Catalogue no. 82-619-M — No. 004
ISSN: 1715-3026
ISBN: 978-1-100-19649-7

Health Analysis Division

Health state descriptions for Canadians: Mental illnesses

by Kellie A. Langlois, Andriy V. Samokhvalov, Jürgen Rehm, Selene T. Spence and Sarah Connor Gorber

Health Analysis Division
24th Floor, R.H. Coats Building, Ottawa, K1A 0T6

Telephone: 1 613 951-3806

Statistics Canada Statistique Canada

Canada

How to obtain more information

Specific inquiries about this product and related statistics or services should be directed to: Health Analysis Division, Statistics Canada, Ottawa, Ontario, K1A 0T6 (telephone: 613-951-1765).

For information about this product or the wide range of data available from Statistics Canada, visit our website at **www.statcan.gc.ca** or contact us by e-mail at infostats@statcan.gc.ca or by phone from 8:30am to 4:30pm Monday to Friday at:

Toll-free telephone (Canada and the United States):	
Inquiries line	1-800-263-1136
National telecommunications device for the hearing impaired	1-800-363-7629
Fax line	1-877-287-4369
Depository Services Program inquiries line	1-800-635-7943
Depository Services Program fax line	1-800-565-7757
Statistics Canada national contact centre:	1-613-951-8116
Fax line	1-613-951-0581

Information to access the product

This product, catalogue no. 82-619-M, is available for free. To obtain a single issue, visit our website at **www.statcan.gc.ca** and select **Publications**.

Standards of service to the public

Statistics Canada is committed to serving its clients in a prompt, reliable and courteous manner and in the official language of their choice. To this end, the Agency has developed standards of service that its employees observe in serving its clients. To obtain a copy of these service standards, please contact Statistics Canada toll free at 1 800 263-1136. The service standards are also published on **www.statcan.gc.ca** under **About us > Providing services to Canadians**.

Health state descriptions for Canadians: Mental illnesses

Recommended citation:

Langlois KA, Samokhvalov AV, Rehm J, Spence ST, Connor Gorber SK. Health state descriptions for Canadians: Mental illnesses. Statistics Canada, catalogue no. 82-619-MIE2005002. Ottawa: Statistics Canada, 2011.

The authors wish to acknowledge the contribution of the Population Health Impact of Disease in Canada (PHI) medical panel: Denis Roy, Robert Spasoff, Doug Manuel, Marie-Dominique Beaulieu, Charles Pless, and Alan Forster. Special thanks to the Centre for Addiction and Mental Health (CAMH) for their expert collaboration and to Dr. Peter Selby, Dr. Arun Ravindran, and Dr. Jorge Soni for their valuable comments on an earlier version of this monograph. Also thanks to Aline Nizigama for her review of the literature and to Elizabeth Lin and Scott Patten for reviewing the health state descriptions in this document. We also thank Andriy Samokhvalov, content editor; Jürgen Rehm, content editor; Cameron McIntosh, review coordinator; Julie Bernier, translation verification and Charlotte Clarke, Rasha Bradic, and Robert Pellarin for their roles in design and composition.

This document is one of a series that covers the major disease groupings that affect Canadians. The series is primarily intended to document the disease classifications used in the Population Health Impact of Disease in Canada research program and help researchers to understand how the PHI estimates were calculated. It is also of interest to health professionals, advocacy groups, and individual Canadians who are looking for an overview of how living with mental illnesses affects day-to-day functioning.

The PHI is a collaboration of Statistics Canada, the Public Health Agency of Canada, and researchers from McGill University, the University of Ottawa, the University of Manitoba, the Institute for Clinical Evaluative Sciences (ICES) and l'Agence de développement de réseaux locaux de services de santé et de services sociaux de la Montérégie. The PHI was funded by Statistics Canada and the Public Health Agency of Canada.

Population Health Impact of Disease in Canada (PHI) team leaders:

Julie Bernier	Jane Boswell-Purdy	William Flanagan	Stephanie Jackson
Jean-Marie Berthelot	Sylvie Desjardins	Sarah Connor Gorber	Kathy White

Statistics Canada
Health Analysis Division

Health state descriptions for Canadians: *Mental illnesses*

Kellie A. Langlois, Andriy V. Samokhvalov, Jürgen Rehm, Selene T. Spence, Sarah Connor Gorber

Published by authority of the Minister responsible for Statistics Canada

January 2012

Catalogue No. 82-619-M, No. 004

Frequency: Occasional

ISSN 1715-3026
ISBN 978-1-100-19649-7

Ottawa

La version française de cette publication est aussi disponible (nº 82-619-M au catalogue, nº 004).

Note of appreciation

Canada owes the success of its statistical system to a long-standing partnership between Statistics Canada, the citizens of Canada, its businesses, governments and other institutions. Accurate and timely statistical information could not be produced without their continued cooperation and goodwill.

Context: Health state descriptions for Canadians

This document provides standardized descriptions for the main health states associated with the progression and treatment of a disease. These descriptions are the first step in measuring how living with a disease and its treatment affects health-related quality of life in terms of physical, mental, and social well-being.

Underlying this approach is a new tool to measure functional health. Among other attributes, pain, limitations to physical functioning, or anxiety can limit an individual's ability to participate in day-to-day activities. We classify these using the Classification and Measurement System of Functional Health (CLAMES), with eleven such attributes that span physical, social and mental well-being. For each attribute, there are four or five levels ranging in severity from no limitations in the attribute to severe limitations. Level 1, for instance, represents no limitations; for the attribute describing pain and discomfort it would read "generally free of pain and discomfort." The table that follows (*Classification and Measurement System of Functional Health (CLAMES)*) shows the complete list of levels for each attribute.

For each health state, we describe a "typical" case, based on a combination of literature review and expert consultation. Although every individual's experience of a given disease will be unique, creating these general descriptions is necessary for measuring health at the population level.

The first step in this process involves conducting an extensive review of the literature on a particular disease, in order to collect information on the main types of the disease, the usual progression, symptoms, and resulting functional limitations, and typical treatment options and their effects.

This evidence is then synthesized in order to create the health state classifications. Essentially, each health state is classified according to 11 CLAMES attributes to represent its overall consequences for functional health. In this way, a large amount of information on the typical experience of a disease is condensed into a more manageable form, which facilitates measurement of the impact of the disease on the population. Next, in order to ensure their clinical accuracy, the health state descriptions and classifications are reviewed by medical experts and revised accordingly.

The classifications are used to elicit preference scores from panels of Canadians based on techniques grounded in utility theory. Preference scores, which indicate the relative preference for a health state compared with full health, help us understand how Canadians view the various aspects of functional health. Along with data on incidence and duration, preference scores contribute to estimates of the impact on the Canadian population of both disease and risk factors that contribute to them.

Measured in terms of years of life lost to premature mortality and year-equivalents of reduced functioning due to the disease, these estimates allow us to determine how many years of life—and how many years of healthy living—are lost due to specific diseases and risk factors. They provide answers to questions such as "what would be the impact of reducing obesity on the health of Canadians?" both in terms of lives saved and in terms of increased health over their lifespan.

For further details on the Population Health Impact of Disease in Canada (PHI) research program, the process of creating the health state descriptions and classifications, and the development of population estimates to which they contribute, please consult the PHI website at http://www.phac-aspc.gc.ca/phi-isp/index.html.

Note to reader : How to read the classification

Health states are classified using 11 attributes, each with 4 or 5 levels. Level 1 indicates no limitations, while level 4 or 5 are the most severe limitations. Please note that these levels are a shorthand for the classification: they are not measurements on an interval scale *(For instance, the difference between level 1 and level 2 is not the same as between level 3 and level 4. In addition, attributes are not equally important in terms of health state preferences. For more information on health state preferences developed from these scores, please see http://www.phac-aspc.gc.ca/phi-isp/index.html)*. A complete list of the attributes and levels appears in the table that follows (*Classification and Measurement System of Functional Health (CLAMES)*).

As an example, we can look at two health states, the first describing the health state at diagnosis for cancers with very good prognosis and the second describing the health state for a more advanced cancer during the last month of life, during terminal care.

Individuals with early stage breast cancer (a very good prognosis) could be described by the following:

- Somewhat unhappy (level 3 of *Emotional State*)
- Mild limitations in the capacity to sustain social relationships (level 2 of *Social Relationships*)
- Moderate levels of anxiety experienced regularly (level 3 of *Anxiety*)

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	3	1	1	2	3	1	1	1	1

By contrast, the following describes terminal care:

- Severe pain or discomfort (level 4 of *Pain or Discomfort*)
- Severe limitations in physical functioning (level 4 of *Physical Functioning*)
- Very unhappy (level 4 of *Emotional State*)
- Always feel tired, and have no energy (level 4 of *Fatigue*)
- Somewhat forgetful, and have some difficulty when trying to think or solve day-to-day problems (level 4 of *Memory and Thinking*)
- Severe limitations in the capacity to sustain social relationships (level 4 of *Social Relationships*)
- Moderate levels of anxiety experienced regularly (level 3 of *Anxiety*)
- Limitations in the use of hands and fingers, require the help of another person for some tasks (level 4 of *Use of Hands and Fingers*)

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
4	4	4	4	4	4	3	1	1	1	4

Classification of the major health states in the progression and treatment of mental illnesses are provided in the summary table.

Classification and Measurement System of Functional Health (CLAMES)

Core attributes

Pain or discomfort

1 Generally free of pain and discomfort
2 Mild pain or discomfort
3 Moderate pain or discomfort
4 Severe pain or discomfort

Physical functioning

1 Generally no limitations in physical functioning
2 Mild limitations in physical functioning
3 Moderate limitations in physical functioning
4 Severe limitations in physical functioning

Emotional state

1 Happy and interested in life
2 Somewhat happy
3 Somewhat unhappy
4 Very unhappy
5 So unhappy that life is not worthwhile

Fatigue

1 Generally no feelings of tiredness, no lack of energy
2 Sometimes feel tired, and have little energy
3 Most of the time feel tired, and have little energy
4 Always feel tired, and have no energy

Memory and thinking

1 Able to remember most things, think clearly and solve day-to-day problems
2 Able to remember most things but have some difficulty when trying to think and solve day-to-day problems
3 Somewhat forgetful, but able to think clearly and solve day-to-day problems
4 Somewhat forgetful, and have some difficulty when trying to think or solve day-to-day problems
5 Very forgetful, and have great difficulty when trying to think or solve day-to-day problems

Social relationships

1 No limitations in the capacity to sustain social relationships
2 Mild limitations in the capacity to sustain social relationships
3 Moderate limitations in the capacity to sustain social relationships
4 Severe limitations in the capacity to sustain social relationships
5 No capacity or unable to relate to other people socially

Supplementary attributes

Anxiety

1 Generally not anxious
2 Mild levels of anxiety experienced occasionally
3 Moderate levels of anxiety experienced regularly
4 Severe levels of anxiety experienced most of the time

Speech

1 Able to be understood completely when speaking with strangers or friends
2 Able to be understood partially when speaking with strangers but able to be understood completely when speaking with people who know you well
3 Able to be understood partially when speaking with strangers and people who know you well
4 Unable to be understood when speaking to other people

Hearing

1 Able to hear what is said in a group conversation, without a hearing aid, with at least three other people
2 Able to hear what is said in a conversation with one other person in a quiet room, with or without a hearing aid, but require a hearing aid to hear what is said in a group conversation with at least three other people
3 Able to hear what is said in a conversation with one other person in a quiet room, with or without a hearing aid, but unable to hear what is said in a group conversation with at least three other people
4 Unable to hear what others say, even with a hearing aid

Vision

1 Able to see well enough, with or without glasses or contact lenses, to read ordinary newsprint and recognize a friend on the other side of the street
2 Unable to see well enough, even with glasses or contact lenses, to recognize a friend on the other side of the street but can see well enough to read ordinary newsprint
3 Unable to see well enough, even with glasses or contact lenses, to read ordinary newsprint but can see well enough to recognize a friend on the other side of the street
4 Unable to see well enough, even with glasses or contact lenses, to read ordinary newsprint or to recognize a friend on the other side of the street

Use of hands and fingers

1 No limitations in the use of hands and fingers
2 Limitations in the use of hands and fingers, but do not require special tools or the help of another person
3 Limitations in the use of hands and fingers, independent with special tools and do not require the help of another person
4 Limitations in the use of hands and fingers, require the help of another person for some tasks
5 Limitations in the use of hands and fingers, require the help of another person for most tasks

Mental illnesses

Table of contents

Summary table
Classification of health states related to mental illnesses

	Core attributes						Supplementary attributes				
	Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory and thinking	Social relation-ships	Anxiety	Speech	Hearing	Vision	Use of hands and fingers
Affective disorders											
Major Depression - Mild	1	1	3	2	2	2	2	1	1	1	1
Major Depression - Moderate	2	2	4	3	2	3	2	1	1	1	1
Major Depression - Severe	3	3	5	4	4	4	3	1	1	1	1
Dysthymia	1	1	3	2	2	3	2	1	1	1	1
Bipolar Affective Disorder - Manic Episode	1	1	1	1	4	4	3	2	1	1	1
Bipolar Affective Disorder - Bipolar Patient, Active treatment	1	1	2	1	1	2	1	1	1	1	1
Anxiety disorders											
Panic Disorder	2	1	3	2	2	3	3	1	1	1	1
Panic Attack	4	3	3	1	4	4	4	1	1	1	1
Agoraphobia (moderate)	2	1	3	2	2	3	3	1	1	1	1
Agoraphobia (severe)	2	1	3	2	2	4	4	1	1	1	1
Social phobia	2	1	3	2	2	4	3	1	1	1	1
Generalized Anxiety Disorder (moderate)	2	1	3	3	2	3	3	1	1	1	1
Generalized Anxiety Disorder (severe)	2	1	3	3	2	4	4	1	1	1	1
Obsessive-Compulsive Disorder (moderate)	2	2	3	2	2	3	3	1	1	1	1
Obsessive-Compulsive Disorder (severe)	2	2	3	2	2	4	4	1	1	1	1
Post Traumatic Stress Disorder (PTSD)	2	1	3	2	4	3	3	1	1	1	1
Childhood conditions											
Attention-Deficit/Hyperactivity Disorder – Combined type	1	1	1	1	4	2	1	1	1	1	1
Autistic Disorder	1	3	1	1	4	5	1	4	1	1	2
Asperger's Disorder	1	2	1	1	1	3	1	1	1	1	2
Separation Anxiety Disorder	2	1	3	1	2	3	3	1	1	1	1
Eating disorders											
Anorexia Nervosa	3	3	3	3	2	2	3	1	1	1	1
Bulimia Nervosa	2	1	3	3	1	1	3	1	1	1	1
Mental retardation											
Mental Retardation – Mild	1	1	1	1	2	2	1	1	1	1	1
Mental Retardation – Moderate	1	3	1	1	4	3	1	2	1	1	1
Mental Retardation – Severe	1	4	1	1	5	4	1	3	1	1	1
Personality disorders											
Personality Disorder – Unspecified	1	1	3	1	2	4	3	1	1	1	1
Schizophrenia											
Catatonic Schizophrenia	3	4	4	2	5	5	1	4	1	1	4
Disorganized Schizophrenia	1	2	4	1	5	5	1	3	2	1	1
Paranoid Schizophrenia	1	1	3	1	4	4	3	1	2	1	1
Undifferentiated Schizophrenia	1	3	3	1	4	3	1	1	1	1	1
Residual Schizophrenia	1	2	3	1	2	3	1	2	1	1	1
Substance use disorders											
Harmful Alcohol Abuse (mild to moderate)	2	2	2	3	2	2	2	1	1	1	1
Harmful Alcohol Abuse (severe)	3	3	3	3	4	3	3	2	1	1	2
Alcohol Overdose	3	4	4	1	5	5	1	4	1	1	1
Alcohol Treatment (acute withdrawal with treatment)	3	2	3	3	4	4	3	1	1	1	1
Alcohol Remission	1	1	2	1	1	2	1	1	1	1	1
Heroin Abuse/Harmful Heroin Use	3	3	2	1	4	4	3	1	1	1	1
Heroin Overdose	3	4	4	3	5	5	3	1	1	1	1
Heroin Treatment (acute withdrawal with treatment)	4	2	4	3	4	3	3	1	1	1	2
Heroin Remission	2	2	3	2	4	2	2	1	1	1	1
Harmful Benzodiazepine Use (mild to moderate)	2	2	2	2	2	1	2	1	1	1	1
Harmful Benzodiazepine Use (severe)	2	2	3	4	4	3	2	2	1	1	1
Benzodiazepine Overdose	3	4	1	4	5	5	1	3	1	1	1
Benzodiazepine Treatment (mild to moderate)	3	1	3	3	2	2	3	1	1	1	1
Benzodiazepine Treatment (severe – acute withdrawal with treatment)	4	2	4	3	4	3	4	1	1	1	1
Benzodiazepine Remission	1	1	1	1	1	2	2	1	1	1	1
Harmful Cannabis Use	2	2	2	2	4	3	3	1	1	2	1
Cannabis Treatment (acute withdrawal with treatment)	2	1	2	2	3	2	2	1	1	1	1
Cannabis Remission	1	1	2	2	3	2	1	1	1	1	1
Harmful Stimulant Use	3	2	3	3	4	4	3	1	1	1	1
Stimulant Overdose	4	4	5	1	5	5	4	1	1	1	1
Stimulant Treatment (acute withdrawal with treatment)	3	3	4	4	4	3	1	1	1	1	1
Stimulant Remission	1	2	2	1	1	2	1	1	1	1	1

Introduction

Mental illnesses largely involve alterations in mood, thinking, and behaviour, as well as other domains of mental functioning, and affect almost all Canadians in some way, either directly or indirectly.[1] They routinely cause significant impairments in emotional functioning, which may lead to social or physical limitations. In some cases, such as in agoraphobia, individuals cannot even leave their homes due to intense anxiety; depression can cause an individual to lose all interest in life. What further complicates mental illnesses is that they are often comorbid with other mental illnesses.

Mental illnesses have a substantial impact on quality of life. Although they are only responsible for 1% of deaths worldwide, mental illnesses account for almost 11% of the disease burden.[2] In fact, of the ten leading causes of years lost due to disability in the world, mental illnesses accounted for four: major depression was ranked number one, alcohol use disorders was ranked second (among males), schizophrenia was fifth among males and sixth among females, and bipolar disorder was seventh among males and eighth among females.[3] In Canada, the estimated economic burden of mental illness was $51 billion in 2003;[4] these estimates include both direct and indirect costs.

This document describes the mental illnesses that have the greatest impact on Canadians in terms of prevalence or severity of disability. It also discusses how they affect the health status of Canadians. As part of a larger project, ICD9 codes were predominantly used in assessing information/collecting data for these health states as they were used to classify the diseases. The ICD9 codes are therefore presented for each condition if one is assigned.[5] ICD10 codes are additionally provided for reference.[6] The Diagnostic and Statistical Manual of Mental Disorders (4th revision)[7] was also consulted for each disorder; the DSM-IV provides diagnostic criteria for each psychiatric disorder.

Affective disorders, which are disturbances primarily in mood, will be described. Of these, major depression, dysthymia, and bipolar affective disorder are included. Anxiety disorders, which cause intense and often persistent anxiety, will also be described, including panic disorder, agoraphobia, social phobia, generalized anxiety disorder, obsessive-compulsive disorder, and post traumatic stress disorder.

Childhood conditions (attention-deficit/hyperactivity disorder, pervasive developmental disorders, including autistic disorder and Asperger's disorder, and separation anxiety disorder), are also described. The functional limitations associated with eating disorders, including anorexia nervosa and bulimia nervosa, are examined. Mental retardation in its mild, moderate, and severe cases is also described. It is important to note that although intellectual disability is the currently recognized/accepted, less stigmatizing phrase for mental retardation, this document refers to the medical condition according to the ICD9 and DSM-IV; therefore, we maintain the clinical terms for these descriptions.

The health state associated with an individual with a personality disorder is examined. Schizophrenia is a disorder of distorted thought and perception. The functional limitations caused by these subtypes are captured here: catatonic, paranoid, disorganized, undifferentiated, and residual schizophrenia.

Finally, the health states associated with disorders caused by substance use, including alcohol, heroin, benzodiazepine, cannabis, and stimulant use will also be described in terms of stages throughout the course of the disorder; specifically, the health states associated with chronic use, treatment, remission, and in some cases, overdose are examined. Coma is often a sequela to overdose but its health state will not be described in this document; the health state associated with coma will be presented in the Neurological document in this series.

Quite often, mental illnesses are comorbid with other mental illnesses. However, measuring comorbidity is a complex task and therefore was not considered in this text. Consequently, the health states and attribute levels described in this document address only those specific to the mental illness being discussed, in the absence of a comorbid disorder. Attribute levels assigned to each health state were scored using CLAMES (see Context at the beginning of this document). Compared to other scoring systems (e.g., HUI, EQ-5D), CLAMES is particularly strong in that it captures impairments in social relationships, a common limitation among individuals with mental illnesses. That being said, CLAMES is a general scale for measuring quality of life and is unable to capture other areas of functioning associated with some mental illnesses. For example, individuals who experience mania, as seen in bipolar disorder, are not scored according to the functional limitations associated with euphoria, since CLAMES captures depression in its emotional scale, rather than happiness. This limitation is addressed where necessary.

Section A - Affective disorders

Affective disorders are disorders primarily characterized by a disturbance in mood, though other mental and somatic disturbances may be present. They affect individuals of all ages, with onset typically in adolescence or young adulthood.[7] They are one of the most common and disabling mental illnesses worldwide.[8] Individuals with a mood disorder typically experience impairments in social, occupational, educational, or other areas of functioning.[1] Despite the long-term, sometimes lifetime, character of certain affective disorders, effective pharmaceutical treatment and management techniques are available for most of them that can help the individual lead a normal, productive life.

