenic microorganisms, i. e., the transmissible biotic (=living) agents that can cause disease and are generally referred to as pathogens, usually cause disease in plants by disturbing the metabolism of plant cells through enzymes, toxins, growth regulators, and other substances they secrete and by absorbing foodstuffs from the host cells for their own use. Some pathogens may also cause disease by growing and multiplying in the xylem or phloem vessels of plants, thereby blocking the upward transportation of water or the downward movement of sugars, respectively, through these tissues. Environmental factors cause disease in plants when abiotic factors, such as temperature, moisture, mineral nutrients, and pollutants, occur at levels above or below a certain range tolerated by the plants.

From Plant Pathology (5th ed.).

#### Words and Expressions

pinpoint /'pinpoint/ v. 确认,查明,精确地识别 meristematic / meristə mætik/ adj. 分生组织的 cambium / kæmbiəm / n. 形成层 differentiate / difp'renfieit/ vt. 分化 photosynthesis / fəutəu sin $\theta$ əsis/ n. 光合作用 translocate /trænslautkeit/vt. 改变…的位置 metabolize /ma'tæbəlaiz/ v. 产生代谢变化 .pathogenic / pæ0a dzenik/ adj. 病原的 malfunction /mæl'fanksen/v. 机能失常 损害,损伤 impairment /im'peament / n. xylem / zailəm, -lem/n. 木质部 vascular / væskjulə/ adj. 导管的 韧皮部 phloem / flauem/ n. mollicutes / molikjuts/ n. 柔膜细菌 protozoa /prautau'zaua/ n. 原生动物 hyperplasia / haipə pleiziə/ n. 增生,增大 hypertrophy /hai'pə: trəfi/ n. 肥大,过度膨胀 amorphous /ə'mɔ:fəs/ adj. 无定向的,无组织的 foodstuff / fu : dstAf/ n. 食品,粮食 transmissible /trænz'misəbl/ adj. 可传染的

#### Notes to the Text

Although infected cells in most diseases are weakened or die, in some diseases, e.g., in crown gall, infected cells are induced to divide much faster (hyperplasia) or to enlarge a great deal more (hypertrophy) than normal cells and to produce abnormal amorphous overgrowths (tumors) or abnormal organs. 虽然在大多数病害中受感染细胞的活力会衰弱