This section will highlight the three most common affective disorders: major depression, dysthymia, and bipolar affective disorder. Diagnosis for major depression and bipolar disorder is made given the occurrence of "episodes".[7] Major depression is a disorder defined by episodes of persistent sadness and a loss of interest in activities. Dysthymia is similar in the sense that it is characterized by persistent feelings of sadness, but the feelings are chronic and less severe. These depressive disorders (major depression, dysthymia) are different from bipolar disorder in that (with these individuals) there has never been a manic, mixed, or hypomanic episode;[7] in bipolar disorder, the mood alternates between episodes of persistent pathological sadness and episodes of extreme happiness and pleasure (mania).

Part 1 - Major depression

Major depression is a disorder characterized by one or more major depressive episodes, without a history of mania. A major depressive episode is diagnosed if an individual experiences either a depressed mood or a loss of interest or pleasure in most activities for a period of at least two consecutive weeks.[7] The individual also experiences at least four additional symptoms that have changed since their previous level of functioning. These symptoms include changes in appetite or weight, sleep, and psychomotor activity; decreased energy; feelings of worthlessness or guilt; difficulty thinking, concentrating, or making decisions; or recurrent thoughts of death or suicidal behaviour.[7] Furthermore, these symptoms are experienced for most of the day, nearly everyday.

Major depression, also known as unipolar major depression or major depressive disorder, is episodic in nature. For example, it can occur only once (as one single episode) or (most often) can be recurring. With recurrent major depression, repeated episodes can occur within periods of time (possibly many years) without symptoms, while others can have clusters of episodes that are very frequent. More than 50% of individuals who experience one major depressive episode suffer from a recurrent episode;[1] in fact, the number of previous episodes is highly predictive of future episodes. An episode can last weeks, months, or even years.

Major depression can occur in all races and age groups, although onset is generally between ages 15 and 30 years. It occurs about twice as often in women than in men.[1,9,10] Approximately 2%-6% of the general population will experience major depression in any 12-month period.[1,9,10,11,12,13] Lifetime prevalence rates vary but have been reported between 2% and 19%,[9,13,14], in Canadian adults over 18 years, specifically, lifetime prevalence rates are about 12%.[1]

No single cause of major depression has yet been identified, but there may be several contributing factors. During the last century, several theories of pathogenesis of depression were proposed. Monoamine theory is the most widespread and supported by clinical and laboratory findings as well as the effectiveness of contemporary antidepressants, acting as selective monoamines reuptake inhibitors.[15] Genetic predisposition theory is supported with family history studies, showing that an individual with an immediate family member who has the disorder is 1.5-3 times more likely to experience major depression.[7,16] The presence of other chronic or severe medical conditions (i.e., heart disease, cancer, diabetes) also increases the risk for experiencing major depression. Finally, a serious loss or any stressful life event, financial problems, or low self-esteem may also contribute to major depression. Though currently the monoamine theory is still the major one, the cumulative impact of genetics, adverse events in childhood and ongoing or recent stress is considered to be the best model of depression.[15]

Major depression is a treatable disorder. The most common treatments include antidepressant medications and psychotherapy/cognitive-behavioural therapy, but they are most effective when used in combination. Treatment is aimed at lessening the duration and intensity of the episodes of depression and preventing recurrence; maintenance treatment may be needed for individuals who experience recurring depression.

Depression is associated with increased death rates, as it is one of the most important risk factors for suicide: up to 15% of individuals with major depression will die by suicide.[7,17,18] The Global Burden of Disease study, conducted by the World Health Organization and the World Bank, ranked (unipolar) major depression as third in 2004 in terms of the overall burden of all diseases in the world (measured by Disability-Adjusted Life Years),[3] and predicts it will rise to second by the year 2030.[8]

Symptom severity is variable: the DSM-IV classifies major depression as mild, moderate, and severe, based on the number of symptoms and degree of impairment at diagnosis. We describe the implications each class of depression has on the health state of a depressed individual.

Major depression – Mild

ICD-9 code: 296.2 ICD-10 Depressive episode – Mild F32.0/Recurrent depressive disorder, current episode mild F33.0

An individual has mild depression if few or no symptoms are present beyond those required for a diagnosis and only minor impairments in occupational or social functioning are experienced.[7] This definition describes a patient with mild depression, and is also considered to describe a patient successfully treated for major depression.

Individuals with mild depression generally experience no limitations in physical functioning, but tend to lack the motivation to complete even the most common tasks. Emotionally, feelings of sadness and despair are constant; feelings of worthlessness and incompetence can be experienced. Periods of worry and fear are noted. Often sleep is disturbed, resulting in a loss of energy and fatigue. Concentration becomes difficult and distracted, and often the individual is indecisive. Social withdrawal is not uncommon.[7,19]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	3	2	2	2	2	1	1	1	1

Major depression – Moderate

ICD-9 code: 296.2 ICD-10 Depressive episode – Mild F32.1/ Recurrent depressive disorder, current episode moderate F33.1

An individual with moderate major depression experiences symptoms and/or functional impairments between those experienced in the mild and severe cases.[7] A moderately depressed individual generally requires a great deal of effort to complete any given task, including a simple conversation. Often work and/or school obligations are neglected. Physical activity is reduced; sleep or appetite problems are common. Concentration and thinking are distracted. The potential for job loss and or loss of a social or familial role results in greater feelings of inadequacy, anxiety and hopelessness for the future.[19]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	4	3	2	3	2	1	1	1	1

Major depression – Severe

ICD-9 code: 296.2 ICD-10 Depressive episode – Mild F32.2-3 (with and without psychotic symptoms)/ Recurrent depressive disorder, current episode severe F33.2-3(with and without psychotic symptoms)

Severe depression is categorized into two groups by the DSM-IV: severe depression without psychotic features, and severe depression with psychotic features.[7] The latter case will not be discussed due to its relative rarity. To be diagnosed as severe, an individual must have several symptoms in addition to those required for a diagnosis and the symptoms result in substantial impairment in occupational or social functioning.[7] This description refers to an individual with severe depression (and without psychotic features) who is either untreated or unsuccessfully treated.

There are significant implications on the health state of a severely depressed individual. Effort is lacking so much that severely depressed individuals may fail to maintain good personal hygiene because even this would be a burden. Excessive feelings of sadness and despair are experienced, and the risk of suicide increases as the severity of the depression increases. There are difficulties in remembering, concentrating and making decisions. Sex no longer becomes enjoyable; and there is almost complete withdrawal from interpersonal contact. Anxiety is experienced regularly. Physical symptoms, including abdominal pain, tension headaches, and musculoskeletal pain may arise.[19]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	3	5	4	4	4	3	1	1	1	1

Part 2 - Dysthymia

Dysthymia is a mood disorder that is characterized by a chronic depressed mood that persists almost daily for at least two years, without a history of mania. During these periods of depressed mood, two or more additional symptoms are present, including poor appetite, overeating, insomnia, hypersomnia, low energy, fatigue, low self-esteem, poor concentration or difficulty making decisions, and/or feelings of hopelessness.[7] Furthermore, these symptoms must cause clinically significant impairment in social, occupational, or other areas of functioning, and must not be due to another medical condition or from the physiological effects of a substance. Symptom-free periods can last no longer than two consecutive months. Dysthymia is also referred to as dysthymic disorder, neurotic depression, depressive neurosis, or chronic depression.[20]

Dysthymia can affect anyone, but is two to three times more likely in women than in men.[1] Symptoms gradually persist over many years; onset is considered early if the dysthymia develops before age 21 and late if onset is at age 21 or after.[7] Lifetime prevalence rates are between 3% and 6% in Canadian adults over age 18,[1] and just under 3% in U.S. adults.[14] The estimated one-year prevalence of dysthymia is between 0.8% and 3.1%.[12,13]

Causes of dysthymia are unknown, but hypotheses on the causes of major depression are parallel. For example, dysthymia is more common among first-degree biological relatives of people with major depression than among the general population.[7] Treatment modalities are also similar: antidepressant medications and psycho- or cognitive therapy, often in combination. The disorder often persists for years, and therefore long-term continued treatment may be necessary, and may help to prevent recurrences. Although prognosis is good with treatment, often dysthymic individuals do not believe they are suffering from depression (rather they are just feeling "down") and consequently do not seek treatment.[21]

Dysthymia resembles major depression in its definition and associated features: the symptoms are similar but are chronic and relatively mild (and hence, not severe enough to meet the criteria for major depression).[22] In addition, an individual with dysthymia is generally more functional than an individual with major depression, but is particularly impaired in social and interpersonal relationships.[23] The implications of dysthymia on an individual's health state are described.

Dysthymia

ICD-9 code: 300.4 ICD-10 – Dysthymia F34.1

Individuals suffering from dysthymia can generally function adequately but at less than peak performance. Difficulties with concentration and decision-making, decreased energy and anxious and irritable moods are experienced. Sleep disturbances are likely and cause at least mild levels of fatigue and decreased energy, which occasionally may be more severe. There are also limitations in social functioning due to social withdrawal and a loss of interest or pleasure in things they once enjoyed (e.g., sex), leading to difficulties with relationships. Persistent feelings of sadness, guilt, hopelessness, and inadequacy often make the sufferer believe it is part of their inherent personality, keeping them from feeling well and happy.[21]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	3	2	2	3	2	1	1	1	1

Part 3 - Bipolar affective disorder

Bipolar disorder is a disorder characterized by one or more manic, depressive or mixed episodes. A manic episode is characterized by a period of at least one week during which there is an abnormally elevated, expansive, or irritable mood. There must also be the presence of at least three additional symptoms that include grandiosity (an exaggerated belief in one's importance); decreased need for sleep; pressured (intense) speech; flight of ideas (thoughts rapidly skip to distantly related ideas in no logical progression); distractibility; increased involvement in goal-directed activities; and involvement in pleasurable activities that have a high potential for painful consequences.[7] This disturbance must be severe enough to cause impairments in social or occupational functioning, or to require hospitalization, and must not be due to another medical condition or from the physiological effects of a substance.[7] A mixed episode, on the other hand, is characterized by a period of at least one week during which the criteria are met for both a manic episode and for a major depressive episode almost every day, and must also cause significant impairments in social or occupational functioning or require hospitalization.[7] Almost all individuals with bipolar disorder experience one or more major depressive episodes as well. For this reason, bipolar disorder is also referred to as manic depression or manic depressive disorder. In general, an individual with bipolar disorder alternates moods from severe highs (mania) to severe lows (depression), often with periods of normal functioning in between. These mood changes can be rapid or gradual.[24]

Similar to major depression, bipolar disorder is episodic in nature, and almost always it is recurring; more than 90% of individuals who experience a manic episode will have future episodes.[7] Furthermore, between 50% and 70% of manic episodes occur immediately before or immediately after a major depressive episode. The pattern of episodes are variable from person to person, however, they tend to be fairly predictable in the same individual. On average, individuals with bipolar disorder experience four episodes in a ten-year period.[7]

Bipolar disorder typically begins in late adolescence or early adulthood, with an average age of onset between 20 and 25 years.[7] It can affect anyone of any race or ethnicity, and is equally common in men and women.[1,9] Lifetime prevalence rates for bipolar disorder have been found to be between 0.2% and 1.7%.[7,11,13] In Canadian adults in particular, studies have reported a lifetime prevalence of 2.4% and a 12-month prevalence of 1%.[1]

The exact cause of bipolar disorder is unknown. A serious life event (such as divorce), a financial problem, or an illness may trigger an episode in some individuals; it may be that some individuals are more prone to emotional or physical stressors. There may also be a chemical imbalance in the brain, which could contribute to the strong tendency for a genetic disposition: about 80% to 90% of individuals suffering from bipolar disorder have a relative with some form of depression.[25] It may occur with no obvious trigger at all.

Although treatment of bipolar disorder may be complicated in certain cases, it is highly manageable. Medication often consists of mood stabilizers, such as Lithium, which is most effective against mania, and antidepressants for depressed episodes. Taking an antidepressant without an accompanying mood stabilizer has the potential to trigger a manic episode, therefore, these two medications are typically prescribed together. Psychological treatment is also available to help the individual detect their particular pattern of episodes, and also to develop strategies for managing the disorder. Due to the recurrent nature of bipolar disorder, treatment is generally long-term to prevent future episodes.

Left untreated, bipolar disorder can impose significant pressures emotionally, both on the sufferer and their families and coworkers. Still, only 27% of bipolar patients are in treatment;[26] frequently individuals (in mania) do not recognize they are ill and need treatment because they feel well. We describe below the impact bipolar disorder has on an individual's health state while they are experiencing an acute, manic episode, and also while they are a patient undergoing active treatment. This information should be combined with the description of major depression for a full interpretation of the bipolar disorder process.

Bipolar affective disorder – Manic episode

ICD-9 code: 296.4 – 296.7 ICD-10 – F31

This description refers to an individual with bipolar affective disorder while in an acute, manic episode. Generally individuals in mania feel excessively happy and excited; euphoric. Although mania is not directly captured in the emotional state attribute below, euphoria causes severe limitations in other attributes. Typically, individuals with mania have inflated self-esteem, unwarranted optimism, and poor judgment, leading to participation in pleasurable activities that may have high potential for painful consequences, such as uncontrolled buying sprees, substance abuse, or unusual sexual behaviours (e.g., infidelity or promiscuity). Social relationships tend to be unstable; the individual is active, outgoing and likely feels they have a lot of friends, but behaves irrationally. Concentration typically becomes difficult and grandiose, and thinking becomes distracted and yet much faster than normal. Speech may become incoherent due to a flight of ideas – sentences are rarely finished because thoughts rapidly change – and because the individual tends to talk really fast. Anxiety and irritability are noted.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	1	1	4	4	3	2	1	1	1

Bipolar affective disorder – Bipolar patient, active treatment

ICD-9 code: 296.4 – 296.7

This description refers to an individual who is being treated for bipolar affective disorder, which assumes the manic and depressive states to be mild (given the success rate for treatment) and chronic. Mania in its mild form is hard to distinguish from simply an optimistic mood; the individual is more lively and talkative than when in their depressed state. This, however, still imposes on an individual's quality of life. Behaviours that are constantly competing with each other can be hard to regulate; for example, when in mania, there is a decreased need for sleep and a heightened sex drive, when in depression, there is persistent fatigue and a diminished sex drive. Although the "polar" (meaning opposite) episodes are mild, they are still forcing the sufferer to deal with opposing demeanours, resulting in cognitive, social and physical limitations.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	2	1	1	2	1	1	1	1	1

Section B - Anxiety disorders

Anxiety disorders are primarily characterized by overwhelming anxiety and fear, and are among the most prevalent mental health disorders. They are typically chronic, last at least six months, and are likely to get progressively worse without treatment. Impairments in social, occupational, and/or role functioning are most common. Antianxiety medication and psychotherapy can significantly improve symptoms.

This section describes the health states associated with the most common anxiety disorders. Panic disorder is an anxiety disorder characterized by unexpected and recurrent panic attacks. Agoraphobia is a disorder in which there is intense fear of public places. In the DSM-IV, individuals with panic disorder may also have agoraphobia; this section describes panic disorder in the absence of agoraphobia, while agoraphobia is described as a separate health state.

Social phobia is an anxiety disorder in which the individual fears situations in which they can be judged by others. An individual with generalized anxiety disorder has persistent unprovoked anxiety. Obsessive-compulsive disorder is a disorder in which the individual has persistent thoughts that produce anxiety, and need to fulfill a compulsion in order to relieve the anxiety. Posttraumatic stress disorder is caused by life-threatening or comparable emotional experiences; flashbacks and recurrent re-living of a traumatic event are the most specific symptoms.[27]

Part 1 - Panic disorder

Panic disorder is an anxiety disorder that is characterized by unexpected and recurrent panic attacks. A panic attack is a sudden episode of intense fear, accompanied by at least four physical symptoms that include heart palpitations, chest pains, nausea, trouble breathing/shortness of breath, flushing or chills, terror, fear of losing control or dying, sensory distortions, and others, which peak within 10 minutes of onset. The individual typically thinks they are having a heart attack or stroke and go to the emergency department thinking they are dying. Panic attacks often occur for no apparent reason, sometimes even during sleep.[28] They can occur more than once a day and typically last only a few minutes. However, between attacks, the individual may experience considerable anxiety and fear in anticipation of having further attacks, particularly about where and when the next attack will take place. This anxiety is likely more disabling than the panic itself, and may be intense enough to trigger another attack.[7,29]

Occasional panic attacks are fairly common; many adults, however, do not develop the anxiety about having further attacks. Panic disorder is diagnosed if the individual has recurrent panic attacks (minimum four in a four-week period), and at least one of the attacks is accompanied by one or more physical symptoms, including persistent concern about having another attack, worry about the implication or consequences of the attack (i.e., having a heart attack), and/or a significant change in behaviour due to the attacks, such as quitting a job.[7] In addition, the panic attacks cannot be due to the physiological effects of a substance or another general medical condition.

Panic disorder typically begins in late adolescence or young adulthood, but children and older adults can also be affected. Lifetime prevalence rates are approximately 1-2%.[7] A study in the U.S. found a 12-month prevalence of 2.7%[12] and a lifetime prevalence of almost 5%.[14] In Canada, 12-month and lifetime prevalence rates are 1.6% and 3.7%, respectively.[1] Women are twice as likely to develop the disorder than men.[7,28,30] Although the disorder is chronic, the symptoms tend to wax and wane over time: some individuals have frequent attacks regularly for months at a time; others have less frequent attacks separated by weeks or months (even years) of remission. Individuals who experience terror in anticipation of the next attack will likely avoid places where panic attacks have occurred, or where they cannot escape easily, where help is not readily available, or where they will face embarrassment if an attack strikes. The avoidance may grow over time and lead to agoraphobia (see the next section), the inability to go anywhere beyond a surrounding that is known and safe due to intense fear. Agoraphobia can develop at any point in the course of panic disorder, but it usually develops within the first year of occurrence.[7] About 1/3rd of individuals with panic disorder develop agoraphobia.[28]

The exact cause of panic disorder is unknown, but there appears to be a genetic component; an individual with a close relative with panic disorder has a 10-20% increase in risk.[28] Stressful life events or periods (e.g., heavy workload), excess caffeine, and/or stimulating drugs may trigger an attack. Separation anxiety and psychological traumas during childhood have also been associated with onset of the disorder.[30] Because there is no laboratory test to diagnose Panic Disorder, and because the symptoms tend to mimic other disorders (e.g., heart attack), diagnosis is frequently not made for years, often after repeated visits to the emergency room and to various doctors.

Early diagnosis and treatment are key components to improved prognosis. However, many people do not seek psychiatric treatment until they develop unbearable anticipatory anxiety or agoraphobia.[28] Benzodiazepines (antianxiety medication) and antidepressants, including serotonin reuptake inhibitors, tricyclic antidepressants, and monoamine oxidase inhibitors, are the most effective medications to reduce or eliminate panic attacks. The most effective treatment (with lower relapse rates) is a combination of medication and psychotherapy. Cognitive-behavioural therapy teaches the patient to examine and analyze their thoughts associated with the situations they fear, and to reassure themselves when they are frightened. Between 70% and 90% of treated patients have significant improvement with their symptoms.[31] Relapse may occur, but recurrent attacks can be effectively treated just like the initial episode.

Panic disorder

ICD-9: 300.01 ICD-10 Panic disorder F41.0

Panic disorder is characterized by unexpected and repeated episodes of intense fear accompanied by physical symptoms. While the panic attack is the hallmark of panic disorder, many people develop intense anxiety between episodes (the chronic phase, which this health state describes), in anticipation of future attacks. Over time, the individual may avoid more and more places; their life may become so restricted that they cannot do everyday activities such as grocery shopping. They may become housebound, unless accompanied by someone they trust. Thus, the individual will likely lose or quit their job: only about 25% of patients with panic disorder are employed.[32] Restrictions in mental functioning, predominantly intense anxiety and depression are also common,[7,29] as are disturbances in concentration. Exacerbation might be accompanied with such somatic symptoms as chest pain and palpitations.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	2	2	3	3	1	1	1	1

Panic attack

ICD-9: 300.02

The panic attack is the core feature of panic disorder and is described in this health state as acute. Panic attacks often occur suddenly and without warning, although they may be a result of classical conditioning. They are defined by a sudden surge of overwhelming fear and have a strong physical component to them, including lightheadedness, a rapid heartbeat, chills or hot flashes, flushing, trouble swallowing, terror, dizziness, and chest pains. Typically the individual experiencing a panic attack feels 'crazy' or 'out of control', and has a feeling of imminent danger. Symptoms of a panic attack peak within 10 minutes, but the frequency and severity of them varies from individual to individual.[7,29]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
4	3	3	1	4	4	4	1	1	1	1

Part 2 - Agoraphobia

Agoraphobia is a disorder characterized by an intense fear of public places, particularly places where help or immediate escape might be difficult (e.g., a bus or train), or places where the individual has previously had a panic attack. The most commonly feared places are elevators, bridges, public transportation, airplanes, and shopping malls; standing in a line or in a crowd of people may also be feared. Often the fear is so extreme that the individual avoids such places; in severe cases, the individual is housebound. Agoraphobia often accompanies another anxiety disorder, especially Panic Disorder (there may be the presence of panic attacks). Alternatively, many individuals with agoraphobia have no history of panic attacks.[33] The health states described in this section refer to a typical case of agoraphobia in the absence of panic attacks.

Agoraphobia can develop at anytime, but onset is typically in late adolescence or early adulthood.[34] It affects between 0.5% and 1% of the population;[12,34,35] the annual incidence rate is about 2 per 1000 people.[36] In Canada, 12-month and lifetime prevalence rates are 0.7% and 1.5%, respectively.[1] Women are more likely to have the disorder than men.[34,35,36] The course is usually chronic.