或死亡,但在某些病害如冠瘿中,被感染细胞则因受诱导而分裂大大加快(增生),或远大于正常细胞(肥大),并发生异常的无定向的过度生长(肿瘤),或产生异常器官。

#### **Exercises**

| I   | . Reading Comprehension   | n There are some qu   | estions or unfinished stat                 | ements followed by   |  |
|---|---|---|--|----------------------|--|
| fo  | ur choices for each, ch   | oose the best one base  | ed on the text.                            |                      |  |
| 1.  | It is not easy for us to  | know if a plant is dis  | eased because                              |                      |  |
|   | A. plants don't have t  | he feeling of discomfor   | t B. plants don't have the feeling of pain |                      |  |
|   | C. plants don't commu   | inicate with us   | D. not mentioned in                        | the text             |  |
| 2.  | A diseased plant may  | result from   |  |                      |  |
|   | A. adverse environmen   | ntal factor   | B. pathogenic organi                       | ism                  |  |
|   | C. pest insects   |   | D. both A and B                            |                      |  |
| 3.  | The responses of plan   | it cells to pathogenic fa   | ictors are                                 |                      |  |
|   | A. invisible  | B. visible  | C. A and B                                 | D. none of these     |  |
| 4.  | The adverse changes   | of a diseased plant may   | be in                                      |                      |  |
|   | A. form   | B. function   | C. integrity of the plant                  | D. all of these      |  |
| 5. The following except can interfere with photosynthesis directly. |   |   |  |                      |  |
|   | A. mildews  | B. mosaics  | C. rusts                                   | D. vascular wilts    |  |
| 6.  | Infected plant cells m  | ay  |  |                      |  |
|   | A. die  | B. divide much faster   | C. be weakened                             | Dall of these        |  |
| I   | . Discussion  |   |  |                      |  |
| 1.  | Give a definition about   | t plant disease.  |  |                      |  |
| 2.  | How do you distinguis   | sh a diseased plant from  | n a healthy one?                           |                      |  |
| I   | . Vocabulary There a  | re some incomplete sen  | tences with four choices f                 | or each, choose the  |  |
| On  | e that best completes   | the sentence.   |  |                      |  |
| 1.  | The cambium cells of  | a healthy plant can   | •  |                      |  |
| A. absorb water and nutrients from the soil B. carry on photo       |   | B. carry on photosynthes  | osynthesis                                 |                      |  |
|   | C. divide and different   | C. divide and differentiate as needed D. store the photosynthetic |  |                      |  |
| 2.  | The symptoms of the   | disease are   |  |                      |  |
|   | A. invisible  | B. measurable   | C. visible                                 | D. B or C            |  |
| 3.  | If are infected, translocation of water and minerals to the crown of the plant will |   |  |                      |  |
|   | be interfered.  |   |  |                      |  |
|   | A. foliage  | B. phloem cells   | C. roots                                   | D. xylem vessels     |  |
| 4.  | Translocation of  | is downward moven   | nent                                       |                      |  |
|   | A. nutrients  |   | B. photosynthetic produc                   | cts                  |  |
|   | C. water  |   | D. all of these                            |                      |  |
| 5.  | is (are) of ca  | ses that infected cells d   | livide much faster or enlar                | ge a great deal more |  |
|   | than normal ones.   |   |  |                      |  |
|   | A. Hyperplasia  | B. Hypertrophy  | C. Tumors                                  | D. All of these      |  |
|   |   |   |  | _                    |  |

| 6.   | By secreting, pathogens disturb the metabolism of plant cells and cause disease in plants. |                         |                           |                     |  |  |
|--|--|-------------------------|---------------------------|---------------------|--|--|
|  | A. enzymes   | B. growth regulators    | C. toxins                 | D. all of these     |  |  |
| 7.   | By growing and multi   | plying in the of        | plants, some pathogens    | block the upward or |  |  |
| the downward translocation of fluid through these tissues.   |  |                         |                           |                     |  |  |
|  | A. flowers   | B. phloem vessels       | C. xylem vessels          | D. both B and C     |  |  |
| 8. is (are) not abiotic factor that can cause plant disease. |  |                         |                           |                     |  |  |
|  | A. Mineral nutrients   | B. Moisture             | C. Mollicutes             | D. Temperature      |  |  |
| N  | . Translation Transla  | te the following senten | ces from Chinese to Engli | sh, or from English |  |  |
| to   | Chinese.   |                         |                           |                     |  |  |

- 1. The visible or otherwise measurable adverse changes in a plant, produced in reaction to infection by an organism or to an unfavorable environmental factor, are a measure of the amount of disease in the plant.
- 2. 受感染的细胞和组织的类别决定将首先被阻碍的生理功能的类型。
- 3. Infection of phloem cells in the veins of leaves and in the bark of stems and shoots, as happens in cankers and in diseases caused by viruses, mollicutes, and protozoa, interferes with the downward translocation of photosynthetic products.
- 4. Pathogenic microorganisms, i.e., the transmissible biotic agents that can cause disease and are generally referred to as pathogens.
- 5. 当非生物性因子如温度、湿度、矿物营养成分和污染物超过或低于植物的耐受范围时, 环境因子就会引起病害。

# Unit 1 (B) Molecular Plant Pathology

Since 1980, great emphasis has been placed on determining the specific molecule and the "genetic connection" of any substance involved in disease development. Because viruses and bacteria are small in size and because a great deal of background information is available on them, more molecular studies have been carried out with them than with the much larger fungi and nematodes. Already the number, location, size, sequence, and function of most or all genes of many viruses are known in detail. Many of these genes have been excised from the virus and have been transferred either to host plants, to which they often convey resistance, or into bacteria, in which they are expressed and the proteins they code for are isolated and studied. Similar transfers have been accomplished with a few bacterial and fungal genes coding for certain pathogenesis-related proteins.