Agoraphobia is diagnosed by the DSM-IV if the individual has anxiety about being in places where it may be difficult or embarrassing to escape or places where they could not get help in the case of a panic attack. These situations are either avoided or endured with extreme anxiety and distress, or the individual insists that someone accompanies them. Finally, the anxiety and/or avoidance must not be better accounted for by another mental health disorder; this may be the case if the individual avoids only one or two situations and therefore is considered to have social or other phobia(s).[7]

Causes of agoraphobia are unknown but several risk factors have been identified, including having panic disorder or an alcohol or substance use disorder, experiencing a stressful life event, being female, or having a tendency to be nervous or anxious.[37] A study of the incidence of agoraphobia identified previous panic disorder as the strongest predictor; having other additional phobias was also a predictor.[36] Another study found individuals with subsequent chronic health conditions and individuals who were widowed or divorced/separated (as opposed to those who were married) at increased risk for agoraphobia.[35]

Treatment for agoraphobia is important for better prognosis,[38] but often individuals are too fearful or embarrassed to seek treatment. It is particularly challenging because the individual is generally made to confront his/her fears and subsequently learn to function effectively. Treatment is often successful and begins with a combination of medication and psychotherapy. Antianxiety and antidepressant medications are commonly prescribed. Cognitive-behavioural therapy helps the individual learn about the disorder, how to cope with it and how to control it (i.e., what makes it worse). Desensitization therapy is a form of exposure therapy in which the individual imagines (or confronts) the situations that cause fear, in order from the least fearful to the most fearful, in order to change their unwanted behaviour.[37] In general, the success of treatment depends on the severity of the disorder.[33]

Agoraphobia (moderate)

ICD-9: 300.2 ICD-10 – Agoraphobia F40.0

Agoraphobia in its moderate form is quite a disabling phobia that causes a high level of anxiety. Individuals are limited to the places and situations that they consider to be safe, or require the accompaniment of a trusted friend or family member. Consequently, they feel helpless and dependent on others. In addition, their social and occupational opportunities are limited or avoided.[7,33]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	2	2	3	3	1	1	1	1

Agoraphobia (severe)

ICD-9: 300.2 ICD-10 – Agoraphobia F40.0

Individuals with severe agoraphobia suffer an extreme level of anxiety and avoid the places and situations in which they are most fearful. In fact, often the individual with severe agoraphobia is housebound. They are unable to leave trusted, safe places and people. As a result, they are unable to work or socialize outside the home, and feel detached and estranged from others. If forced to undergo the feared situation, individuals experience intense anxiety and considerable dread, "break out in a" sweat, or have a rapid heart rate or high blood pressure. As well, nausea, abdominal pain, diarrhea, and headaches are common. Symptoms of a panic attack may also be experienced: lightheadedness, dizziness, flushing, chest pain, trouble swallowing, and a feeling of a loss of control.[7,33]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	2	2	4	4	1	1	1	1

Part 3 - Social phobia

Social phobia, also known as social anxiety disorder, is a disorder characterized by a fear of situations in which there is potential for embarrassment or humiliation in front of others. There are generally two subtypes of social phobia: one involves a fear of speaking in front of people, whether it be public speaking or simply talking with a person of authority; the other subtype involves more generalized anxiety and complex fears, such as eating in public or using public washrooms, and in these cases individuals may experience anxiety around anyone other than family.[39] The anxiety becomes worse when the individual anticipates/fears that they will do something embarrassing and thus be singled out or ridiculed. Although the individual is aware that this anxiety is excessive and unreasonable, they cannot overcome it. Consequently, the individual desperately tries to avoid these situations, causing interference in work, school, or other daily activities. In extreme cases, the individual eventually avoids, or endures with intense distress, all social interaction, resulting in withdrawal even from friends and family.

Social phobia is one of the most common anxiety disorders,[40] and is among the most common psychiatric illnesses.[41] The lifetime prevalence rate of social phobia ranges from 3-13%;[1,7,14,42] Canadian rates are between 8% and 13%.[1,43] The one-year prevalence rate in Canada is 6.7%;[43] in the U.S., it is about 7%.[12] About half of all cases have the speaking fear subtype, and half the complex fears subtype.[43] Women are more likely than men to have the disorder.[40,42,43,44] Onset is typically in childhood or early adolescence,[7,44] a critical time period for developing social skills; rarely does social phobia develop in later adulthood.[44] The usual course is chronic and lifelong; some estimate an average duration of about 20 years.[38] Symptoms may fluctuate with stress and demands, and may enter remission for an unspecified period of time.

The DSM-IV diagnoses social phobia if there is striking and persistent fear towards a situation in which the individual is exposed to potential scrutiny by others, and exposure to the situation provokes anxiety. The individual realizes that this fear is excessive and unreasonable but still either avoids the situation or undergoes it despite intense anxiety or distress. For a diagnosis to be made, the avoidance or distress must cause significant impairments in the individual's daily routine, or in their occupational and social functioning. In addition, the fear is not due to the physiological effects of a substance or a medical condition.[7] Finally, if the individual is under 18 years of age, these symptoms must have occurred for at least six months.

Although the exact cause of social phobia is unknown, it appears that individuals with relatives that have the disorder are at greater risk of developing it, suggesting a genetic predisposition.[7,38,40,41] Familial and environmental factors, particularly in early childhood, also likely play a role: child-rearing style— overprotective parents may restrict the child from exposure to challenging or stressful situations, in which case the child does not develop effective coping skills, causing anxiety and avoidance; parental/peer (social) modeling— a child may observe the reactions and behaviours of his/her parents or friends and develop the same fears; behavioural inhibition—most individuals with social phobia were shy as children and were always uncomfortable in front of others.[41] It is possible that an embarrassing, humiliating, or traumatic event can precede the disorder, at which time the individual develops fear for that particular situation.

Early diagnosis and treatment of social phobia are essential in improving prognosis of the disorder and preventing comorbidity with other disorders. However, many individuals with social phobia do not seek treatment for their disorder,[42] likely because they are either embarrassed to see a professional or because they feel their shyness is part of their personality or simply a social problem rather than a mental health problem.[38,42] To escape the constant anxiety, often individuals use alcohol as self-medication because they are aware that alcohol consumption can reduce their performance anxiety;[41,42] rates of social phobia are nine times higher among individuals who abuse alcohol.[41] Medication and psychotherapy are effective treatments. Cognitive-behavioural therapy, specifically exposure therapy, gradually teaches the individual to become more comfortable in the situations that create fear. Techniques

for controlling the anxiety, such as relaxation/breathing exercises, are also taught. Group and family support therapy are effective in educating others about the disorder.

Social phobia

ICD-9: 300.23 ICD-10 – Social phobia F40.1

Individuals with social phobia experience intense anxiety and worry about any situation in which others could judge them. Physical symptoms, including a rapid heart rate, blushing, or trembling, often accompany the anxiety, which may be a source of further humiliation. Individuals with social phobia are constantly worried about looking foolish in front of others; for example, during public speaking, the individual has a fear of being embarrassed that others see their hands or voice tremble. Furthermore, the anxiety experienced may develop days or weeks before the social situation, and continue for days or weeks after the situation (individuals constantly experience guilt and worry over what others thought of them and their performance/how they were judged). This has tremendous implications for health. Social and occupational functioning are the areas most affected by this disorder; the individual likely has a hard time making friends or dating due to fear of the situation; opportunities at work may be limited and the individual may turn down promotions to avoid more social situations. Severe anxiety may cause the individual to avoid all social situations, such as drop out of school or quit their job, out of desperation to avoid public scrutiny. Low self-esteem and loneliness often result. Individuals with social phobia are at increased risk for depression and suicide.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	2	2	4	3	1	1	1	1

Part 4 - Generalized anxiety disorder

Generalized anxiety disorder (GAD) is a disorder characterized by generalized and persistent excessive anxiety and worry that is accompanied by somatic symptoms such as muscle tension. Individuals with GAD are always thinking about the "what ifs", and fear the worst in every situation. This worry is exaggerated and unrealistic, with nothing specific to provoke it. Everyday concerns such as work, health or finances can cause marked discomfort and distress.[7,45]

GAD is the broadest anxiety disorder in its class, and one of the most common. It affects about 3% of the general population in a given year;[7,12] lifetime prevalence is about 5%.[7,14] Women are more likely to have the disorder than men.[7,46,47] GAD most often begins in childhood or adolescence, but onset in adulthood is not uncommon. Onset is typically gradual, with symptoms likely developing more slowly than other anxiety disorders.[46,48] A stressful life event may cause the onset of symptoms.[49] The usual course of symptoms is chronic, with periods of exacerbation, particularly during times of stress, and remission. Although individuals with GAD report having feelings of anxiety their whole life, the focus of their worry may shift from one concern to the next over the course of the disorder.

The DSM-IV diagnoses GAD if the individual experiences excessive anxiety and worry about life circumstances (events or activities, such as work or school), which occurs more often than not for at least six months. In addition, the individual has a hard time controlling the worry. At least three of the following symptoms accompany the worry: restlessness or feeling on edge; being easily fatigued; difficulty concentrating; irritability; muscle tension; or sleep disturbances. These symptoms cause clinically significant impairment in important areas of functioning, and are not the result of physiological effects of a substance or general medical condition.[7] Symptoms typically vary in combination and severity.

The exact cause of GAD is unknown but there are likely a number of factors that contribute to the disorder. It has been suggested that GAD may have a genetic contribution.[7,47,48] The brain's neurotransmitters (specifically serotonin and norepinephrine) may be disrupted. The buildup of stressful life situations or having a serious illness may trigger anxiety. Certain personality types that are prone to feelings of anxiety or worry or feelings of insecurity may also increase the risk of developing the disorder.

Individuals with GAD frequently seek treatment. The two most common treatments are medication and psychotherapy, which can be taken alone or in combination. Benzodiazepines (antianxiety medications) are effective for symptom reduction but are highly addictive and therefore can only be taken for short periods of time. Buspirone, another antianxiety medication, is also effective and can be used on an ongoing basis.[46] Cognitive-behavioural therapy can help the individual to identify negative thoughts and behaviours and replace them with positive ones. During behavioural therapy, individuals with GAD learn techniques that they can use to cope with and reduce the anxiety, such as relaxation. Exposure therapy may be utilized to narrow down the anxiety-causing stimuli and help them to cope with their fears. Although no single treatment is best for everyone, GAD is treatable and remission can be successfully attained.

Generalized anxiety disorder (moderate)

ICD-9: 300.02 ICD-10 - Generalized anxiety disorder F41.1

Individuals with generalized anxiety disorder (GAD) have chronic, exaggerated worry and tension even when nothing seems to be provoking it. The persistent anxiety that they feel is much more severe than the normal anxiety experienced by the average individual. In general, individuals with GAD always anticipate disaster, and excessively worry about health, family, or work. Even though individuals with GAD may have stretches of time when they are not consumed by their worries, they are anxious all of the time. Individuals with a moderate case of GAD will typically not avoid the situations that cause them to suffer anxiety; on the contrary, many can be relatively productive socially and uphold employment.[45]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	3	2	3	3	1	1	1	1

Generalized anxiety disorder (severe)

ICD-9: 300.02 ICD-10 - Generalized anxiety disorder F41.1

Individuals with severe GAD experience intense anxiety over things both large and small, such as work, finances, or even car repairs. Their constant worry and anticipation for disaster may cause them to be restricted in their daily lives, avoiding situations that make them anxious. Even the simplest daily tasks can be difficult to overcome. A relatively high percentage of individuals with severe GAD are likely to be unemployed.[50] Depression often results because the individual is frustrated that they cannot control the anxiety. Lack of concentration and fatigue are common because the individual cannot relax and has trouble falling or staying asleep. The individual may experience a feeling of dread or a general loss of interest in life.[45]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	3	2	4	4	1	1	1	1

Part 5 - Obsessive-compulsive disorder

Obsessive-compulsive disorder (OCD), as its name implies, is a disorder that is characterized by obsessions, compulsions, or, most commonly, both. The obsessions are persistent, unwanted thoughts that produce intense anxiety, and the individual typically has compelling urges to perform repetitive, ritual-like behaviour(s) (i.e., compulsions) to ease and control this anxiety. However, the compulsions only relieve the anxiety temporarily, and may in fact contribute to worse functioning because they start controlling the individual. Consequently, the individual often experiences significant personal distress and/or social and occupational limitations.[7,51]

Symptoms of OCD usually first appear in childhood, adolescence or young adulthood, with onset earlier in males. Onset is typically gradual. In childhood, the disorder is more common in boys than girls;[7,52] however, among adults, men and women are equally affected. The lifetime prevalence rate of the disorder is about 2%;[7,12,53,54,55] one-year prevalence rates are slightly lower. The usual course of the disorder is chronic and relapsing, with symptoms waxing and waning over time. Predominant symptoms are variable and may differ over the course of the disorder.[56] If the individual does not seek treatment, the disorder tends to get worse over time and with age. Diagnosis of OCD, therefore, is of crucial importance so that the individual can get appropriate treatment. However, OCD is a very secretive disorder: individuals with OCD are often embarrassed of their obsessions and compulsions and may attempt to avoid or resist the symptoms. Often they feel humiliated and therefore do not seek professional help. Unfortunately, there is approximately a 10-year gap between the onset of symptoms and seeking help, with the receipt of a correct diagnosis and/or treatment potentially taking another 7 years.[57]

Obsessive-compulsive disorder is diagnosed if the individual experiences either obsessions or compulsions in a manner that well exceeds normal. By definition, obsessions are recurrent and persistent thoughts that are intrusive and inappropriate and cause striking anxiety or distress.[7] (These thoughts cannot simply be excessive worries about real-life problems as these would likely be deemed appropriate.) The individual tries to ignore or neutralize these thoughts by thinking or acting something else. Finally, the individual is aware that these obsessional thoughts are a product of their own mind. Compulsions are defined as repetitive behaviours or mental acts that are performed rigidly in response to an obsession.[7] These behaviours are intended to prevent or reduce distress or to prevent a feared situation from occurring, but are disconnected from reality and extremely excessive. In addition to the criterion for obsessions or compulsions, the individual must recognize that these behaviours are excessive and unreasonable for a diagnosis to be made, and they must be time consuming (at least one hour per day), cause marked distress, and interfere with normal, occupational, or social functioning. Finally, these symptoms must not be due to the direct physiological effects of a substance or a general medical condition.[7] It is important to note that the DSM-IV requires the presence of either obsessions or compulsions only (as opposed to both). However, most people with compulsions have associated obsessions, despite the fact that those with obsessive disorders may not have compulsive behaviours. It is rare, however, that a diagnosis is made without the presence of compulsion rituals.[54]

Individuals with OCD are aware of the fact that their obsessions and compulsions do not make sense, and typically make some attempt to resist them. Nevertheless, the urge to perform the ritual is overwhelming and only leads to worse anxiety and distress if they do not perform them. The most common obsession is that concerned with contamination by dirt or germs (also known as misophobia). The individual consequently tries to avoid all sources of contamination, such as doorknobs. The associated compulsion (and the most common) is extensive washing, showering or cleaning, possibly up to hours per day. Mental distress about being contaminated may be alleviated once the skin is raw from washing too much. Another common obsession is that of repeated doubts; for example, the individual constantly wonders if they have left the door to their house unlocked or if they turned off the stove. The associated compulsion is checking. The individual is worried that if they do not check carefully enough, they may harm others. This checking often leads to greater doubt, and they check again. The individual may even recruit family or friends to also check to ensure it has been checked correctly. This doubt leads to difficulty in concentrating and endless uncertainty. Consequently, the individual may not even go to work because they are constantly checking.

Other common obsessions include concerns about symmetry, requiring that objects or events be in a certain order or position, or a sense that something horrible or dangerous will happen if a particular ritual is not performed. Other common compulsions involve counting, ordering, hoarding, repeating actions, and requesting or demanding assurances. Washing, checking, and ordering compulsions are especially common in children.[7]

The exact cause of OCD is unknown. A genetic component is implied, since first-degree biological relatives of individuals with OCD have a higher risk of developing the disorder.[7] Abnormal activity in the brain, including poor functioning of the chemical serotonin, may also be associated.[52,53] Behavioural conditioning has also been suggested to play a role in the development (and maintenance) of the disorder: compulsions are the result of learned responses intended to reduce or avoid anxiety from the associated obsession; it is this compulsion that negatively reinforces the obsession-compulsion cycle.[52] With treatment, however, most patients show a significant improvement in their symptoms and quality of life, particularly that the symptoms no longer interfere with functioning or cause severe distress. Serotonin reuptake inhibitors (antidepressants) are often prescribed and are effective in reducing the obsessive-compulsive symptoms.[51] Behavioural therapy may also be prescribed so that individuals with OCD can face the situations that cause them anxiety and attempt to de-sensitize them. Repeated exposure to the anxiety-provoking stimulus may cause the individual to no longer fear it. This, in turn, may prevent future episodes of OCD. Behavioural therapy also teaches the individual techniques to avoid their compulsive ritual, and to deal with the anxiety. Education for patients and their families is crucial to the success of treatment.[58]

Obsessive-compulsive disorder (moderate)

ICD-9: 300.3 ICD-10 - Obsessive-Compulsive Disorder F42

Individuals with moderate obsessive-compulsive disorder (OCD) suffer intensely from recurrent, unwanted obsessions and compulsions which they feel they cannot control. Often the obsessions/compulsions require an excessive amount of time to complete; even getting dressed may take a few hours. Hence, these obsessions/compulsions interfere with an individual's daily functioning. Limitations in social and occupational functioning are the most common; individuals with OCD may not be able to carry out normal responsibilities because of the time required to complete a ritual. Psychological well-being is affected due to extreme anxiety and distress. Depression is often experienced. Relationships with parents, family and friends are affected, likely due to conflict from provoking other people to engage in their ritualistic behaviours. Concentration and other mental tasks are likely disrupted due to obsessional distractions.[7,51]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	3	2	2	3	3	1	1	1	1

Obsessive-compulsive disorder (severe)

ICD-9: 300.3 ICD-10 - Obsessive-Compulsive Disorder F42

Individuals with severe obsessive-compulsive disorder (OCD) suffer from the same limitations as an individual with moderate OCD, but on a larger scale. Anxiety and distress are extremely intense. Their preoccupation with obsessions and/or compulsions interferes with almost all areas of general functioning. The impairments to social and occupational functioning may lead to low self-esteem, lower career aspirations, marital problems, guilt, depression, sleep disturbances, and even greater anxiety. Avoidance of objects or situations that provoke anxiety can cause the individual to become housebound.[7,51]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	3	2	2	4	4	1	1	1	1

Part 6 - Post traumatic stress disorder (PTSD)

Post traumatic stress disorder (PTSD) is a disorder caused by a traumatic event that is outside the normal realm of human experience, such as rape, assault, torture, being kidnapped or held captive, military combat, severe car accidents, and natural or manmade disasters. In general, the traumatic event involves real or threatened physical harm to the self or to others, and causes intense fear, hopelessness, and/or horror. Emotional impairment results due to anxiety, depression, recurrent flashbacks, difficulty sleeping and concentrating, and feelings of guilt of having survived when others may not have.[7,59,60]

Lifetime prevalence of PTSD is approximately 8% in the population;[7,14,61] 12-month prevalence in the U.S. is 3.5%.[12] Women are more likely to develop the disorder than men.[39,61] PTSD can occur at any age, and can be a chronic condition. Immediately following the traumatic event, the individual becomes estranged or oddly unaffected because they are generally in shock. Soon thereafter, the individual experiences recurrent images or thoughts of the traumatic event through nightmares or flashbacks. These may be triggered by a simple ordinary occurrence such as a car backfiring (resembling the sound of gunfire), and tend to be so realistic that the individual believes they are reliving in the situation. Symptoms typically begin within three months of the traumatic event, and last at least one month. Rarely, there is a delay of up to years before symptoms appear. About half of individuals with PTSD fully recover within three to six months of symptoms, but others can experience symptoms for years. Severity of the disorder is increased if the traumatic event was unanticipated. The disorder may also be more severe or longer lasting if the traumatic event was caused by another individual such as in rape or kidnapping.[7] The most common traumatic events associated with PTSD in men are combat exposure and witnessing; in women, rape and sexual molestation are the most common.[61]

PTSD is diagnosed based on six criteria. First, the individual must have been exposed to a traumatic event that involved actual or threatened death or injury to themselves or to others, and responded with intense fear or horror. Second, the traumatic event is re-experienced persistently in the form of at least one of the following: recurrent and invasive recollections of the event, recurrent distressing dreams of the event, acting or feeling that the event is recurring (i.e., flashbacks), or intense distress at, or physically reacting to, exposure to cues that resemble an aspect of the event. Third, the individual persistently avoids stimuli that are associated with the trauma, as seen in the following: efforts to avoid thoughts, feelings or conversations of the trauma, including efforts to avoid activities, places, or people that may bring back thoughts or feelings of the trauma; inability to recall one or more important aspects of the trauma; markedly diminished interest or participation in significant activities; feelings of detachment or estrangement from others; restricted ability to feel emotions, particularly those of intimacy; and/or the sense of a foreshortened future (e.g., does not expect to have a career). Fourth, the individual experiences persistent symptoms of increased arousal that was not present before the trauma, as shown in the following: difficulty falling or staying asleep, irritability or outbursts of anger, difficulty concentrating; hypervigilance; and/or exaggerated startle response. Fifth, the symptoms experienced have been present for more than one month. And finally, the symptoms cause clinically significant impairment in social, occupational, or other area of functioning.[7]

The cause of PTSD is often obvious: it is the traumatic event that triggers feelings of terror and causes flashbacks. Factors that affect the likelihood of developing the disorder include the severity, duration, and proximity of the individual's exposure to that traumatic event.[7,62] Risk is highest among individuals who thought they would be killed or seriously injured during the traumatic event.[63] It has been suggested, however, that a susceptibility to the disorder may have a genetic contribution.[7,39] In addition, pre-existing mental disorders, lack of social support, childhood experiences, depression, and personality factors (e.g., neuroticism) may increase the risk of developing PTSD.

Early diagnosis of PTSD is essential to improve prognosis; individuals who remain ill one year after the traumatic event rarely recover completely.[60,61] Treatments for PTSD can improve symptoms, and patients should be treated with the long-term goal of achieving full remission. Effective medications include selective serotonin reuptake inhibitors (SSRIs) and/or tricyclic antidepressants; benzodiazepines are also effective. SSRIs appear to be the first-

line treatment for long-term therapy of chronic PTSD.[64] Psychotherapy, particularly cognitive-behavioural therapy, is also an effective treatment; individuals learn how to change their thought patterns to overcome anxiety. Support groups are also recommended to share thoughts and feelings of the traumatic event and gain confidence in coping. In some cases, continued treatment may be required for many years to prevent relapse.