The beginnings of molecular plant pathology can probably be traced to the isolation by W. Stanley in 1935 of the tobacco mosaic virus as a crystalline protein, which he believed to be infectious. Although 2 years later it was shown that the protein also contained a small amount of RNA, it was not until 1956, when Gierrer and Schramm showed that the ribonucleic acid and not the protein of tobacco mosaic virus was responsible for the infection of plant cells and for the reproduction of complete virus particles. In the meantime, in 1941 Beadle and Tatum showed that one gene codes for one enzyme. The following year (1942) Flor showed that a single gene is responsible for pathogenicity in the flax rust fungus and that the rust fungus gene corresponds to a single gene for resistance in the flax plant (the gene-for-gene concept). In 1953, Watson and Crick showed that DNA exists in a double helix and their discovery impacted greatly all of biology. In the mid-1960s, studies of tobacco mosaic virus led to the full elucidation of the genetic code according to which specific base triplets of DNA (and RNA) code for a certain amino acid. This was followed by the description in the 1970s through the 1990s of all the genes of tobacco mosaic and of many other viruses.

By the mid-1970s, the studies of A. tume faciens revealed that the T-DNA of its Ti plasmid contained several genes of which two, coding for growth regulators, were responsible for the production of tumors (galls) by the infected plants. It was later shown that the two genes could be removed and replaced with one or more genes from other organisms such as plants, other bacteria, viruses, and even animals, genes that could be transferred into and expressed (translated) by the plant cells. This discovery made possible the introduction of foreign genes into plants at will and, combined with tissue culture, which made possible the production of whole plants from single cells, it ushered in the era of

genetic engineering of plants. Subsequently, it was discovered that foreign DNA can be introduced into plant cells in several ways, including using viruses as vectors, bombarding plant cells with foreign DNA, and growing plant cells in the presence of foreign DNA. Several viral genes coding for the coat protein or other structural or nonstructural proteins, and some noncoding regions, have been engineered into plants, and many of them have been shown to make the plant more or less resistant to the virus. Also, some bacterial and fungal genes, coding for enzymes that break down the cell wall of the pathogen, have been engineered into plants and have provided the plant with resistance to these pathogens.

In 1984, P. Albersheim and colleagues identified the molecule in the cell wall of the comycete Phytophthora megasperma that acts as the elicitor of the defense response in its soybean host. It was shown later that the elicitor accomplishes this by interacting with a receptor molecule on the plant cells. In the same year, the first avirulence gene was isolated from the bacterium Pseudomonas syringae pv. glycinea by B. J. Staskawicz and colleagues. These two discoveries helped launch research that improved our understanding of pathogen virulence and plant disease resistance greatly. In 1986, bacterial hypersensitive response protein (hrp) genes were discovered. It was thought at first that the hrp genes were required for bacterial pathogenicity and production of the hypersensitive response; it is known now that they affect the transport of proteins in pathogenic bacteria and also the transport of bacteria into plant cells.

The first practical results of molecular plant pathology in improving disease resistance came in 1986 when R. Beachy and colleague obtained tobacco plants resistant to tobacco mosaic virus (TMV) by transforming them; i.e., introducing into them the coat protein gene of the virus in a way that the plants could express the gene and produce the virus protein. Such transformed plants are called transgenic, and the resistance they acquire is called pathogen-derived resistance. In 1989, M. B. Dickman and P. E. Kolattukudi transformed a fungus, that normally could enter host plants only through wounds, with a cloned gene coding for the enzyme cutinase. That enzyme enabled the fungus to penetrate host plants directly through the cuticle, thereby proving that cutinases play a role in the direct penetration of some plants by fungi. Two years later, in 1991, R. Broglie and coworkers showed that plants transformed with the gene that codes for chitinase exhibit enhanced resistance to disease by fungi that contain chitin in their cell walls. In the meantime, in 1990, R. Cheim and colleagues obtained transgenic tobacco plants that expressed increased disease resistance by transforming them with the gene for stilbene synthetase, the enzyme that synthesizes a phytoalexin.

Discoveries in molecular plant pathology came fast and furious in the 1990s. The concept of systemic acquired resistance (SAR) burst onto the scene through the discovery of D. F. Klessig and colleagues and J. Ryals and co-workers that salicylic acid, a relative of aspirin, is associated with SAR. The first fungal avirulence gene (avr9) was isolated from Cladosporium fulvum by P. J. G. M. De Wit, while the first plant resistance gene (Hm-1)

was isolated from corn by S. P. Briggs and J. D. Walton. The latter also showed that Hm-1 operates by producing a protein that detoxifies the host-selective toxin of the pathogen Cochliobolus carbonum. The only resistance gene conferring resistance in tomato to a bacterial pathogen through the hypersensitive response was isolated by G. B. Martin and colleague in 1993. In subsequent years, dozens of plant disease resistance genes were isolated from many plants. All these genes shared a leucine-rich repeat in the protein they coded for. Tomato plants transformed by B. Baler and co-workers with the tobacco plant resistance gene N, which makes tobacco resistant to tobacco mosaic virus, were also made resistant to the virus, proving that at least some resistance genes may function in species other than the one in which they normally occur. Furthermore, it was shown by V. M. Williamson and colleagues (1998) that a single cloned disease-resistance gene from tomato can confer resistance to both a nematode pathogen and an insect. It was also shown during this period that plant pathogens produce proteins that actively suppress the defense reactions of their host plants. In addition, the avirulence proteins of some pathogens contain signals that allow these proteins not only to be introduced into plant cells, most likely through the bacterial hrp protein system, but also to move into and function in the plant nucleus.

A new type of defense against pathogens was unveiled when it was discovered that many organisms, including plants, fungi, and animals, are capable of "RNA silencing," i. e., of regulating genes based on targeting and degrading sequence-specific RNAs. In plants, RNA silencing has been shown to serve as a defense against virus infections. As would be expected, however, many plant viruses carry genes that encode proteins that suppress the silencing of their RNA by the plant. RNA silencing can be induced experimentally and targeted to a single specific gene or to a family of related genes. It is believed that RNA silencing genes will soon play an important role in engineering resistance into plants.

Advances in molecular plant pathology have also provided a new set of diagnostic tools and techniques that are used to detect and identify pathogens even when they are present in very small numbers or in mixtures with other closely related pathogens. Such tools include detection with monoclonal antibodies, analysis of isozymes or of fatty acid profiles of pathogens, analysis of fragments of their nucleic acids produced by specific enzymes, calculation of percentages of hybridization of their nucleic acids, and determination of nucleotide sequences of the nucleic acids of the pathogens. Since the mid-1980s, segments of DNA (probes), complementary to specific segments of the nucleic acid of the microorganisms, have been labeled with radioactive isotopes or with color-producing compounds and are used extensively for the detection and identification of plant pathogens. Numerous techniques, often referred to by their acronyms, have been developed and are used; some of them are better suited for diagnosing one or more types of pathogens. For at least some pathogens, PCR, with selected differential random sequences of different species, can be effective for the detection and identification of each of these species. At other tests, PCR of sequence segments of rDNA internal transcribed spacer

(ITS) regions are used or PCR of other genes or spacers of the fungal DNA is carried out. The product is then differentiated by digestion with restriction enzymes and gel electrophoresis and detection of differential random fragment length polymorphisms (RFLP) or use of PCR together with DNA hybridization in a reverse dot blot hybridization (RDBH) assay using PCR of selected RAPD markers. Reverse transcription PCR (RT-PCR) or immunocapture RT-PCR (IC/RT-PCR), direct binding PCR (DB-PCR), and a combination of PCR and enzyme-linked immunosorbent assay (ELISA) tests are often used successfully, especially for viruses.