Post traumatic stress disorder

ICD-9: 309.81 ICD-10 - Post traumatic stress disorder F43.1

This health state refers to an individual diagnosed with chronic PTSD (i.e., symptoms have lasted more than three months), who is not undergoing treatment for the disorder. Individuals with PTSD re-experience the trauma in their thoughts and feelings through nightmares or flashbacks. The individual likely avoids situations that remind them of the event, which may interfere with interpersonal functioning and potential feelings of detachment. Anniversaries of the event are particularly difficult.[39] Depression, anxiety, and sleep disturbances are typical consequences of their trauma. The individual experiences painful guilt at the fact that they survived and others did not, or at the things they had to do in order to survive. They may particularly feel guilty if they had to observe the serious injury or unnatural death of another person. Hyperarousal is also typical; individuals with PTSD startle easily and have excessive alertness, are irritable, aggressive, and possibly even violent. They also experience difficulty concentrating and memory disturbances.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	2	4	3	3	1	1	1	1

Section C - Childhood conditions

The conditions described in this section are the most common mental health disorders in childhood, including attention-deficit/hyperactivity disorder (ADHD), autistic disorder, Asperger's disorder, and separation anxiety disorder. Each of these disorders, by definition, has a diagnosis before 18 years of age. attention-deficit/hyperactivity disorder affects about 5% of the school-age population and contributes to poor school performance, conduct disorders, and difficulties in family and friend relationships.[7,65,66] Autistic disorder and Asperger's disorder are pervasive developmental disorders (PDDs), which are characterized by significant impairment in specific areas of development, such as social interaction skills and/or communication skills, as well as the presence of stereotyped behaviour, activities or interests.[7] The distinction between autistic disorder and Asperger's disorder is generally made in terms of severity: autistic disorder is at the lowest functioning end of the PDD continuum, whereas Asperger's relates to the mildest and highest functioning end of the continuum.[7,67,68] Finally, separation anxiety disorder is a disorder typically in children that is characterized by a fear of being alone or being without an individual the child is attached to, usually a parent.

Part 1 - Attention-deficit/hyperactivity disorder

Attention-deficit/hyperactivity disorder (ADHD) is characterized by inattention, hyperactivity and impulsivity, and is one of the most common mental health conditions in children.[7,65,66] Boys are three times more likely to develop ADHD than girls.[66] Symptoms of ADHD usually arise between the ages of three and five but are typically most prominent in the elementary school grades and often persist throughout adulthood. In fact, approximately 75% of cases will continue to have the diagnosis through adolescence, and over half of the cases continue into adulthood.[69] Severity of childhood ADHD and treatment of ADHD in childhood has been found to predict adult ADHD.[70] The functional limitations described in this section refer to the health state of a child diagnosed with ADHD; those associated with an adult are not captured here.

The DSM-IV makes a clinical diagnosis of ADHD based on the presentation and persistence of symptoms. Depending on which symptoms (pattern) are predominant over the past six months, diagnosis can be one of three subtypes. An individual is diagnosed with ADHD–predominantly inattentive type, if they have six or more of the following symptoms of inattention: often pays little attention to details or makes careless mistakes in school/work; has difficulty staying attentive in tasks or activities; seems not to listen when spoken to directly; fails to finish a task or does not follow through on instructions; often has difficulty with organizing tasks and activities; either avoids, dislikes, or is reluctant to partake in tasks that require sustained effort (e.g., schoolwork, homework); often misplaces items that are required for tasks or activities; is easily distracted by external stimuli; and/or is often forgetful in daily activities. An individual is diagnosed with ADHD–predominantly hyperactive-impulsive type, if they have six or more of the following symptoms of hyperactivity or impulsivity: often fidgets or squirms; often leaves seat/chair when remaining seated is expected; often runs around in inappropriate situations; has difficulty playing quietly; is often "on the go"; often talks excessively; often blurts out answers to questions that have not been completed; has difficulty waiting their turn; or often interrupts/intrudes others. The third subtype of ADHD is ADHD–combined type, in which the individual has at least six of the inattentive symptoms and at least 6 of the hyperactive-impulsive symptoms. Additional criteria for making these diagnoses are that some symptoms (that cause impairment) must have been present before seven years of age, and some impairment must be shown in at least two settings (e.g., school, home). Finally, it must be evident that the symptoms are causing clinically significant impairment in social, academic, or occupational functioning, and the symptoms do not occur during the course of, or are not better accounted for, another mental disorder.[7]

The majority of children with ADHD have combined type;[7] therefore, it is the subtype we describe in this section.

The underlying causes of ADHD are unknown at this time. Some assume that it stems from the home environment (i.e., allowing the child to watch too much television or consume excess sugar), but there are likely genetic influences; ADHD has been found to be more common in first-degree biological relatives of children with ADHD than in the general population.[7,71] Non-genetic factors that may be linked to ADHD include premature birth, exposure to high levels of lead in early childhood, brain injuries, and the maternal use of alcohol or tobacco. There are theories that ADHD arises as a developmental failure in the brain that impairs self-control and inhibition.[71]

The most effective treatment for ADHD is currently considered to be a combination of medication with psychotherapy, behavioural therapy, and/or emotional counseling. Ritalin (a short-acting methylphenidate) is the most common medication and helps to reduce hyperactivity and improve the ability to focus, work and learn. Other medications with similar effects include dextroamphetamine (Dexadrine or Dextrostat), pemoline (Cylert) and Adderall. Medication should not be taken indefinitely and is often discontinued to assess the child's condition. Psychotherapy is another mode of treatment that helps individuals with ADHD to learn new behaviours that will raise self-esteem and teach them how to deal with the emotional effects of ADHD. Parents may also be encouraged to attend skills training in order to learn techniques for managing their child's behaviour.[72]

Attention-deficit/hyperactivity disorder – combined type

ICD-9: 314.01 ICD-10 - Attention-deficit/hyperactivity disorder F90.0

This health state describes a child diagnosed with ADHD-combined type who is currently taking a psychostimulant medication (i.e., Ritalin) as a form of treatment. Insomnia and appetite suppression are the two most common side effects. The major symptoms of ADHD-combined type are inattention, hyperactivity and impulsivity. Children with ADHD-combined type have a hard time keeping their minds on a specific task and may get bored after only a few minutes. Concentration, organization, and completion of tasks (particularly for a new task) are difficult. Consequently, the child often suffers low academic achievement and poor school performance, and has more school suspensions and expulsions, leading to conflict with parents and teachers (and other school authorities). Hyperactivity is characterized by fidgeting and squirming, bouncing from task to task, and excessive talking. Impulsivity is manifested by impatience: the child may not think before they act, which may lead to participation in potentially dangerous activities without consideration of the consequences. In general, individuals with the disorder have difficulties in adapting to the demands of social life and often fail to reach the goals they strive for themselves. Children with ADHD also have a difficult time making and keeping friends, and family relationships are impaired. The child with ADHD is often punished for being disruptive and thought of as lazy or irresponsible due to their lack of self-application.[7,71] Fine and gross motor abilities development may be slightly impaired in some cases.[73] Many of these children experience more depressive symptoms in comparison to children without ADHD.[74]

Classification

Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	1	1	4	2	1	1	1	1	1

Part 2 - Pervasive developmental disorders

Pervasive developmental disorders (PDDs) are disorders that cause significant impairment in several areas of development, including social interaction skills, verbal and non-verbal communication skills, and/or the presence of stereotyped behaviour, interests and activities.[7] In general, these impairments become evident as the child fails to meet age-appropriate development, as compared to other children the same age. In the majority of cases, individuals are diagnosed before three years of age.

There are five PDDs in the DSM-IV, each with their own specific diagnostic criteria: autistic disorder, Asperger's disorder, Rett's disorder, childhood disintegrative disorder, and pervasive developmental disorder not otherwise specified.[7] These PDDs are defined based on the number and type of symptoms present, which could range from mild to severe, or the age of onset of the symptoms. This section describes the health states associated with a diagnosis of autistic disorder and of Asperger's disorder, as they are the most common of the PDDs.

Part 2a - Autistic disorder

Autistic disorder is a lifelong pervasive developmental disorder that affects all of mental development. It is at the opposite end of the PDD continuum from Asperger's and is more involved with lower functioning. Specifically, autistic disorder is characterized by impaired social interaction and communication, as well as behaviour patterns that are repetitive and restricted. It is the most prevalent of the PDDs, with prevalence rates ranging from 2 to 20 cases per 10,000.[7,75,76,77] Rates are 4-5 times higher in males than females.[7,78] Although onset is typically prior to age three years, most children are not diagnosed before age four[76,78] because symptoms of the disorder are difficult to identify in infancy. Possible signs during infancy include lack of eye contact and/or facial responsiveness, a failure to respond to parents' voices, and a lack of protective reflexes when falling. Symptoms become more noticeable as the child falls farther behind in development compared with other children the same age.

Autistic disorder is defined as a spectrum disorder because it affects each individual differently and at varying degrees. Approximately 30% of individuals with autistic disorder are high functioning (have a normal IQ),[75] while the majority have at least some degree of mental retardation.[75,77] At least half of individuals with autistic disorder learn to speak, but many others are mute. Those that can speak tend to use language in unusual ways: some may not be able to combine words into a meaningful sentence; some repeat words as a parrot would (referred to as echolalia); others may only be able to speak single words.

Diagnosis of autistic disorder is based on observation of the individual's communication, behaviour, and developmental levels. Diagnosis is made if the individual has at least six or more of the following items: 1) qualitative impairment in social interaction as seen in at least two of the following: a) marked impairment in nonverbal behaviours; b) failure to develop relationships with peers at the appropriate developmental level; c) lack of wanting to share enjoyment or interests with others; d) lack of social or emotional reciprocity; 2) qualitative impairments in communication as seen in at least one of the following: a) delay in or lack of the development of spoken language; b) marked impairment in initiating or sustaining a conversation (among individuals who do speak); c) peculiar or repetitive use of language; d) lack of make-believe or social imitative play at the appropriate developmental level; and 3) at least one of the following that is evidence for restricted repetitive patterns of behaviour: a) preoccupation with restricted patterns of interest that are abnormal in either intensity or focus; b) inflexible adherence to non-functional routines or rituals; c) repetitive motor mannerisms; and d) persistent preoccupation with parts of objects. In addition to these items, the disturbance must be apparent by delays or abnormal functioning in at least one of social interaction, language as used in social communication, or symbolic or imaginative play, prior to three years of age. Finally, these disturbances must not be better accounted for by Rett's disorder or childhood disintegrative disorder.[7]

Though no specific cause of autistic disorder has been identified, there is evidence for a strong genetic component. Current heritability estimates are above 90%,[79,80] although no one gene has been identified. Parents who already have a child with autistic disorder are at increased risk for having another one with autistic disorder, a risk of approximately 3% (50+ times higher than the population rate).[77,81,82] This risk extends to other forms of PDDs, such as Asperger's disorder. Non-genetic factors are associated with disruption of normal brain development, and usually occur prenatally. Examples include prenatal exposure to a viral infection, use of maternal anticonvulsants, and hypothyroidism or other medical conditions of the mother.[83,84,85]

There are several approaches for treatment of autistic disorder. Interventions typically consist of highly structured, specialized education programs that are tailored to the individual. These interventions target specific areas of communication, social skills, play, cognition, and independence. Individuals with more severe impairments may require education in managing the basic needs and tasks for daily living. Medications may be prescribed to treat specific symptoms if those symptoms pose a threat to the child or interfere with patient education,[86] such as aggression or self-injurious behaviour. Behavioural approaches focus on rewarding the child each time they attempt or perform a new skill, in order for the child to perform it more often. Parental involvement in any treatment program is a factor for greater success. In general, the earlier treatment begins, the more opportunities there are for learning; in turn, the child's developmental rates, particularly skill and language development, may improve.[83]

Autistic disorder

ICD-9: 299.0 ICD-10 Childhood autism – F84.0

Individuals with autistic disorder experience significant impairments in communication; engaging in a meaningful conversation can be extremely difficult. The inability to communicate effectively contributes to frustration – the individual may not be able to tell others what he/she needs and therefore may simply scream or throw things. Hearing capacity is usually normal although some individuals with autistic disorder act as if they are deaf; some are particularly sensitive (often painfully) to sound. Social interaction is also limited. Though it is hard to estimate the functional limitations associated with the use of hands and fingers, typically their fine motor skills are either underdeveloped or not used properly, or both. Individuals with autistic disorder have a difficult time forming relationships with others, and are often teased and tormented by peers. They tend to have a preference for being alone, provide little or no eye contact, and are non-responsive to verbal cues or normal teaching methods. Aggressive behaviour toward others or self may be present; temper tantrums are not uncommon in young children. Often individuals with autistic disorder have an obsessive interest in a single item, idea or person. They tend to have their own routines and rituals, engage in repetitive activities (e.g., rocking or banging their head), and are resistant to change in daily routines (for example, if their toothbrush has been moved, they may get very upset). Walking, bathing or dressing themselves may be difficult due to their inflexible and rigid behaviours.[7,83,85]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	3	1	1	4	5	1	4	1	1	2

Part 2b - Asperger's disorder

Asperger's disorder, or Asperger syndrome, refers to the mildest and highest functioning end of the pervasive development disorder continuum, and is characterised by delays in the development of social skills and behaviour. While there are some similarities to autistic disorder, there are also some very important differences. The unusual restricted and repetitive patterns of behaviour and activities, as well as severe and sustained impairment in social interactions, are common features of both Asperger's and autistic disorder. However, individuals with autistic disorder tend to be socially isolated, whereas individuals with Asperger's are motivated to approach others. In addition, individuals with autistic disorder tend to have a preoccupation with parts of objects or rituals whereas those with Asperger's are more likely to be completely encompassed in a topic about which they may spend endless time learning. As well, a child with Asperger's does not show the same delays as a child with autistic disorder in the areas of cognitive development or developing language skills.[67,68]

Asperger's disorder is diagnosed if the individual has qualitative impairment in social interaction, as seen by two or more of the following: a) marked impairment in multiple nonverbal cues such as eye contact or facial expressions; b) failure to develop relationships with peers at the appropriate developmental level; c) a lack of seeking shared enjoyment or interests with others; and/or d) a lack of social or emotional reciprocity. The individual also shows restricted repetitive and stereotyped patterns of behaviour, as evidenced by any of the following: an abnormally intense preoccupation with one or more stereotyped and restricted patterns of interest; inflexible adherence to specific, non-functional routines/rituals; stereotyped and repetitive motor mannerisms (e.g., hand or finger flapping); and/or persistent preoccupation with parts of objects. These symptoms cause clinically significant impairment in social, occupational or other areas of functioning. Finally, there is neither a clinically significant delay in language or cognitive development, nor in age-appropriate self-help skills, adaptive behaviour, or curiosity about the environment.[7]

Prevalence of Asperger's disorder is comparable to autistic disorder; prevalence rates for Asperger's have ranged from 2.5 to 36 per 10,000,[75,87] with boys at least four times more likely to have the disorder than girls.[7,87] Onset is typically between the ages of two and six years but it is often not recognized until later (average age at diagnosis is around 7 to 11 years),[88,89] possibly as late as young adulthood. This is likely because language and cognitive skills are considered normal; it is not until the child enters pre-school that the social difficulties become apparent. These social difficulties often become more striking over time.[7] It has been found that children with severe language impairments have been shown to receive a diagnosis (1.2 years) earlier than other children, whereas children with hearing impairments receive a diagnosis (10 months) later than other children.[88] Similar to autistic disorder, no single cause has been identified, but there is likely a genetic component. There is a higher risk for Asperger's and other autism spectrum disorders in family members of an affected child.[7]

Though the majority of individuals with Asperger's disorder improve with age, early diagnosis and treatment is optimal for long-term prognosis. Treatment of Asperger's disorder is most effective when multiple therapies are combined and customized to the child's developmental and behavioural needs. Psychotherapy, behaviour modification, social skills training programs, and familial/parental education and support are interventions that aid the individual and their family to cope more effectively with changing social goals and demands.[90,91,92,93] Educational interventions (i.e., special education services) may be required, particularly when the goal is to integrate the individual into a regular classroom.[68,94] Some pharmacological interventions may be prescribed to alleviate associated symptoms of Asperger's, such as hyperactivity or depression. Individuals with Asperger's often complete high levels of education, gain employment and live independently.

Asperger's disorder

ICD-9: 299.8 ICD-10 – Asperger's syndrome F84.5

An individual with Asperger's disorder experiences delays in the development of social skills and behaviour. As opposed to autistic disorder, language development is normal: the individual likely has an extensive vocabulary and most have strong verbal skills. Often speech is pedantic (putting excessive emphasis on details) and formal. There are some difficulties, however, with nonverbal communication such as facial expressions or body posture. Verbal communication may be impaired and is likely due to social dysfunction: the individual often fails to adhere to the give-and-take of a conversation and will pursue/maintain a topic that may be inappropriate. Mentally, individuals with Asperger's have average or above average intelligence (IQ). Often the individual will have intense preoccupations about a certain topic or interest, and possess extensive knowledge of facts and information on that topic. For some individuals, this preoccupation will remain through adulthood and form the basis for their career; in others, this area of preoccupation will be replaced by another. Clumsiness and poor motor skills may be apparent, particularly in the use of hands and fingers. Many individuals with Asperger's have a tendency to rock or pace while concentrating; hyperactivity is often present. Repetitive and restrictive behaviour is common and the individual is likely resistant to change. Individuals with Asperger's are typically considered socially awkward. They experience difficulties in social interaction, appear to be uninterested in sharing interests or experiences, tend to be unaware of occurrences in their surroundings, and are egocentric. Despite the desire to fit in socially, they are often viewed by peers as odd and therefore experience social isolation, peer rejection and frequently are victims of bullying. As adults, social interaction likely improves but can be challenged by the demands of marriage (and/or living with others) and working with other people.[7,67,68]

Classification

Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	2	1	1	1	3	1	1	1	1	2

Part 3 - Separation anxiety disorder

Separation anxiety disorder (SAD) is a disorder usually first diagnosed in childhood or adolescence, and is characterized by excessive anxiety of separation from the home or from people to whom the individual is attached (e.g., parents). Although there is a period in childhood during which it is developmentally appropriate (12-24 months of age) to experience separation anxiety, individuals with SAD experience extreme anxiety over long periods of time. The individual becomes markedly afraid to leave an attachment figure because they fear that if they are apart, something bad will happen to prevent their re-uniting, such as death or an accident. The individual with SAD may undergo temper tantrums or experience a panic attack. Many refuse to participate in activities outside the home; about 75% of individuals refuse to go to school.[95] Consequently, SAD significantly interferes with academic and social functioning.

SAD typically develops in middle childhood, but early onset can occur before six age years of age.[7] Rarely, SAD will develop in adulthood. It is often first diagnosed when the child begins school. Lifetime prevalence rates are between 3% and 5%;[7,14,95,96,97,98] prevalence decreases with age. SAD is more common in females than males.[7,98,99] Most children with SAD grow out of the disorder; approximately one-third of cases persist into adulthood.[98] The typical course involves periods of exacerbation and remission. The higher the number of SAD symptoms, the more likely SAD will persist. Moreover, individuals with persistent SAD tend to receive at least one comorbid diagnosis.[100]

The DSM-IV diagnoses SAD if the individual experiences excessive anxiety when faced with separation from home or from someone the individual is attached to. This anxiety must be developmentally inappropriate, and manifested by at least three of the following: recurrent excessive distress when anticipating separation from home or someone the individual is attached to; persistent and excessive worry about losing major attachment figures or attachment figures being harmed; persistent and excessive worry about getting lost or another event in which the individual will never reunite with the major attachment figure; persistent unwillingness or refusal to go to school or other locations due to fear of separation; persistent and excessive fear or reluctance to be alone or without major attachment figures at home; persistent unwillingness or refusal to sleep unless near a major attachment figure or refusal to sleep away from home; recurrent nightmares revolving around separation; and/or repeated complaints of physical symptoms (e.g., headache, nausea) when faced with or anticipating separation from someone the individual is attached to. These symptoms must have been present for at least four weeks and onset before age 18 years. Finally, the disturbances from SAD cause significant impairment in social, academic/occupational, or other area of functioning.[7]

SAD may develop after a highly stressful life event, such as the death of a parent. Although there is no particular cause, there is some evidence that its development has a genetic contribution; twin and adoption studies have shown that children who have a biological relative with the disorder are more likely to develop SAD than the general population.[7,96,97] Parental factors also seem to play a role; SAD is more common in the offspring of women with panic disorder,[7] or other anxiety or depressive disorders.[96,97] Individuals with SAD typically have other anxiety or depressive disorders themselves, particularly social anxiety and panic disorder.[97]

The most widely used mode of treatment for SAD is psychotherapy. Cognitive-behavioural therapy teaches the child to identify their unrealistic fears and anxious thinking and develop more appropriate coping strategies to reduce the anxiety. Exposure therapy is a form of psychotherapy in which the individual is gradually exposed to anxiety-causing separation (by slowly increasing the distance from parents or other major attachment figures). This forces the individual with SAD to confront their fears with the goal of reducing their anxiety over time. Family therapy may also be recommended; parents can learn more about the disorder and its consequences, and they can learn how to help encourage their child to face new situations and avoid excessive criticism. Rewards and praise are sometimes effective. Medication therapy is typically only required if the individual has persistent symptoms after attempting psychotherapy; an antidepressant may help if the individual is also depressed.[97]

Separation anxiety disorder

ICD-9: 309.21 ICD-10 - Separation anxiety disorder of childhood F93.0

Separation anxiety disorder (SAD) occurs mostly in children. Individuals with SAD become excessively afraid of leaving a loved one, usually a parent, and are preoccupied with thoughts that frightening things may happen while they are separated (i.e., they or the attachment figure may die or be injured). In anticipation of separation, the individual is nervous and may cry or cling to the attachment figure. Panic attacks are not uncommon. Occasionally, the individual will hit the person forcing the separation. When separated, individuals with SAD typically experience physical symptoms in addition to the marked anxiety, including headaches, stomachaches, nausea, and vomiting. Consequently, the individual may refuse to go to school or participate in activities outside the home and therefore may become socially withdrawn. Reactions (tantrums) to attachment figures when forced with separation contribute to parental frustration, resentment, and family conflict. Impairments in social and personal functioning are common. Depression, difficulty concentrating, and sleep problems may be experienced.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	1	2	3	3	1	1	1	1

Section D - Eating disorders

Eating disorders are characterized by severely disturbed eating behaviours. They typically begin in adolescence or young adulthood and affect women ten times more than men.[1,7] According to recent research studies, prevalence of the most common eating disorders is 0.3-1% for anorexia nervosa and perhaps three times that for bulimia nervosa.[7,101,102] Prevalence of completely defined anorexia nervosa among young females and males respectively is 0.04% and 0%; for bulimia nervosa, prevalence is 0.3% among young females and 0.2% among young males.[103] While binge eating disorder is more common in all age groups, anorexia nervosa and bulimia nervosa are more disabling and more often become a subject of medical attention. Thus, the two most common, well-defined and disabling eating disorders are discussed in this section.

Anorexia nervosa is an eating disorder characterized by refusal to maintain normal body weight. Bulimia nervosa is an eating disorder characterized by eating excessive amounts of food in one sitting and then trying to compensate for the over-eating by ridding the body of the food.

Individuals with anorexia nervosa or bulimia nervosa are similar in terms of their disturbed perception in body shape and weight, in their level of dissatisfaction with their bodies, and in their fear of gaining weight. However, individuals with anorexia nervosa often feel in control of their eating and body weight, whereas individuals with bulimia nervosa feel out of control. In addition, individuals with bulimia nervosa are often within normal body weight, while individuals with anorexia nervosa are generally below the healthy weight range.

Individuals with an eating disorder often have a perfectionistic attitude toward school or work, low self-esteem, and distorted body image. Female athletes under pressure to be thin (e.g., gymnasts, swimmers) are especially vulnerable. Although the causes of developing an eating disorder are unknown, a variety of factors are likely involved. Society's views and the media tend to portray the message that thinness is attractive, which may contribute to a distorted body image. First-degree relatives of individuals with an eating disorder are more likely to develop an eating disorder, which suggests the disorders may be genetically predisposed.[7,104] Finally, individuals with other emotional or psychological disorders, particularly substance abuse, personality disorders, or affective disorders (depression), are at higher risk of developing an eating disorder.

Treatment generally involves a combination of nutrition education and psychotherapy, including individual and family counseling. Medication may also be useful. Treatment is most effective if started early in the course of the disorder.[105]

There are two subtypes of anorexia nervosa: the first type is restricting type, in which the individual participates in dieting, fasting, and/or excessive exercise to achieve weight loss and does not regularly engage in binge-eating or purging behaviour (self-induced vomiting or misuse of laxatives, diuretics, or enemas); the second type is binge-eating/purging type, in which the individual regularly engages in binge-eating and/or purging behaviour (at least weekly).[7] The ICD10 criteria for anorexia nervosa includes starvation and exercise as the main components of anorexia nervosa (and binging and purging are not considered); binging and purging behaviours are, however, included in the ICD10 criteria for bulimia nervosa.[6] Therefore, although it is recognized that both types of the disorder may alternate with each other, the health state described in this section represents an individual with restricting type anorexia nervosa.

The functional limitations associated with binging and purging behaviours are described in the health state for an individual with bulimia nervosa, in accordance with the ICD10. It should be noted that many individuals have combinations of eating disorder symptoms that may not be sufficient for a diagnosis of anorexia nervosa or bulimia nervosa; these individuals are therefore diagnosed as "Eating disorder not otherwise specified."[7]

Part 1 - Anorexia nervosa

Anorexia nervosa is a mental disorder occurring predominantly in females (90% of cases or more),[7] and is characterized by refusal to maintain normal body weight, intense fear of becoming obese that does not diminish despite weight loss, and distorted body image resulting in a feeling of being fat. It is one of the most common psychiatric conditions in young women.[106]

Anorexia nervosa affects between 0.3% and 1% of women.[101,102] Onset is typically in mid- to late-adolescence, with behaviour likely beginning as innocent dieting. Gradually, weight loss becomes an obsession and progresses to extreme and unhealthy weight loss. The individual will starve him/herself and yet still exercise excessively to continue losing weight. Despite extreme weight loss, the individual still views him/herself as fat, becomes socially withdrawn and preoccupied with food. Suicide attempts occur in about 20-30% of subjects.[107] Course and symptom severity is extremely variable; some individuals suffer a single episode, others fluctuate between weight gain and relapse, and some experience chronic symptoms over many years.[7] At some point during the course of anorexia nervosa, over 50% of individuals will develop bulimic symptoms, typically within the first five years,[108] although this course will not be described here. Prevention measures are not known at this time.

The DSM-IV clinically diagnoses anorexia nervosa if the following criteria are met: refusal to maintain a minimally normal body weight (body weight less than 85% of that expected) or failure to make expected weight gain during growth periods; intense fear of gaining weight or becoming fat; a distorted view of one's body weight or shape, unnecessary influence on self-evaluation, or denial of the seriousness of their low body weight; and amenorrhea—absence of at least three consecutive menstrual cycles (in postmenarcheal females).[7]

No specific cause of anorexia nervosa has been identified; it is likely that the causes of anorexia nervosa are due to multiple factors. There is increased risk among first-degree biological relatives of individuals with anorexia nervosa,[7,104] suggesting a genetic component. Society's views of thinness may also contribute. Personality variables (i.e., perfectionism) may influence or be a consequence of anorexia nervosa.[109] Prognosis is improved with early detection and intervention. Approximately half of those who fully develop the symptoms of anorexia nervosa recover within five years;[110] an estimated 5% to 20% will eventually die from complications related to anorexia nervosa.[7,111] The malnutrition that results from anorexia denies the body of essential nutrients it needs to function normally, and therefore it slows down its processes to conserve energy. Menarche may be delayed in prepubertal females.[7] Starvation can also affect most major organ systems. Estrogen levels are low. Constipation, abdominal pain, lethargy, bradycardia, and cold intolerance are also experienced. Electrolyte imbalance is one of the most dangerous sequelae.[112] Over time, reproductive problems, osteoporosis, continued low BMI, and major depression may arise. Death may result due to suicide, starvation, or electrolyte imbalances.[7]

Treatment generally involves a comprehensive approach: individual therapy, family therapy, behaviour modification, and nutritional rehabilitation, with weight gain as the ultimate goal.[105] Antidepressants may be helpful if the individual is depressed. Parents are considered a vital part of the treatment process. Inpatient treatment may be necessary to restore weight and address physiological sequelae of starvation (i.e., fluid and electrolyte imbalances), particularly if the individual is more than 25% below ideal body weight or has been ill for more than two years. Relapse rates are approximately 35%.[113,114] Self-directedness (i.e., have a clear sense of one's self and one's goals) is associated with better outcomes in individuals with anorexia nervosa.[115]

Anorexia nervosa

ICD-9: 307.1 ICD-10 - Anorexia nervosa F50.0

An individual diagnosed with anorexia nervosa is extremely fearful of becoming fat and therefore restricts their total food intake, often so much that they consume only a few foods. Malnourishment caused by (semi-) starvation may result in muscle wasting, dehydration, abdominal pain, amenorrhea, constipation, cold intolerance, cardiac arrhythmias, impaired renal function, and osteoporosis. Lethargy and fatigue are common, also due to lack of food/energy intake; self esteem levels are low; depression, anxiety, and irritability are experienced. Cognitive deficits are also common with starvation. The individual may eventually become socially withdrawn and may experience somatic/sexual dysfunction, particularly in severely underweight individuals. Many individuals with anorexia nervosa are in denial of their illness; often medical attention is sought by concerned family members.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	3	3	3	2	2	3	1	1	1	1

Part 2 - Bulimia nervosa

Bulimia nervosa is an eating disorder characterized by cycles of binging and purging: the individual begins the cycle by eating large amounts of food in a single sitting, typically until the individual is uncomfortably full. The individual then tries to compensate for the overeating and tries to rid the body of the food that was consumed. There are two types of bulimia nervosa, depending on the method of compensation: purging type, in which the individual regularly engages in self-induced vomiting or the misuse of laxatives, diuretics, or enemas; or non-purging type, in which the individual uses other methods of compensation, such as fasting or excessive exercise, but does not regularly engage in self-induced vomiting or the use of laxatives, diuretics, or enemas.[7] Self-induced vomiting is the most common method to compensate for binge eating, present in 80-90% of individuals who present for treatment,[7] and therefore this section describes the health state of an individual with bulimia nervosa, purging type.

Bulimia nervosa predominantly affects females (about 90% of cases).[7,116] Approximately one to three percent of young women develop bulimia nervosa in their lifetime.[7,102] Bulimia nervosa typically begins in adolescence or early adulthood but can be difficult to identify because of extreme secrecy; in addition, most individuals with bulimia nervosa are within the normal weight range,[7] so their eating disorder is not as obvious as if they were severely underweight (the disorder does occur in obese individuals but it is uncommon). The course of bulimia nervosa may be chronic or intermittent, with symptoms and remissions fluctuating over the years. Weight fluctuations are also common due to alternating binging and purging behaviours.

Bulimia nervosa is clinically diagnosed if the individual participates in recurrent episodes of binge eating; binge episodes are characterized by eating a larger amount of food than most people would eat in a specific amount of time and is accompanied by a lack of control over eating (i.e., can't stop eating) during this episode. In addition to the recurrent episodes of binge eating, the individual participates in inappropriate behaviour in order to prevent weight gain, such as self-induced vomiting or the misuse of laxatives. The binge eating and inappropriate behaviour must have occurred, on average, at least twice per week for three months and does not occur exclusively during episodes of anorexia nervosa. Finally, the individual's body shape and weight disproportionately influences their self-evaluation.[7]

Similar to anorexia nervosa, the causes of bulimia nervosa are not known. Cultural ideals and social attitudes toward body appearance are likely to contribute, as well as self-valuation based on body weight and shape. There is some evidence that obesity during adolescence or a genetic predisposition toward obesity contributes to the development of the disorder.[117] Other factors that are associated with bulimia nervosa are a history of sexual or physical abuse, substance misuse, anxiety disorders, low self-esteem, perfectionism, parental weight/body shape concern and peer pressure. Binge eating is typically triggered by depressed mood, interpersonal stressors, hunger following dietary restraint, or feelings of self-depreciation. The individual binge eats to reduce these feelings but self-criticism and depression tend to follow. Suicide attempts are relatively common.[107] Although preventive measures are not known at this time, early detection and interventions can reduce the severity of symptoms and improve prognosis.[118,119]

Treatment of bulimia nervosa usually involves a combination of individual therapy, family therapy, behaviour modification and nutritional rehabilitation. Cognitive-behavioural therapy focuses on self-monitoring of eating and purging behaviours and changing the thought pattern that leads the individual to binge and purge. Medication (i.e., antidepressants, antianxiety medication) may also be prescribed, particularly if the individual is experiencing depression or anxiety. If the bulimia nervosa is severe, the individual may have to be admitted to an eating disorders treatment program. Poor prognosis has been found to be associated with premorbid and paternal obesity, a history of substance misuse, and presence of a personality disorder.[105,116]

Bulimia nervosa

ICD-9: 307.51 ICD-10 - Bulimia nervosa F50.2

An individual with bulimia nervosa engages in binge eating behaviours and then attempts to compensate for the excessive food intake by purging. Individuals with bulimia nervosa are usually embarrassed about their eating problems and often feel out of control; therefore, they binge as inconspicuously as possible. The individual typically experiences anxiety, depression, and negative affect, using body weight and shape as the main measure of their self-worth. Self-esteem is low. The individual may have scars on the surface of their hand from contact with the teeth while pushing fingers down their throat to induce vomiting.[7] Frequent, repeated purging may lead to increased dental cavities and loss of dental enamel (from the acid in the vomit), and potential fluid and electrolyte abnormalities may cause serious medical complications (rarely even death).[7] Fatigue is common due to malnutrition and electrolyte imbalances. Irregular menstruation or amenorrhea may be present.[120]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	3	3	1	1	3	1	1	1	1

Section E - Mental retardation

Mental retardation is a permanent condition characterized by subaverage intelligence, which causes limitations in learning and adaptive functioning. Individuals with mental retardation may be able to live independently in the community and obtain various levels of employment, depending on the level of severity; as severity increases, the need for training and support may be required to complete even simple, daily tasks. This section describes the health states of individuals living with mild, moderate, or severe mental retardation.

Mental retardation occurs in all races and cultures, although there is a higher prevalence overall among males than females. Approximately 1-3% of the population are affected;[7,121,122] of all cases, the majority (about 85%)[7] are classified as having mild severity;[122] approximately 10% and 4% of cases have moderate and severe mental retardation, respectively.[7] The time of onset typically depends on the cause of the mental retardation. In general, mental retardation is caused by any condition or event that impairs the development of the brain before birth (prenatal), during birth (perinatal), or in childhood (postnatal). Specifically, potential causes include, but are not restricted to, chromosomal deficits (Down syndrome), inherited disorders (Fragile X syndrome, hypothyroidism), errors of metabolism, brain injury or infection (insufficient oxygen during birth, meningitis), prematurity or low birth weight, fetal malnutrition, drug or alcohol misuse during pregnancy (fetal alcohol syndrome), maternal infections (rubella or hypertension), and severe emotional neglect or abuse (including under-stimulation of the infant/child). Unfortunately, in many cases, no specific cause can be identified,[7,121] although the likelihood of identifying the particular cause increases with severity of mental retardation.

According to DSM-IV criteria,[7] an individual is diagnosed with mental retardation if they have significantly below average intellectual functioning, as defined by an intelligence quotient (IQ; measured using a standardized, individually administer/red intelligence test) at or below 70. In addition, limitations in adaptive functioning (i.e., effective coping of common life demands) are present in at least two of the following skill areas: communication, home living, self-care, self-direction, use of community resources, functional academic skills, social/interpersonal skills, work, leisure, health, and safety. Finally, onset must occur before age 18 years. The DSM-IV classifies mental retardation into four stages based on severity: mild (IQ score of 50-55 to approximately 70), moderate (IQ score of 30-35 to 50-55), severe (IQ score of 20-25 to 35-40), and profound (IQ score of less than 20-25). (The ICD10 has more specific IQ cut-offs; see the health state descriptions for each stage below.) The health state of an individual with profound mental retardation will not be described in this chapter because it accounts for only 1% of all individuals with mental retardation.[7]

Mental retardation is often suspected when the affected individual fails to meet age-appropriate developmental milestones. In infancy, abnormal development may be apparent by floppy or spastic muscle tone, lack of visual or auditory response, and/or inadequate sucking response.[123] Eventually, motor delays in sitting or walking and language and behavioural abnormalities may be evident, but often are not identified until the preschool period. In more severe cases, the symptoms tend to be more obvious, and appear at a younger age. In particular, some individuals may have noticeable physical or neurological abnormalities that may be suggestive of mental retardation, such as unusual facial features, a head that is too small or large, deformities of the hands or feet, and seizures.[121]

Mental retardation is a permanent condition, however the majority of individuals with mental retardation can receive comprehensive, individualized programs that are aimed at teaching the adaptive skills necessary to increase their level of independence: reading, writing, and basic math, taking care of personal needs (i.e., dressing, bathing), communicating with others, home living (i.e., cooking, cleaning the house), social skills (i.e., manners, playing games), and health and safety. Social programs are also important for the individual with mental retardation to gain self-esteem. In early adulthood, treatment is also directed at learning vocational skills to support employment; involvement in the workforce improves adaptive skills and the success of community living.[124] Emotional support for the family is also an integral part of treatment.

Mental retardation – mild

ICD-9 code: 317 ICD-10 – Mild mental retardation F70

An individual is diagnosed with mild mental retardation if they have an IQ score of 50-69,[6] and the majority of cases fall within this category. Individuals with mild mental retardation typically develop social and communication skills adequate for self support, but may need assistance during times of unusual stress. Academic skills can be acquired up to the 6th grade level. Given appropriate supports, individuals with mild mental retardation can usually live successfully in the community, either in independent or supervised settings, and 80% are employed (in mainly unskilled or semiskilled jobs).[123]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	1	1	2	2	1	1	1	1	1

Mental retardation – moderate

ICD-9 code: 318.0 ICD-10 – Moderate mental retardation F71

An individual is diagnosed with moderate mental retardation if they have an IQ score of 35-49.[6] About 10% of individuals with mental retardation fall within this category.[7] Individuals with moderate mental retardation can generally acquire adequate communication skills and benefit from social and occupational skills training, but their academic level does not usually progress beyond the 2nd grade level. They are slow in learning to speak and have fair motor coordination. The majority of individuals are able to manage unskilled or semiskilled work in sheltered conditions with supervision and guidance. They typically live in supervised settings and are able to attend to their personal needs and care under supervision.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	3	1	1	4	3	1	2	1	1	1

Mental retardation – severe

ICD-9 code: 318.1 ICD-10 – Severe mental retardation F72

An individual has severe mental retardation if they have an IQ score of 20-34.[6] Approximately 3-4% of all individuals with mental retardation fall within this category.[7] Individuals with severe mental retardation may learn to talk and communicate although they have only limited speech skills and vocabulary. The individual can contribute to simple and self-care tasks under close supervision. Motor coordination is poor. Most live in group homes or with their families; however, the likelihood of neurological, neuromuscular, visual, auditory, and cardiovascular conditions in severe mental retardation may require specialized nursing or other care. Self-injurious behaviour, including head-banging, biting, and scratching is not uncommon in children with severe mental retardation.

Classification

Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	4	1	1	5	4	1	3	1	1	1

Section F - Personality disorders

A personality disorder (PD) is characterized by a stable pattern of inner experience and behaviour that deviates significantly from the expectations of society, and can lead to marked impairments in social and occupational functioning. PDs are considered a major mental health problem because of their prevalence and the disability they produce.[69] Ultimately, individuals with a PD have difficulties with interpersonal relationships, and often demonstrate irritability, hostility, and fearfulness. Their personality traits (i.e., attitudes, thoughts, behaviours, temperament) are expressed inappropriately and become maladaptive. According to the DSM-IV,[7] there are ten PDs that are distinctively diagnosed and are grouped into three clusters based on descriptive similarities: Cluster A includes Paranoid, Schizoid, and Schizotypal PDs—individuals with these disorders likely appear odd or eccentric; Cluster B includes Antisocial, Borderline, Histrionic, and Narcissistic PDs—individuals with these PDs often appear dramatic, emotional or unpredictable; Cluster C includes Avoidant, Dependent, and Obsessive-Compulsive PDs—individuals with these disorders tend to appear anxious or fearful.[7] The functional limitations associated with all personality disorders are similar in terms of attribute levels, therefore only one health state is described for an individual diagnosed with an unspecified personality disorder.

PDs affect between 6% and 15% of the population.[125,126] The most common of the PDs are obsessive-compulsive (with a prevalence rate of 7.7% according to DSM-IV criteria), avoidant (6.6%), paranoid (5.6%), borderline (5.4%), and schizotypal (5.2%).[126] PDs typically become recognizable by adolescence or early adulthood, although some individuals may not seek clinical attention until much later. It is possible for a PD to become exacerbated after the loss of a significant supporting person or situation.[7] The course of a PD is relatively stable over time.

The DSM-IV diagnoses a PD if an individual exhibits maladaptive behavioural and cognitive patterns which are evident in at least two of the following areas: cognition, affectivity, interpersonal functioning, or impulse control. In addition, the pattern must be pervasive and inflexible across a wide range of personal and social situations, and cause clinically significant distress in social, occupational, or other areas of functioning. Evidence of the PD must have been present in at least adolescence or early adulthood, with a stable pattern of long duration. The pattern cannot be due to another mental disorder, or due to the physiological effects of a substance or a general medical condition.[7] Specific (types of) PDs require their own criteria, which are presented in the DSM-IV manual.[7] PD diagnoses apply only to the completely formed personality, thus, they are rarely made before age 18 and are therefore not usually assessed in children and adolescents.[69]

The causes of developing a PD are unknown. Researchers believe that a specific situation or event (e.g., loss of a parent or friend) can trigger the behaviours common in PDs, particularly events in early childhood that have the potential to influence behaviour in later life. A genetic vulnerability to developing a PD has also been suggested.[127,128,129] Social factors, such as parental neglect, overprotection, or abuse may contribute to personality or other psychiatric problems in children. Because onset of PDs is usually in adolescence, a time when the personality stabilizes and matures, individuals with PDs are prone to developing maladaptive coping mechanisms and low self-esteem.[127,128,129]

Overall, there is no cure for a PD, but treatments are available to improve prognosis. Depending on the PD, pharmacological interventions can be targeted at reducing impulsivity (e.g., olanzapine, neuroleptics) and depression (e.g., serotonin reuptake inhibitors); antipsychotics may be used in cases of distorted thinking. Psychotherapy (individual, group, or family) is directed towards management of the disorder, including education about the illness, support, and social skills training. Psychotherapy, however, may be difficult for an individual with a PD because they may be reluctant to build a trusting relationship with the therapist. Character modification may be necessary to improve mood instability and impulsive behaviours, or for the individual to learn ways to cope with rejection and abandonment fears, self-destructive behaviours, or other traits associated with the particular PD being treated. Inpatient care is rarely required.

Due to the generally low rate of compliance with treatment, the following health state describes the functional limitations associated with an individual diagnosed with a PD who is not undergoing treatment.