An area of molecular plant pathology that is going to pay multiple dividends in the future is that of genomics, i.e., sequencing of the entire genomes of plants and their pathogens. Already, the genomes of the experimental plant Arabidopsis thaliana, of several plant viruses and viroids, and of the plant pathogenic bacteria Ralstonia solanacearum and Xylella fastidiosa, the white rot fungus Phanerochaete chrysosporium, and the model nematode Caenorhabditis elegans have been sequenced in their entirety. Significant progress has already been made in sequencing the entire genomes of the very destructive plant pathogenic fungi Magnaporthe grisea, cause of rice blast; Ustilago maydis, cause of corn smut; Cochliobolus heteroserothus, another pathogen of corn; Botrytis cinerea, the gray mold of many fruits and vegetables; Fusarium graminearum, cause of head scab of wheat; and Phytophthora infestans, cause of the blight of potato and of many other pathogens of crops. Once the genomes have been sequenced, it will be easier to locate, identify, compare, isolate, and manipulate the genes for pathogenicity in the pathogens and of resistance in their host plants, as well as manipulate the introduction of them into specific locations of the plant genome where they would be most effective.

The molecular phase of plant pathology is expected to develop a great deal more and to make contributions in ways that we can hardly imagine at present. One area in which molecular plant pathology is expected to contribute greatly and to provide tremendous benefits is the area of detection, identification, isolation, modification, transfer, and expression of genes for disease resistance from one plant to another. Several such resistance genes have already been identified, isolated, transferred into susceptible plants, and, when expressed, made the plants resistant. The possibility that molecular plant pathology can modify and combine resistance genes makes likely the future utilization of resistance genes from unrelated plants or from other organisms, and perhaps even the synthesis of artificial genes for resistance for incorporation into crop plants. The practical implications of such developments cannot be overestimated, as they are likely to revolutionize the control of plant diseases by providing us with cultivars that can resist disease in the presence of the pathogen, without the need to use any pesticides.

From Plant Pathology (5th ed.).

#### Words and Expressions

excise /ek'saiz/ vt.

切除

convey /kən'vei/ vt.

传送,传播,传递,传导

tobacco mosaic virus

烟草花叶病毒

crystalline /'kristəlain/ adj.

结晶的

ribonucleic acid /raibənju: kli:ik æsid/

核糖核酸(RNA)

pathogenicity / pæ0ədzi nisiti/ n.

致病性

flax rust fungus

亚麻锈病菌

gene-for-gene concept

基因对基因假说

double helix

双螺旋 (结构)

triplets of DNA (and RNA) code

DNA (和 RNA) 的三联体编码

amino acid 氨基酸

T-DNA

转移 DNA (transferred DNA)

Ti plasmid

Ti (tumor-inducing) 质粒

tumor /'tju:mə/ n.

瘤

菌瘿, 虫瘿 gall /go:1/n.

bombard / bomba: d/ n.

viral /'vairəl/ adj.

elicitor /i'lisitə/ n.

病毒的

oomycete / jouo maisi: t/n.

卵菌 诱导子,激发子,诱导剂

avirulence /ə'viruləns/ n.

非病原性,无毒性

virulence / virulens/ n.

致病力,毒力

过敏反应蛋白 hypersensitive response protein (hrp)

transgenic / trænz'dzenik/ adj.

转基因的

pathogen-derived resistance

来源于病原物的抗性

cutinase / kju: tineiz/ n.

角质 (蛋白)酶

cuticle /'kiu:tikl/ n.

角质膜, 表皮

chitin / kaitin / n.

角质素,几丁质

stilbene synthetase

茂(均二苯代乙烯)合成酶

phytoalexin / faitəuə leksin/ n.

植保素

systemic acquired resistance (SAR)

系统获得抗性

salicylic acid

水杨酸

aspirin / esperin/ n.

阿司匹林

detoxify /di: 'toksi fai/ vt.

使解毒

host-selective toxin

寄主选择性毒素, 寄主专化性毒素

leucine-rich /'lu:si:n 'ritʃ, 'lu:sin 'ritʃ/ adj.

富含亮氨酸的

RNA silencing

RNA 沉默

monoclonal antibody

单克隆抗体