Personality disorder – unspecified

ICD-9: 301.83 ICD-10 – Personality disorder F60.x

Individuals with a PD exhibit the whole spectrum of personality features that do not allow for adequate social functioning. Some tend to be extremely unstable emotionally - feelings range from intense and inappropriate anger, to feelings of guilt and shame and depression, to feelings of inadequacy and inferiority. Others may show impulsive behaviour, sexual promiscuity or reckless driving, and possibly suicide attempts, particularly at times of crisis (i.e., a change in job or relationships, a therapist's or family member's vacation). Mentally, individuals with a PD may have an unstable self image and identity confusion, or may be hypersensitive to rejection and feel hurt by criticism or disapproval. Interpersonal relationships are extremely unstable as well; attitudes towards family members or friends may suddenly shift from great admiration and love to disappointment, dislike and anger. A major characteristic of a PD is social withdrawal or rejection. Individuals with a PD tend to have an impaired capacity for attachment. Family life is often disrupted, and occupational and social functioning are limited. Anxiety is experienced. Occasionally, individuals with a PD may perform self-harmful behaviours.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	3	1	2	4	3	1	1	1	1

Section G - Schizophrenia

Schizophrenia defines a group of disorders, characterized by a continuum of signs, the most prominent of which are distorted thought and perception. An individual with the most common type - paranoid schizophrenia - presents with various hallucinatory experiences and delusional thinking, either due to the hallucinations or to the wrong interpretation of real stimuli.[130] The group of so-called negative signs always develops in schizophrenia patients – flattened affect, lack of emotions and volition/will, etc.[131] In most cases, the individual has difficulty thinking clearly and making decisions, managing and expressing emotions, and relating to others; therefore, a substantial part of them is socially withdrawn. Depending on the presence of certain predominant symptoms, diagnosis can be one of five types: catatonic, paranoid, disorganized, undifferentiated, or residual schizophrenia.[7] This section will describe the functional limitations associated with each subtype. Although the course of the disease may be variable, with some individuals experiencing exacerbations and remissions, the health states discussed here refer to those individuals in which the disease and disturbances of the disease remain chronic.

Schizophrenia affects approximately 1% of the population[132] and is observed worldwide. Onset is usually between the late teens and the mid-30s;[1] late-onset cases (after age 45) have been reported but onset before adolescence is rare. Men and women are affected equally, but onset is earlier in men (between ages 18 and 25) than in women (between ages 25 and 35); late-onset cases are more common in women.[1,7,133] Although the onset of schizophrenia may be sudden, most cases display a variety of signs and symptoms gradually. In general, symptoms characteristic of schizophrenia involve dysfunctions in both the cognitive and emotional domains: perception, inferential thinking, attention, language and communication, affect, fluency, thought and speech production, behavioural monitoring, volition and drive.

Though there are some instrumental methods for evaluation of the level of structural and functional brain dysfunction in patients with schizophrenia, diagnosis is made based on clinical signs, case and family history.[130] The DSM-IV clinically diagnoses schizophrenia if two or more of the following symptoms are present for a significant portion of time during a one-month period: delusions, hallucinations, disorganized speech, disorganized or catatonic behaviour, or negative symptoms including affective flattening (restricted emotional expression), alogia (restricted thought and speech), or abulia (restricted initiation of goal-directed behaviour). Furthermore, since onset, the symptoms result in social or occupational dysfunction. There must also be continuous signs of the disturbance that persist for at least six months. Finally, a diagnosis of schizoaffective disorder or mood disorder with psychotic features has been ruled out, and the symptoms/disturbance is not due to the physiological effects of a substance or a general medical condition.[7] Diagnostic criteria for each subtype are described in the health states presented below.

The cause of schizophrenia is unknown, but there is some evidence that genetics are a factor: first degree biological relatives of individuals with schizophrenia are about 10 times more likely than the general population to develop it.[7,133,134] Environmental factors that occur during development, such as a viral infection (e.g., prenatal exposure to the flu) or hormonal and physical changes during puberty may also trigger the disorder. Functional abnormalities in the brain may be a cause or a consequence of schizophrenia.[1]

In spite of the number of antipsychotic agents implemented into clinical practice, prognosis of schizophrenia is still relatively poor: only a quarter of patients show full psychopathological remission and 56% show social remission.[130] In 2004, schizophrenia was one of the top ten leading causes of disability (measured in years lost to disability) globally.[3] Approximately 1/3rd of people diagnosed with schizophrenia require institutionalization for the rest of their lives,[135] and about 40% of individuals will attempt suicide over the course of the disease (approximately 10% will successfully complete suicide)[1,7,136] due to psychosis and/or depression. However, with early diagnosis and effective treatment, individuals with schizophrenia can prevent further symptoms and increase the likelihood of recovery. Treatment generally consists of pharmacological interventions and psychotherapy, either in isolation or more effectively, in combination. Hospitalization may be required to treat delusions or hallucinations, particularly

if the individual is having suicidal thoughts, is unable to care for themselves, or has severe problems with drugs or alcohol. Antipsychotic medications help to reduce some symptoms of schizophrenia but have considerable side effects, resulting in a high potential for noncompliance.

Schizophrenia, regardless of subtype, has many implications for an individual's health state. Social withdrawal is apparent early in the disease process, therefore most have limited social contacts and the majority (60% to 70%) do not marry.[7] Academic performance is typically impaired; consequently, many are unable to finish school. Occupational functioning is also limited: reported employment rates vary significantly but are generally between 10% and 40%.[130,137,138] Emotional and financial losses result, as do reduced self-esteem, hopelessness and isolation. Most importantly, there is a stigma associated with schizophrenia that leads to embarrassment due to lack of public understanding of the disease.

Although it is not infrequent for individuals to have symptoms characteristic of more than one subtype, diagnosis is based on the most prominent symptoms, in this order: Catatonic type is designated when the prominent symptoms are catatonic (a state of muscular rigidity and/or mental stupor), regardless of the presence of other symptoms; Disorganized type is assigned whenever disorganized speech, disorganized behaviour and flat or inappropriate affect are prominent (unless Catatonic type is present); Paranoid type is designated whenever delusions or hallucinations are frequent and prominent (unless Catatonic or Disorganized type is present); Undifferentiated type is assigned whenever there are prominent, active-phase symptoms that do not meet the criteria for Catatonic, Disorganized, or Paranoid types; and finally, Residual type is designated when there is continuous evidence of the disorder but active-phase symptoms are not present.[7]

The following health states describe an individual diagnosed with each subtype of schizophrenia during which time the disturbances and symptoms of the disease are active and chronic. The descriptions do not capture the individual while undergoing treatment.

Catatonic schizophrenia

ICD-9: 295.2 ICD-10 – Catatonic schizophrenia F20.2

An individual is diagnosed with catatonic schizophrenia if their symptoms are dominated by at least two of the following: motor immobility or stupor, excessive motor activity, extreme negativism (a state in which people resist efforts to physically move their limbs or themselves), peculiarities of involuntary movement, and echolalia (parrotlike repetition of a word or phrase just spoken by another person) or echopraxia (the repetitive imitation of the movements of another person).[7] Onset of this subtype is typically sudden. In general, an individual with catatonic schizophrenia might represent two alternative conditions similar in psychopathological sense: at one end, the individual may be in a catatonic stupor and seem immobile and unresponsive or negativistic, shown in their resistance to instructions (they often do the opposite of what they are asked) or maintenance of a rigid posture if attempted to be moved. On the other hand, they may demonstrate catatonic agitation characterized by uncontrolled excitement and repetitive stereotypic movements. Mimicry in terms of echolalia and echopraxia is common.[7]

During periods of stupor and immobility, individuals are unable to move around and take care of personal needs, and therefore require help with daily tasks such as getting dressed or eating. The use of hands and fingers are restricted throughout periods of rigidity. During the episodes of excitement, the individual will require supervision in order to avoid harming themselves or others, but they can generally function well. Speech disturbances are common: some individuals may not speak at all, and if they do speak, their speech is generally meaningless or consist of echolalia and repetitive chatter. Individuals with catatonic schizophrenia might experience moderate pain and discomfort, particularly due to maintaining uncomfortable postures for long periods of time; fatigue may also result. Individuals

with this subtype often have depression and are socially isolated; memory and thinking is also impaired, particularly during periods of stupor due to lack of concentration.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	4	4	2	5	5	1	4	1	1	4

Disorganized schizophrenia

ICD-9: 295.1 ICD-10 –Hebephrenic schizophrenia F20.1

The predominant symptoms of an individual diagnosed with disorganized schizophrenia are significantly disorganized speech, disorganized behaviour, and flat or inappropriate affect.[7] Delusions or hallucinations may be present but are disconnected and do not revolve around a coherent theme. Language is generally incoherent and may be accompanied by silliness and laughter without an appropriate stimulus (often an individual with disorganized schizophrenia will laugh when experiencing pain or cry when hearing a joke that others find funny). The individual may even make up new words or use words in strange ways. Behaviour is inappropriate and there is a lack of goal orientation, which may lead the individual to disregard bathing, meal preparation and other activities of daily living. Auditory hallucinations often cause limitations in hearing. The individual may display unpredictable agitation (i.e., shouting and swearing) or inappropriate sexual behaviour (i.e., public masturbation). Disturbances in thought are common; the individual will tend to have difficulty organizing ideas, and often do not think rationally or logically. This subtype generally follows a continuous course without periods of remission, and onset is abrupt. Disorganized schizophrenia tends to be the most severe of all subtypes of schizophrenia.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	2	4	1	5	5	1	3	2	1	1

Paranoid schizophrenia

ICD-9: 295.3 ICD-10 –Paranoid schizophrenia F20.0

An individual is diagnosed with paranoid schizophrenia if their predominant symptoms include delusions or auditory hallucinations which attempt to maintain cognitive functioning and affect, and they do not experience symptoms characteristic of the Disorganized or Catatonic types.[7] The delusions are usually organized around a coherent theme, and are often persecutory or grandiose. More specifically, persecutory delusions often center on the belief that others are "out to get them" in some way. Consequently, the individual may develop grandiose delusions in which they are extremely famous, important or powerful and are protecting themselves from the perceived persecutions. Hallucinations generally relate to the delusional theme. Onset of this type tends to be later in life than other subtypes of schizophrenia, but the features are relatively more stable over time. Paranoid schizophrenia tends to be the least severe of all subtypes of schizophrenia.[7]

Individuals with paranoid schizophrenia have minimal impairment in functioning unless they act upon their delusional thoughts. Overall, they may be depressed, angry and argumentative with moderate levels of anxiety. Hearing is impaired due to auditory hallucinations; also, the individual is often distracted therefore the quality of their hearing (e.g., receiving information) is limited. Emotionally, an individual with paranoid schizophrenia appears deprived of emotions and exhibits flattened affect. Their memory and thinking is impaired with delusions and hallucinations as they are often confused and indecisive about what is real and what is imaginary. Their capacity to sustain social relationships is impaired due to hostile and suspicious behaviour, and often delusional jealousy occurs with the deep belief that their sexual partner is unfaithful. Severe attacks may require hospitalization as persecutory themes may cause the individual to become violent and/or suicidal. Paranoid schizophrenia may have a considerably better prognosis than other types of schizophrenia because the individual is generally able to maintain occupational functioning and independent living.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	3	1	4	4	3	1	2	1	1

Undifferentiated schizophrenia

ICD-9: 295.8 ICD-10 –Undifferentiated schizophrenia F20.3

An individual is diagnosed with undifferentiated schizophrenia if they experience symptoms that meet the criteria for schizophrenia, but do not meet the criteria for paranoid, disorganized, or catatonic types.[7] Typically the individual will have fragments of different symptoms (i.e., delusions, hallucinations, incoherence); although these symptoms may remain over a long period of time, a stable pattern of characteristics may emerge later in life. The functional limitations associated with this subtype of schizophrenia include personal hygiene issues, impaired emotional state (depression), some thought disorders including the inability to concentrate, and social functioning limitations caused by social withdrawal.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	3	3	1	4	3	1	1	1	1	1

Residual schizophrenia

ICD-9: 295.5 ICD-10 –Residual schizophrenia F20.5

An individual is diagnosed with residual schizophrenia if they are exhibiting two or more symptoms that meet the criteria for schizophrenia, but prominent delusions, hallucinations, disorganized speech, and disorganized or catatonic behaviour are absent.[7] In general, this diagnosis is made when an individual has experienced at least one episode of schizophrenia but is currently not exhibiting symptoms or the symptoms are relatively minor. Nevertheless, individuals with residual schizophrenia experience a lack of motivation and interest in life, and have considerable impairments in personal hygiene practices. In addition, illogical thinking, social isolation or withdrawal, and some deficiency in speech are common. The course of this subtype may be continuous over many years (possibly involving acute exacerbations).[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	2	3	1	2	3	1	2	1	1	1

Section H - Substance use disorders

This section will describe the most prevalent substance use disorders. Substance use disorders are those that relate to the taking of a drug (including alcohol) of abuse, including prescribed and over-the-counter medications, as well as illicit drugs.[7] The substances highlighted in this section include alcohol, cannabis, heroin, benzodiazepines, and stimulants.

It must be noted that psychoactive substance use and abuse are one of the major risk factors contributing to the global burden of disease: in 2000, approximately 4% of the global burden was attributed to alcohol and 0.8% to illicit drugs.[139] In addition, alcohol and illicit drugs are associated with over 80 recognized disease and injury conditions,[139] all of which are preventable.

The clinical effects vary depending on the substance used, the duration, and dosage. The most dangerous form of substance abuse occurs when users mix several drugs. The health states described below do not consider the limitations associated with use of combined substances. For each substance, the functional limitations described refer to: abuse (chronic, mild to moderate or severe, or both), overdose, acute withdrawal with treatment, and remission. Coma is often the result of an overdose but the health state descriptions for overdose do not capture the comatose state. Coma will, however, be described in another document in this series: Neurological Disorders. There are also two sequelae specific to chronic alcohol abuse that will not be described in this chapter. Liver cirrhosis is the disease in which the major pathogenic mechanism is the gradual replacement of damaged liver tissue with connective tissue, thus leading to gradual but irreversible liver function decrease. This condition often results from long-term alcohol and other hepatotoxic substance abuse. Hepatic encephalopathy is damage to the brain and nervous system that results from liver disorder complications, and causes changes in reflexes and consciousness. Both of these conditions will be described in the Digestive Disorders document of this series.

The DSM-IV defines substance use disorders in terms of dependence and abuse, and substance-induced disorders in terms of intoxication and withdrawal; other DSM-IV diagnoses for substance use disorders, such as substance-induced delirium or substance-induced mood disorder, will not be described in this section. The criteria for diagnosis of substance dependence, substance abuse, substance intoxication, and substance withdrawal will be described below; note that these criteria are applicable to all classes of substances.

Substance dependence is diagnosed if the individual has a pattern of repeated self-administration of the substance and three or more of the following symptoms, which occur at any time in the same 12-month period: 1) tolerance (need for increased amounts of the substance to achieve the desired effect); 2) withdrawal (a change in behaviour (with physical or mental associations) that results from decreased blood or tissue concentrations of the substance); 3) use of the substance over a longer period than intended or in larger quantities; 4) persistent desire for the drug or unsuccessful efforts to control/cut-down use of the substance; 5) much time spent in activities that are necessary to obtain, use, or recover from the substance; 6) social, occupational or recreational activities are limited or avoided due to substance use; and 7) substance use continued despite knowledge of physical or psychological problems that are likely caused by or exacerbated by the substance.[7]

Substance abuse is different from dependence in that it does not include any symptoms of tolerance/withdrawal or compulsive patterns of use; substance abuse rather includes the harmful consequences of repeated substance use. In particular, an individual is diagnosed with substance abuse if their pattern of substance use leads to clinically significant impairment as shown in at least one of the following: 1) major role obligations have not been fulfilled as a result of the recurrent substance use; 2) substance use is initiated or recurrent in physically hazardous situations (e.g., driving a car); 3) use results in repeated substance-related legal problems (i.e., drug trafficking or driving under the influence); and 4) the substance use is continued despite persistent social or interpersonal problems that are caused or worsened by the effects of the substance. In addition, these manifestations must have occurred repeatedly during

a 12-month period and the symptoms have never met the criteria for substance dependence (in that particular class of substances).[7]

Substance intoxication is diagnosed when a reversible substance-specific syndrome develops due to recent ingestion of (or exposure to) a substance. In addition, during or shortly after use, the physiological effects of the substance on the central nervous system cause behavioural and/or psychological changes. The most common changes are impairments in perception, wakefulness, thinking, attention, judgment, psychomotor behaviour, and interpersonal behaviour. Finally, the symptoms of substance intoxication are not due to a general medical condition or are not better accounted for by another mental disorder.[7]

When an individual stops or decreases use of a substance after heavy, prolonged use, the individual may experience substance withdrawal, the development of behaviour changes with physiological and cognitive associations. For a diagnosis, this change causes clinically significant distress or impairment in important areas of functioning, such as social or occupational, and these are not due to a general medical condition or better accounted for by another mental disorder.[7] Most of the symptoms of withdrawal are simply the opposite as those seen in intoxication using the same substance. Individuals going through withdrawal typically have a craving to re-administer the substance in order to prevent or relieve the withdrawal symptoms.

Remission is defined by the DSM-IV in terms of four stages: an individual is in early full remission if no criteria for abuse or dependence is met for at least one month but less than 12 months, or is in early partial remission if one or more criteria for abuse or dependence is met (but the full criteria have not been met) for at least one month but less than 12 months.[7] An individual is in sustained full remission if none of the criteria for abuse or dependence has been met at any time during the previous 12 months or longer, or is in sustained partial remission if the full criteria for dependence has not been met in the previous 12 months or more, but one or more criteria has been met for abuse or dependence.[7] For the purposes of the health states described below, remission is defined as no symptoms present for at least one month.

Part 1 - Alcohol abuse / harmful alcohol use

Alcohol is a central nervous system (CNS) depressant that is produced by fermenting or distilling various fruits, vegetables or grains. The chemical name is ethanol or ethyl alcohol; in its pure form, it is a clear, colourless liquid. The effects of alcohol depend on the amount of pure ethyl alcohol consumed; one serving contains between 10 and 15 g of ethanol.[140] The alcohol enters the bloodstream from the gastrointestinal tract and is broken down by liver enzymes. As the blood-alcohol concentration (BAC) increases (i.e., the amount of alcohol in the bloodstream), the nervous system becomes more depressed; thinking, judgment, and perception are impaired and reaction times are slower.

Moderate use of alcohol (i.e., up to two drinks per day) is generally not considered harmful for most adults. An average drink equals one bottle of beer or wine cooler (12 ounces), one glass of wine (5 oz.), or one 1.5 oz. glass of distilled spirits (i.e., whisky). Binge drinking occurs when an individual consumes a large quantity of alcohol (five or more drinks for men, four or more for women)[140] in one sitting and is greater cause for concern. The first episode of alcohol intoxication is often in the mid-teens, despite the fact that the Canadian legal age to drink and purchase alcohol is 18 or 19 years, depending on the province.

Alcohol is the most popular drug of abuse.[141] In 1996, a national U.S. survey found that 70% of men and 60% of women consume alcohol.[7] About 1.1% of adults in the U.S. drink on a daily basis;[141] the 12-month prevalence of alcohol use disorders is between 7-10%.[140,142] The highest prevalence of alcohol abuse is between the ages of 26 and 34 years.[7] Individuals with alcoholism come from all levels of education and socioeconomic status. Alcohol is accountable for high levels of mortality, morbidity and social problems with more than 60 causes of death attributed to alcohol consumption.[139] Often the typical course consists of periodic or weekend binges with little to no alcohol consumption during the week. However, over time, episodes of drinking become more frequent. Alcoholism is a progressive disease that is often long-term, relapsing and possibly fatal. The individual with alcoholism will often devote long periods of time to consuming alcohol, despite the psychological and physical consequences.

An individual with alcoholism who is confronted about their drinking problem typically becomes hostile and defensive. The following symptoms strongly suggest alcoholism: craving (the need for daily or episodic use), impaired control (the inability to stop drinking once drinking has begun), physical dependence (withdrawal if stopped abruptly), and tolerance (the need to consume increasing amounts to achieve the same effect). It is likely that a number of factors influence the development of alcoholism. Risk factors include: genetics (individuals with a parent who abuses alcohol are more likely to abuse alcohol themselves; risk increases with the number of family members affected),[7] nutritional deficiencies, and endocrine imbalances. Psychological factors likely also play a role: depression, desire for relief from anxiety, desire to avoid responsibility, low self-esteem, conflict in family relationships, certain personality traits such as isolation or loneliness, and/or sexual immaturity. Sociocultural factors include the availability of alcoholic beverages (including price), social attitudes that approve frequent consumption and drunkenness, lifestyle, peer pressure, and stress. In addition, individuals who start drinking at an early age (i.e., 14 years or younger) have increased risk for developing alcohol dependence at some point in their lives.[143]

The ingestion of alcohol produces symptoms of intoxication that are characterized by mental and/or physical changes such as mood lability, impaired judgment, poor concentration, and inappropriate sexual or aggressive behaviour.[7] Slurred speech, unsteady gait, lack of coordination, and impairment in memory and/or attention are also present.[7] Severe intoxication can lead to amnesia (blackout) of the events that occurred when the drinking took place. Intoxication lowers social inhibitions, produces euphoria, increases confidence, and represses fears in the drinker. Existing emotions can also be magnified: if the individual is angry, they may become hostile or aggressive; if the individual is depressed, they feel more depressed and may be suicidal.

Individuals who abuse alcohol may consume it in dangerous situations, such as before driving a car. Therefore, the individual may encounter legal difficulties as a result (i.e., Driving Under the Influence), or cause car crashes. In fact, alcohol accounts for as many as 55% of fatal car crashes in the U.S.[7] Almost every organ in the body is affected by alcohol and can develop serious complications that may cause premature death. Up to 15% of individuals who consume alcohol heavily over long periods of time develop liver cirrhosis and pancreatitis.[7] Anemia is common; poor eating habits can lead to severe nutritional deficiencies. The muscles of the heart may deteriorate over time, potentially leading to heart failure. The immune system becomes weakened causing increasing susceptibility to infections. Heavy drinking over time has also been associated with the development of some cancers (e.g., throat, mouth, liver), high blood pressure, and cardiac and brain damage. Men who chronically abuse alcohol may experience reduced testosterone levels, erectile dysfunction, sterility, enlargement of the breasts and decreased testicular size. Women may experience menstrual irregularities, early menopause, and potential infertility. Alcohol use during pregnancy can lead to spontaneous abortion, or side-effects to the baby including fetal alcohol syndrome (i.e., causing growth retardation), abnormal features of the face and/or head, or potential central nervous system abnormalities (i.e., mental retardation). In general, chronic use of alcohol may reduce an individual's life expectancy by 15 years.[141] There is a 15% lifetime risk for suicide among individuals with alcoholism,[141] and 25% of all suicides are related to alcoholism.[144]

Alcohol withdrawal is characterized by withdrawal symptoms that develop about four to 12 hours after the cessation of prolonged, heavy alcohol intake,[7] and is often referred to as a "hangover". Symptoms of withdrawal include: headache, autonomic hyperactivity (i.e., sweating), hand tremors/shaking, insomnia, anxiety, anorexia, dry mouth, and nausea or vomiting. The individual may become withdrawn and profoundly depressed. Sleep disturbances can persist for months. Up to 5% of individuals with alcoholism experience severe complications of withdrawal, such as grand mal seizures, delirium or tremors.[7] The individual will typically ingest more alcohol to avoid or ease the withdrawal symptoms.

Detecting and treating alcoholism is complicated by denial.[144] Treatment includes the drug naltrexone, a medication that disrupts the chemical activity in the brain sites that give pleasure from alcohol, thereby reducing the cravings associated with alcohol. Disulfiram is a medication that blocks the metabolism of alcohol, whereby producing toxic symptoms and causing the individual to vomit and/or suffer a severe headache if they have a drink. It is sufficiently uncomfortable that the individual will not risk ingesting alcohol and is therefore considered a very effective treatment if the individual is compliant. Detoxification under medical supervision can help to control the symptoms of withdrawal; an IV injection(s) of glucose may be necessary to control hypoglycemia. Supportive programs that include detoxification, rehabilitation, and aftercare achieve the best long-term results. Individuals need a strong support system; Alcoholics Anonymous (AA) is a well-known systematic support group that is very common and very effective in combating alcohol abuse. Exercise and proper nutrition are also very important aspects in preventing relapse. Individuals who are in remission must continue to avoid all alcoholic beverages—total abstinence is the only effective treatment. Once in remission, it is highly likely that an individual recovering from alcoholism will lose control after their first drink and severe problems will develop again.[7]

Alcohol abuse (mild to moderate)

ICD-9: 303.9 ICD-10 – Harmful Alcohol Use F10.1

This health state refers to an individual who harmfully uses alcohol on a mild to moderate scale. The individual continues to use alcohol despite the consequences in terms of mental and physical health. Social, occupational and familial relationships are affected. As the individual continues to drink in excess, the risk that they will become a severe abuser of alcohol and experience the functional limitations associated with the health state below (Alcohol abuse – severe) increases.[7,144]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	2	3	2	2	2	1	1	1	1

Alcohol abuse (severe)

ICD-9: 303.9 ICD-10 – Harmful alcohol use F10.1

Alcoholism is characterized by impaired control over drinking, preoccupation with the drug alcohol, and continuing use of the substance despite adverse consequences in the individual's life. Continuing misuse of alcohol interferes with an individual's physical and mental health, social and family relationships, and academic and/or occupational responsibilities. Impairments in memory and perception are especially common. Other signs of severe abuse include denial, blackouts, and morning drinking (to avoid withdrawal symptoms). Anxiety and depression are common, particularly among females who abuse alcohol.[7,144] Expressive speech may deteriorate, in particular, as consumption increases. Tremors and decreases in fine motor functions are likely to present.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	3	3	3	4	3	3	2	1	1	2

Alcohol overdose

ICD-9: 980.0

Alcohol affects the nervous centers responsible for respiratory and cardiovascular systems control. An alcohol overdose can lead to depression of these involuntary actions, possibly resulting in death (e.g., due to respiratory arrest). Warning signs include low pulse rate, decreased respiratory rate, low blood pressure, mental confusion, seizures, vomiting, and general non-responsiveness. Treatment of an alcohol overdose typically requires hospitalization and medical supervision of the individual, administration of I.V. fluids (glucose), and if necessary, a gastric lavage or intubation.[7,144]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	4	4	1	5	5	1	4	1	1	1

Alcohol treatment (acute withdrawal with treatment)

ICD-9: 291.81, 94.63

The treatment phase for an individual with alcoholism begins with management of withdrawal. During withdrawal, the individual has many physical complaints including headaches, nausea or vomiting, and in severe cases, hallucinations and delirium. Treatment includes possible hospitalization, medication for the withdrawal symptoms, and social support therapy. Relapses are common; the outcome of treatment over time depends on the motivation and confidence of the patient.[7,144]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	2	3	3	4	4	3	1	1	1	1

Alcohol remission

ICD-9: 303.03

An individual in remission from alcohol abuse must alter their lifestyle; they must avoid the people and places that they visited while they were abusing alcohol to prevent the desire to drink. Many also attend weekly support meetings and even take medications to avoid a relapse. Though individuals may achieve stable remission from alcoholism, many of them continually live with the constant urge to drink.[7,144]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	2	1	1	2	1	1	1	1	1

Part 2 - Harmful heroin use

Heroin is a semi-synthetic opioid, a class of drugs that are often prescribed as analgesics, anesthetics, antidiarrheal agents, or cough suppressants.[7] Opiates are derived from the opium poppy and depress the central nervous system while producing mind-altering effects. Some opiates are used to treat severe acute pain, such as Morphine or OxyContin, a natural substance in the opium poppy; however, opiates are not appropriate to treat chronic pain because there is a high potential for addiction. Heroin is made by chemically changing the properties of morphine. It is one of the most commonly misused drugs in the opioid class.[7] The supply for heroin is only available through illicit markets.

Heroin use and abuse commonly begins in the late teens or early 20s and has a male to female ratio of 3:1.[7] Prevalence for heroin is around 1% of the general population.[7,145] Increasing age is associated with increased prevalence. Dependence generally begins after age 40 years.[7] Heroin addiction is found at every income level, age and social standing.

Heroin is typically taken by injection, but it can be smoked or ingested in tablet or liquid form. When it is heated and burned, heroin fumes are released and can be inhaled. It can also be added to cigarettes (tobacco or marijuana). Drug (urine) tests can detect heroin for 12-36 hours after administration.[7] The effects of heroin use can be felt in seconds if the drug was administered intravenously; if the drug was snorted, inhaled or injected into a muscle under the skin, the effects can be felt in minutes. In general, mental functioning is clouded due to depression of the central nervous system; with large doses, cardiac function and breathing can be slowed so much as to cause coma or even death. The essential feature of heroin intoxication is the presence of significant behaviour or psychological changes that develop during or shortly after heroin use.[7] Symptoms include: initial euphoria followed by apathy, pupillary constriction, drowsiness (or even coma), slurred speech, memory impairments, inattention to the environment (possibly to the point of ignoring harmful events), dysphoria, psychomotor agitation, impaired judgement, and impaired social or occupational functioning. After the initial effects, users feel drowsy. Symptoms of heroin intoxication typically last for several hours. The severity of these symptoms depends on the dose and tolerance level. At large doses, the individual cannot be aroused. With regular use, tolerance develops and therefore the individual requires more of the drug to achieve the desired effect. Over time, higher doses contribute to physical dependence and addiction.

An individual that is dependent on heroin has a regular pattern of compulsion in taking the drug and typically plans their daily activities around obtaining and using heroin. In addition, the individual has a significant level of tolerance and experiences withdrawal symptoms on abrupt termination of the substance.[7] The physical dependence of heroin generally increases in intensity with increased dosage and duration of using. Individuals who abuse heroin but who are not dependent on the drug typically do not use as often or do not develop significant withdrawal symptoms.[7] However, they may encounter legal difficulties due to intoxication or possession, as do individuals with dependence. Psychological dependence involves craving the drug and a compulsion to continue use.

Withdrawal develops after the cessation of heroin use that has been heavy and prolonged.[7] It is not life threatening but causes severe discomfort. Withdrawal symptoms typically begin within 5-12 hours after the last dose, peak between 36-72 hours and are usually over within 7-10 days. Severity of the symptoms increases with the size of the dose and duration of dependence. Symptoms of withdrawal generally consist of anxiety, restlessness, and aches often located in the back and legs; sensitivity to pain and a desire to obtain heroin (i.e., cravings) tend to accompany acute withdrawal symptoms. Other symptoms include: uneasiness, irritability, diarrhea, anorexia, abdominal cramping, nausea, vomiting, runny nose, watery eyes, sweating, chills and shivering, goose bumps, muscle spasms, pain in the bones and muscles, and yawning. Anxiety and insomnia are very common and may persist for months; emotional depression can last for years.[146] Full recovery may not be complete for six months or longer.

Treatment of heroin addiction varies depending on the individual, but is most effective when the abuse is identified early. Typically individuals are prescribed methadone, a synthetic opiate that relieves the cravings, minimizes

withdrawal symptoms, and blocks the effects of heroin (therefore individuals who continue to use will no longer feel the effect and consequently have no incentive to continue using). Methadone programs help with remission as well; methadone is medically safe even if used continuously for more than 10 years.[147] Through a methadone maintenance program, the individual receives a stable, legal supply of methadone which is only supplied and taken orally once per day. Despite the potential for methadone addiction, the individual is able to participate in other aspects of therapy and is slowly weaned from the drug once they are confident that they can live a more normal life.

Medication alone does not stop the addiction. Other treatment options include detoxification programs that aim to minimize the severity of the withdrawal symptoms and other medical complications. Detoxification typically takes about one week, and can be through a special clinic or at home with the help of a doctor and support from family and friends. Detoxification programs, however, are only useful when it leads into long-term treatments (e.g., methadone). The best drug-free treatments appear to be therapeutic community residential programs that last three to six months.[147] Cognitive interventions focus on educating and training the individual to build a new life that is socially productive. Unfortunately, relapse is common in recovering heroin addicts.

Heroin abuse / harmful heroin use

ICD-9: 304.0 ICD-10 – Harmful heroin use F11.1

Heroin is a highly addictive drug in the opioid family that has mind-altering effects on the user. Individuals who use heroin become both physically and psychologically dependent on it. When this dependence occurs, the addict's primary purpose in life becomes seeking out and using the drug. Individuals who use heroin tend to be moody, anxious, and at risk of depression. Those who use regularly over the long-term may experience infectious diseases due to injections with contaminated needles (i.e., HIV/AIDS, Hepatitis B and C, tuberculosis), needle marks, collapsed veins, immunological changes, arthritis, pulmonary disorders, abscesses, infection of the lining and valves of the heart, bacterial infections, and neurological disorders. Malnutrition typically results due to decreases in appetite. Difficulties in sexual functioning are common; women have irregular menstrual cycles and disturbed reproductive function; men often experience erectile dysfunction. Smoking heroin can often lead to pneumonia and other lung conditions. Criminal activity, violence and prostitution are also common among heroin addicts.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	3	2	1	4	4	3	1	1	1	1

Heroin overdose

ICD-9: 965.01, E850.0

Individuals who overdose on heroin can slip into a coma or even die if they do not get immediate medical attention. This health state, however, refers to an individual who has overdosed on heroin but is not in the coma state. Drugs, such as Naloxone, need to be administered in order to reverse the unconsciousness caused by a heroin overdose.[148] Restraints are put in place before administering Naloxone because an addict recovering from unconsciousness is agitated, delirious and combative. Observation for at least 24 hours must also occur to make sure that there is no further respiratory depression. An overdose of heroin is typically unintentional and is a particular risk on the street. Death from overdose is not uncommon.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	4	4	3	5	5	3	1	1	1	1

Heroin treatment (acute withdrawal with treatment)

ICD-9: 292.0, 94.66

Withdrawal from heroin is very physically uncomfortable but it is not life threatening. Peak intensity usually occurs 36-72 hours after the last administration of heroin, and may last up to two weeks. Some symptoms of withdrawal include: excessive yawning, bouts of chills alternating with bouts of excessive sweating, hot flashes, tremors, increased irritability, insomnia, depression, muscle spasms, and severe aching. Prescription drugs can be administered to help with the short-term symptoms of withdrawal as well as for the long-term cravings experienced by addicts. Therapy is also an important part of treatment as it helps the individual to regain their life after addiction. Most of the initial therapy occurs while the individual is living in a residential unit.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
4	2	4	3	4	3	3	1	1	1	2

Heroin remission

ICD-9: 304.03

Recovering heroin addicts may continue to take prescription drugs that help to alleviate their cravings for heroin, such as methadone, which can be taken indefinitely if necessary. However, they still need to make important lifestyle and behaviour changes, such as avoiding people and/or places that are associated with their former drug habit, if they want to stay drug-free. Depression, anxiety and insomnia may still remain from the withdrawal syndrome and can last for many years. Residual physical symptoms and impairments in concentration, memory and thinking may also remain. Opioid cravings typically continue over the long term; about 20-30% of individuals with opioid dependence achieve long-term abstinence.[7] A person returning to heroin after a period of remission has a high risk of fatal overdose because they lose their tolerance.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	3	2	4	2	2	1	1	1	1

Part 3 - Harmful benzodiazepine use

Benzodiazepines are antianxiety substances considered in the Sedative-, Hypnotic-, or Anxiolytic-Related (SHA) Disorder chapter of the DSM-IV. SHAs are a class of substances that include all prescription sleeping medications and virtually all prescription antianxiety medications. In general, these substances are central nervous system depressants. At large doses, they can be lethal, especially when mixed with alcohol.[7] SHAs are available by prescription or through the illegal market.

Benzodiazepines are the most commonly used substance in the class of SHAs, and are among the most routinely prescribed medications (e.g., Valium, Xanax) for treating chronic anxiety. Individuals seek relief from anxiety and tension, with the desired effects consisting of relaxation and calmness. Some individuals may take benzodiazepines to "come down" from cocaine or amphetamines.[7] Sustained use of benzodiazepines often leads to physical dependence, and sudden stopping of the drug can be life threatening. Individuals using benzodiazepines for more than one month must be weaned off the drug in order to reduce the number and severity of withdrawal symptoms.

Approximately 10% of Canadians report using benzodiazepines at least once per year, and 1 in 10 of these individuals continue using for more than one year.[146] Women are more likely to use benzodiazepines for medical reasons,[146] putting them at higher risk for abuse.

An individual with physiological benzodiazepine dependence has considerable levels of tolerance and withdrawal. A diagnosis of dependence is only made, however, if the individual also shows evidence of other problems, such as intense drug-seeking behaviour and avoiding/reducing activities in order to obtain the drug.[7] Intoxication from benzodiazepines develops during or shortly after use of the drug and closely resembles alcohol intoxication, causing clinically significant behavioural or mental changes. For example, sexual behaviour may become inappropriate and aggressiveness may be seen; judgment, social, and occupational functioning likely becomes impaired. At least one of the following signs also occurs: slurred speech; incoordination; unsteady gait; nystagmus (uncontrolled eye movements); attention or memory impairment; and/or stupor or coma.[7] Intense and/or repeated intoxication may cause severe depression, which can lead to suicide attempts and completed suicides.

Benzodiazepine withdrawal occurs after the cessation or reduction in heavy and prolonged benzodiazepine use.[7] Shortly after (a few hours to a few days after last use), the individual experiences at least two of the following: autonomic hyperactivity (increased heart rate, sweating); hand tremors; insomnia; nausea or vomiting; quick-passing visual, tactile, or auditory hallucinations or illusions (often in the context of delirium, particularly in severe withdrawal); psychomotor agitation; anxiety; and/or grand mal seizures.[7] These symptoms cause clinically significant impairment in social, occupational or other areas of functioning. And finally, these symptoms cannot be due to a general medical condition and are not better accounted for by another mental disorder. Some individuals can develop a delirium that may potentially be life threatening. Grand mal seizures occur in about 20-30% of individuals who are undergoing withdrawal without treatment.[7] In general, the longer the individual has been using benzodiazepines, as well as the higher the doses used, the longer the withdrawal will last and the more likely it is that the individual will experience severe withdrawal. Less intense symptoms are likely to persist for several months.

Individuals who are addicted to benzodiazepines must be slowly weaned off the drug or suffer extreme withdrawal symptoms. Individuals undergoing withdrawal should be supervised by experts who can safely switch their medication from short-acting to longer-acting medications, and then slowly reduce these longer-acting medications over periods of months or potentially even years. Counseling and support therapy should also be given.

Harmful benzodiazepine use (mild to moderate)

ICD-9: 304.1 ICD-10 – Harmful use of other psychoactive substances F19.1

This health state refers to an individual who harmfully uses benzodiazepines on a mild to moderate scale; for example, an elderly person who has been prescribed benzodiazepines for 30 days but finishes the pills in 10 days. The individual typically functions well, but may continually negotiate with the treating physician to escalate the dose and prescription. The longer this continues, the more likely it is that the individual become a severe abuser of benzodiazepines[7] and experiences the functional limitations associated with the health state below (Harmful Benzodiazepine Use – severe).

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	2	2	2	1	2	1	1	1	1

Harmful benzodiazepine use (severe)

ICD-9: 304.1 ICD-10 – Harmful use of other psychoactive substances F19.1

Severe abusers of benzodiazepines continue to use the drug after their need for it has passed, and can develop both a physical and a psychological dependence on it. Individuals use benzodiazepines in order to obtain a euphoric effect or to decrease self-awareness. Intoxication resembles drunken-like behaviour, with slurred speech and disorientation. Individuals who are addicted also experience anxiety and aggressive behaviour when they are unable to obtain more benzodiazepines and will often manipulate the health care system so that they can continue to receive their prescriptions. Work or school commitments may be neglected as a result of intoxication; social relationships may be affected due to arguments over use of the substance. Hazardous behaviour (i.e., driving after use) can occur.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	3	4	4	3	2	2	1	1	1

Benzodiazepine overdose

ICD-9: 969.4, E853.2

An individual who overdoses on benzodiazepines will eventually enter into a deep sleep that can progress into either a stupor or coma. This health state refers to an individual who is not in the coma state. During an overdose there is significant cardiovascular and respiratory depression. Initial symptoms of an overdose are impaired thinking, disorientation, slurred speech, muscle weakness, and lack of muscle coordination. Toxic psychoses may also occur, including hallucinations and paranoid delusions. Anxiety is mostly absent during an overdose but increases in the days following. Treatment of an overdose of benzodiazepines includes gastric lavage (stomach pumping), possible intubation (if there is respiratory arrest), and possible aspiration (if the contents of the stomach are vomited into the lungs), as well as a prolonged hospital stay. Overdose on benzodiazepines can be accidental or deliberate, but are rare in the absence of alcohol involvement or the involvement of another drug.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	4	1	4	5	5	1	3	1	1	1

Benzodiazepine treatment (mild to moderate)

ICD-9: 292.0, 94.66

Treatment for an individual who uses benzodiazepines for a short duration is typically done on an out-patient basis. Mild to moderate supervised treatment by the family physician is often sufficient; hospitalization is rarely necessary. Counseling and support are highly recommended.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	1	3	3	2	2	3	1	1	1	1

Benzodiazepine treatment (severe - acute withdrawal with treatment)

ICD-9: 292.0, 94.66

The withdrawal from severe benzodiazepine use must be managed medically because it can be life threatening. An individual experiencing withdrawal is typically switched to a different, longer-lasting medication and then slowly weaned off of it while undergoing counseling and support. While actually withdrawing from benzodiazepines, individuals are anxious, irritable, and they experience insomnia, hallucinations and panic. Long-term hospitalization is often needed for complete withdrawal and recovery.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
4	2	4	3	4	3	4	1	1	1	1

Benzodiazepine remission

ICD-9: 304.13

An individual in remission from harmful use of benzodiazepines must find ways to relax and reduce anxiety on their own, such as meditation or exercise. They also need to avoid situations and people who may lead them back into abusing the drug.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	1	1	1	2	2	1	1	1	1

Part 4 - Harmful cannabis use

Cannabis is the most commonly used illegal drug.[149] It comes in three forms, each derived from the hemp plant, Cannabis sativa. One form is marijuana, the cut and dried leaves and stems of the plant; the second and third forms, hashish and hashish oil (the concentrate of hashish), both come from the dried resin that seeps out from the leaves. Typically, marijuana and hashish are smoked, but they may be taken orally when mixed with tea or food. The psychoactive effects of cannabis (the "high") come from the cannabinoid delta-9-tetrahydrocannabinol (THC). The amount of THC varies but hashish oil usually contains the most, thereby producing the strongest psychoactive effects, followed by hashish and then marijuana.

Cannabis is relatively inexpensive and rarely difficult to obtain. Cannabis users come from all age groups and all education and income levels. Prevalence of cannabis use disorders is highest among individuals between ages 18 and 30 years, and higher in males than females.[7] In a study of Canadian youths, lifetime prevalence rates of marijuana use were 40%.[150] Overall, prevalence of use appears to decline with age.[151]

Frequently cannabis use begins in adolescence or early adulthood. Often individuals begin using because they are curious or because they want to conform to their friends; difficulty in school or boredom may contribute. Risk factors include peer drug use, parental alcoholism or drug use, low parental monitoring, criminal behaviour, and delinquency.[144] Chronic use can lead to mental and physical dependence. In addition, tolerance develops with repeated use; therefore regular, high-dose smokers often need to increase their daily dose to achieve the desired effects (or must abstain from using for several days in order to restore their original sensitivity). Individuals with cannabis dependence may spend hours everyday acquiring and using cannabis for a period of months or years, potentially despite knowledge of physical or psychological problems. Individuals who use cannabis to relieve stress are at higher risk for psychological dependence.[146]

Cannabis intoxication is defined by the DSM-IV as the maladaptive behavioural or psychological changes that occur during or shortly after the use of cannabis, and are accompanied by at least two of the following: conjunctivitis (red eyes), increased appetite, dry mouth, and tachycardia.[7] If the cannabis is smoked, intoxication is achieved within minutes and usually lasts about two to four hours, depending on the dose; if the cannabis is orally ingested, intoxication takes longer to develop but lasts longer than if smoked. Some users feel happy and talkative while intoxicated; others become quiet and withdrawn. Immediate effects of intoxication include sedation, dilated pupils, coughing, mood elevation, euphoria, talkativeness, bronchodilation, altered time perception and slow reaction time. Lethargy, psychomotor and perceptual impairment, paranoia, impaired judgment and motor coordination are other effects. In general, the larger the dose, the longer these effects may last, but it may also depend on the characteristics of the user (i.e., rate of absorption, tolerance). Short-term memory, concentration and abstract thinking generally improve after a few weeks of abstinence, but impairments may be persistent for several years.

Prolonged use of cannabis has been shown to result in low sperm counts in men and fertility problems in women[141] and weaken the immune system. Chronic heavy users appear less motivated and ambitious than others. Cognitive impairments, particularly attention and memory, may persist even after prolonged abstinence. Adjustment problems, reduced communication and social skills, and an inability to focus attention are not uncommon with chronic use. Respiratory diseases may occur with smoking as the drug administration of choice; the lungs can be damaged and lead to persistent coughing, wheezing, asthma, emphysema, increased phlegm, and lung infections. These symptoms and effects are additive to tobacco smoking, putting individuals who smoke cannabis and tobacco at increased risk for lung, neck and head cancers at a younger age; there are even greater amounts of known carcinogens in marijuana smoke than tobacco smoke.[7] Women who use cannabis during pregnancy are more likely to have premature or low birth weight babies.

There is no specific withdrawal syndrome defined in the DSM-IV, but in persons who chronically use large doses of cannabis, abrupt termination can lead to withdrawal symptoms such as anxiety, irritability, insomnia, loss of appetite and weight loss, dysphoria, nausea and sweating. These symptoms generally subside in less than a week, but some disturbances (i.e., sleep) can last for years. Treatment rarely includes admission to a detoxification centre or professional attention. Support, reassurance by family and friends, and lifestyle changes, such as avoiding the people, places and things that relate to cannabis use, are encouraged. In adolescents, without early intervention, developmental milestones may be disturbed or delayed.

Harmful cannabis use

ICD-9: 304.3 ICD-10 – Harmful cannabis use F12.1

Cannabis is the general term used to describe marijuana, hashish, and hashish oil. The health state described in this section refers to an individual who uses cannabis daily. These individuals experience impaired motor performance, impaired judgement, and difficulty with complex mental processes including short-term memory deficits and decreased attention/concentration span, which can interfere with work or school performance. Drowsiness and sedation generally accompany intoxication; overall lethargy and a lack of motivation and interest in life are not uncommon. Depth perception, and impaired motor coordination, including slow reaction times, cause driving and other skilled activities to be dangerous. Fatigue and anxiety are common once the effects of intoxication have worn off. Teenagers, in particular, who abuse cannabis frequently, tend to lose communication with family, experience mood swings and academic underachievement, and deny use despite obvious signs of intoxication or evidence of drug paraphernalia; they are also at increased risk for dropping out of school. Damage to the lungs can also occur due to long-term use of the drug; short-term memory deficits and concentration typically improve over a few weeks of abstinence.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	2	2	2	4	3	3	1	1	2	1

Cannabis treatment (acute withdrawal with treatment)

ICD-9: 292.0, 94.66

An individual undergoing acute withdrawal of cannabis does not generally seek treatment. Most often, friends and family members of addicted individuals work together and form an intervention. They then provide support and reassurance to the individual as the individual withdraws from the drug and changes their lifestyle. Other times, the chronic/addicted individual relies on self monitoring and cutting down over a period of time. Symptoms of withdrawal are not life threatening; in fact, the clinical significance of cannabis withdrawal symptoms are uncertain.[7] These symptoms include insomnia, nausea, irritability, anxiety and loss of appetite. Depression may also occur.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
2	1	2	2	3	2	2	1	1	1	1

Cannabis remission

ICD-9: 304.33

Once an individual is no longer using cannabis, it is important for them to alter their lifestyle so that they are no longer coming into contact with people, places and things that may entice them to begin using again. Long-term problems with sleep often occur in individuals who have had a cannabis addiction.[7] Residual symptoms of depression and memory loss may remain for an undetermined period of time.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	1	2	2	3	2	1	1	1	1	1

Part 5 - Harmful stimulant use

This section describes harmful use of stimulants, which includes both amphetamines and cocaine. Amphetamines are substances that can be obtained through prescriptions for obesity (many amphetamines serve as appetite-suppressants), ADHD, and/or narcolepsy,[7] and are usually taken orally or intravenously; methamphetamine is taken intranasally ("snorted" up the nose). Cocaine is a white powder that is extracted from the coca bush and is usually taken intranasally, but can be smoked or injected. Cocaine is almost only available through the illicit market. Both amphetamines and cocaine are potent central nervous system stimulants and have similar behavioural and psychoactive effects - use typically produces an instant feeling of euphoria and confidence. The psychoactive effects of amphetamines last longer than those of cocaine[7] and amphetamines are therefore taken fewer times per day. Possession, trafficking and prescription "shopping" of stimulants are illegal and may result in a criminal record. Stimulant use is evident in urine tests for 1-3 days following last use, but up to 7-12 days in users with repeated high doses.[7]

Stimulant use affects all races and socioeconomic groups, but is most common between the ages of 18 and 30 years,[7] with males more commonly affected than females. Most users are episodic recreational users. Often individuals begin using to control their weight, enhance their energy, or are introduced to stimulants through illicit markets. The course usually consists of chronic or episodic use (binges alternating with brief drug-free periods; i.e., high weekend use but less during the week). In some cases the binge period ends only when the drug supply is depleted. Tolerance develops with repeated use; therefore, users often increase their daily intake over time. In 1996 in the U.S., approximately 5% of adults reported using stimulants to get "high";[7] about 10% of the population had ever used cocaine.[7] It is estimated that only about 5-10% of individuals who try cocaine eventually use it on a more intensive basis.[146]

The essential feature of stimulant intoxication is significant behavioural or psychological changes that occur during or shortly after use.[7] These changes potentially include euphoria, hypervigilance, anxiety, anger or tension, interpersonal sensitivity, impaired judgment, and/or impaired social or occupational functioning. The severity of these symptoms depends on the dose and characteristics of the individual (i.e., level of tolerance, length of use). Two or more of the following are additionally present: 1) tachycardia or bradycardia; 2) pupillary dilation; 3) elevated or lowered blood pressure; 4) perspiration or chills; 5) nausea or vomiting; 6) evidence of weight loss; 7) psychomotor agitation or retardation; 8) muscular weakness, respiratory depression, chest pain, or cardiac arrhythmias; 9) confusion, seizures, dyskinesias, dystonias, or coma.[7] Severe intoxication by a stimulant can lead to overdose, which may result in death.

Intoxication typically lasts less than an hour, therefore, frequent use is required to maintain a high. Dependence on stimulants can develop after using the drug for only very short periods of time.[7] Individuals with dependence typically exhibit aggressive or violent behaviour, particularly with high doses, in which case the individual may become dangerous. Anxiety is intense; psychotic episodes are possible and resemble episodes seen in paranoid schizophrenia.[141] Most users dependent on stimulants have a disregard for consequences of negative behaviours. Legal difficulties are likely as a consequence of possession or obtaining the drug through illegal markets. A large amount of money can be spent quickly and in short time periods, resulting in potential financial catastrophes. Theft, prostitution and/or drug dealing may be carried out in order to buy or exchange more drugs. Often users need to discontinue use for days in order to obtain additional funds for more drugs. Work or family responsibilities may be neglected because the drugs are considered more important.

Individuals that use stimulants for a long period of time often have impaired personal hygiene and signs of malnutrition. Nosebleeds are common in individuals who use intranasally; sinusitis and/or nasal septum damage may develop. Individuals who smoke stimulants are at increased risk for respiratory problems. Long-term dependence may cause sexual dysfunction, social isolation, and erratic behaviour. Heart attacks, heart palpitations and arrhythmias, stroke and sudden death have been associated with cocaine use among healthy persons.[7]

Stimulant users often use other central nervous system depressants (i.e., alcohol, cannabis) during withdrawal to help reduce their irritability and induce sleep. Stimulant withdrawal ("crashing") develops within a few hours to several days later following the cessation of (or reduction in) prolonged and heavy stimulant use.[7] The individual experiences a dysphoric mood, and at least two of the following physiological changes: fatigue; vivid, unpleasant dreams; insomnia or hypersomnia; increased appetite; and/or psychomotor retardation or agitation. These symptoms cause significant impairment in social or occupational functioning. Often an individual experiencing withdrawal has temporary but intense, depressive symptoms; depression with suicidal ideation/behaviour can occur. The individual typically requires several days of rest to recuperate.

Treatment of harmful stimulant use generally begins with the individual admitting there is a problem. Medication can be prescribed to control the effects of withdrawal; the induction of vomiting (or performing a gastric lavage) may be necessary. Suicide precautions may be necessary, in addition to close supervision and treatment of depression. Inpatient therapy may be required but support and self-help groups (i.e., Narcotics Anonymous) are often sufficient. Cognitive-behavioural therapy can help the individual change their attitudes and behaviours toward stimulant use; recovery programs help to teach coping skills. Reassurance, counseling and supportive care should remain after the completion of treatment.

Harmful stimulant use

ICD-9: 304.4 ICD-10 – Harmful cocaine use F14.1 / – Harmful use of other stimulants F15.1

The central nervous system stimulants include both cocaine and amphetamines. Despite their individual chemical differences, the behavioural effects of these drugs are remarkably similar and they are both highly addictive. Stimulants cause an initial sense of "high" and well being shortly followed by agitation that can cause violent behaviour. Individuals using stimulants experience symptoms of anxiety, irritability, physical discomfort, insomnia and confusion. They also have impaired personal and work relationships, as well as financial and legal problems. Weight loss and malnutrition are typical, resulting from a decreased appetite when intoxicated. Prolonged use of stimulants may also lead to a paranoid psychosis, including hallucinations, delusions, and feelings of panic.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	2	3	3	4	4	3	1	1	1	1

Stimulant overdose

ICD-9: E854.2

An overdose of stimulants causes tremors, convulsions and delirium, possibly ending in a comatose state. Arrhythmias and/or cardiovascular failure may result in death. Individuals who overdose experience extreme anxiety, which could last for days. Treatment of an overdose includes administration of IV fluids, medication for the withdrawal symptoms, close monitoring, and possible intubation; a gastric lavage may also be necessary.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
4	4	5	1	5	5	4	1	1	1	1

Stimulant treatment (acute withdrawal with treatment)

ICD-9: 292.0, 94.66

The functional limitations associated with withdrawal of stimulants include overwhelming fatigue, sleepiness, and depression. Attention and concentration are impaired, and the individual experiences intense hunger, eventually causing weight gain. Individuals can also become paranoid or suffer from physical complaints, such as chills, nausea, or vomiting. Individuals withdrawing from stimulants need to be closely monitored for depression because they are at a high risk for suicide. After an individual has gone through withdrawal, they may initially need in-patient therapy followed by continuous support and reassurance.[7]

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
3	3	4	4	4	3	1	1	1	1	1

Stimulant remission

ICD-9: 304.43

An individual in remission from an addiction to stimulants must perform self-monitoring as the key to abstinence, as well as a strong support network. Meetings with self-help groups are also important as is integrating new coping skills and life management strategies into their lifestyle. Cravings for the drug may continue for years; therefore the individual must avoid the people and/or places that are affiliated with using.[7] Residual physical symptoms may remain.

Classification										
Core attributes						Supplementary attributes				
Pain or discomfort	Physical functioning	Emotional state	Fatigue	Memory & thinking	Social relationships	Anxiety	Speech	Hearing	Vision	Use of hands and feet
1	2	2	1	1	2	1	1	1	1	1

References

1. Government of Canada. The Human Face of Mental Health and Mental Illness in Canada, 2006. Minister of Public Works and Government Services Canada. Catalogue no.: HP5-19/2006E.

2. Murray CJL, Lopez AD, eds. Summary: The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020. Cambridge, MA: Published by the Harvard School of Public Health on behalf of the World Health Organization and the World Bank, Harvard University Press, 1996.

3. WHO (World Health Organization). The global burden of disease: 2004 update. Switzerland: WHO Press; 2008.

4. Lim K-L, Jacobs P, Ohinmaa A, Schopflocher D, Dewa CS. A new population-based measure of the economic burden of mental illness in Canada. Chronic Diseases in Canada 2008; 28(3):92-98.

5. WHO (World Health Organization). International Classification of Diseases, Ninth revision. Basic Tabulation List with Alphabetical Index. Geneva: World Health Organization; 1978.

6. WHO (World Health Organization). International Statistical Classification of Diseases and Related Health Problems, Tenth revision (2nd ed.). Geneva: World Health Organization; 2004.

7. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1995.

8. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. PLoS Medicine 2006; 3(11):e442.

9. Weissman MM, Bland RC, Canino GJ, Faravelli C, Greenwald S, Hwu H-G, Joyce PR, Karam EG, Lee C-K, Lellouch J, Lepine J-P, Newman SC, Rubio-Stipe M, Wells E, Wickramaratne PJ, Wittchen H-U, Yeh E-K. Cross-National epidemiology of major depression and bipolar disorder. JAMA 1996; 276(4):293-299.

10. Beaudet MP. Psychological health – depression. Health Reports 1999; 11(3):63-75.

11. Lepine J-P. Epidemiology, burden, and disability in depression and anxiety. Journal of Clinical Psychiatry 2001; 62(suppl 13):4-10.

12. Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry 2005; 62(6):617-627.

13. Waraich P, Goldner EM, Somers JM, Hsu L. Prevalence and incidence studies of mood disorders: A systematic review of the literature. Canadian Journal of Psychiatry 2004; 49(2):124-138.

14. Kessler RC, Berglund P, Demler O, Jin R, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry 2005; 62(6):593-602.

15. aan het Rot M, Mathew SJ, Charney DS. Neurobiological mechanisms in major depressive disorder. CMAJ 2009; 180(3):305-313.

16. Lieb R, Isensee B, Höfler M, Pfister H, Wittchen H-U. Parental Major Depression and the Risk of Depression and Other Mental Disorders in Offspring. Arch Gen Psychiatry 2002; 59:365-374.

17. Sartorius N. The economic and social burden of depression. J Clin Psychiatry 2001; 62(suppl 15):8-11.

18. Hansson L. Quality of life in depression and anxiety. International Review of Psychiatry 2002; 14:185-189.

19. Judd LL, Akiskal HS, Zeller PJ, Paulus M, Leon AC, Maser JD, Endicott J, Coryell W, Kunovac JL, Mueller TI, Rice JP, Keller MB. Psychosocial disability during the long-term course of unipolar major depressive disorder. Arch Gen Psychiatry 2000; 57:375-380.

20. Waintraub L, Guelfi JD. Nosological validity of dysthymia. Part I: historical, epidemiological and clinical data. Eur Psychiatry 1998; 13:173-180.

21. Brunello N, Akiskal H, Boyer P, Gessa GL, Howland RH, Langer SZ, Mendlewicz J, Paes de Souza M, Placidi GF, Racagni G, Wessely S. Dysthymia: clinical picture, extent of overlap with chronic fatigue syndrome, neuropharmacological considerations, and new therapeutic vistas. Journal of Affective Disorders 1999; 52:275–290.

22. Bagby RM, Ryder AG. Diagnostic discriminability of dysthymia and depressive personality disorder. Depression and Anxiety 1999; 10:41–49.

23. Oxman T, Barrett JE, Sengupta A, Katon W, Williams JW, Frank E, Hegel M. Status of minor depression or dysthymia in primary care following a randomized controlled treatment. General Hospital Psychiatry 2001; 23:301–310.

24. Emilien G, Lucia Septien, Brisard C, Corruble E, Bourin M. Bipolar disorder: How far are we from a rigorous definition and effective management? Progress in Neuro-Psychopharmacology & Biological Psychiatry 2007; 31:975–996.

25. Mental Health America. Bipolar Disorder: What you need to know. Available at: http://cms.nmha.org/index.cfm?objectid=74F71A5E-1372-4D20-C830054B471F27A0. Accessed November 2010.

26. Goodwin FK, Jamison KR. Manic-Depressive Illness. Oxford: Oxford University Press; 1990.

27. Nash J, Potokar J. Anxiety Disorders. Medicine 2004; 32(7):17-21.

28. Miller MC., ed. Panic disorder. Harvard Mental Health Letter 2001; 17(9):1-5.

29. Taylor S, Asmundson GJG, Wald J. Psychopathology of Panic Disorder. Psychiatry 2007; 6(5):188-192.

30. Vanin JR, Vanin SK. Blocking the cycle of panic disorder. Postgraduate Medicine Online May 1, 1999; 105(5).

31. Mental Health America. Panic Disorder. Available at: http://www.mentalhealthamerica.net/go/panic-disorder. Accessed November 2010.

32. Ettigi P, Meyerhoff A, Chirban J, Jacobs RJ, Wilson RR. The quality of life and employment in panic disorder. J Nerv Ment Dis 1997; 185(6):368-372.

33. Hackmann A. Agoraphobia: clinical features and treatment strategies. Psychiatry 2007; 6(6):254-257.

34. Hayward C, Killen D, Taylor CB. The relationship between agoraphobia symptoms and panic disorder in a non-clinical sample of adolescents. Psychological Medicine 2003; 33:733-738.

35. McCabe L, Cairney J, Veldhuizen S, Herrmann N, Streiner DL. Prevalence and correlates of agoraphobia in older adults. American Journal of Geriatric Psychiatry 2006; 14(6):515-522.

36. Bienvenu OJ, Onyike CU, Stein MB, Chen, L-S, Samuels J, Nestadt G, Eaton WW. Agoraphobia in adults: incidence and longitudinal relationship with panic. B J Psychiatry 2006;188:432-438.

37. Mayo Clinic. Agoraphobia. Available at: http://www.mayoclinic.com/print/agoraphobia/DS00894/DSection=all&method=print. Accessed November 2010.

38. Kasper S. Social phobia: The nature of the disorder. Journal of Affective Disorders 1998; 50:S3-S9.

39. National Institute of Mental Health. Anxiety Disorders. National Institutes of Health 2009. Publication No. 09 3879. Available at: http://www.nimh.nih.gov/health/publications/anxiety-disorders/nimhanxiety.pdf. Accessed November 2010.

40. Wittchen H-U, Fehm L. Epidemiology, patterns of comorbidity, and associated disabilities of social phobia. Psychiatric Clinics of North America 2001; 24(4):617-641.

41. Miller MC., ed. Social phobia – Part 1. Harvard Mental Health Letter 1994; 11(4):1-3.

42. Kessler RC. The impairments caused by social phobia in the general population: Implications for intervention. Acta Psychiatr Scand 2003; 108(Suppl 417):19-27.

43. Stein MB, Kean YM. Disability and quality of life in social phobia: Epidemiologic findings. Am J Psychiatry 2000; 157:1606-1613.

44. Wittchen H-U, Fehm L. Epidemiology and natural course of social fears and social phobia. Acta Psychiatr Scand 2003; 108(Suppl 417):4-18.

45. Tyrer P, Baldwin D. Generalized Anxiety Disorder. Lancet 2006; 368:2156-2166.

46. Mayo Clinic. Generalized Anxiety Disorder. Available at: http://www.mayoclinic.com/health/generalized-anxiety-disorder/DS00502. Accessed November 2010.

47. National Institute of Mental Health. Generalized Anxiety Disorder. Available at: http://www.nimh.nih.gov/health/publications/anxiety-disorders/generalized-anxiety-disorder-gad.shtml. Accessed November 2010.

48. Mental Health America. Generalized Anxiety Disorder (GAD). Available at: http://www.mentalhealthamerica.net/go/generalized-anxiety-disorder. Accessed November 2010.

49. Gliatto MF. Generalized anxiety disorder. American Family Physician 2000; 62(7):1591-1600, 1602. Available at: http://www.aafp.org/afp/20001001/1591.html. Accessed November 2010.

50. Leon AC, Portera L, Weissman MM. The social costs of anxiety disorders. B J Psychiatry 1995; 166(Suppl 27):19-22.

51. Cyr NR.Obsessive Compulsive Disorder. AORN Journal 2007; 6(2):277-280.

52. Keeley ML, Storch EA, Dhungana P, Geffken GR. Pediatric obsessive-compulsive disorder: A guide to assessment and treatment. Issues in Mental Health Nursing 2007; 28:555-574.

53. Zohar J, Sasson Y, Chopra M, Amiaz R, Nakash N. Obsessive-compulsive disorder. In: Nutt DJ, Ballenger JC, Editors. Anxiety Disorders. Malden, MA, USA: Blackwell Science Ltd.; 2003. pp. 83-94.

54. Rasmussen SA, Eisen JL. Clinical features and phenomenology of obsessive compulsive disorder. Psychiatric Annals 1989; 19(2):67-73.

55. Bobes J, Gonzalez MP, Bascaran MT, Arango C, Saiz PA, Bousono M. Quality of life and disability in patients with obsessive-compulsive disorder. Eur Psychiatry 2001; 16:239-245.

56. Sasson Y, Zohar J, Chopra M, Lustig M, Iancu J, Hendler T. Epidemiology of obsessive-compulsive disorder: A world view. J Clin Psychiatry 1997; 58(Suppl 12):7-10.

57. Hollander E. Obsessive-compulsive disorder: The hidden epidemic. J Clin Psychiatry 1997; 58(Suppl 12):3-6.

58. Rowa K, Antony MM, Swinson RP. Behavioural treatment of obsessive Compulsive Disorder. Behavioural and Cognitive Psychotherapy 2000; 28:353-360.

59. Nemeroff CB, Bremner JD, Foa EB, Mayberg HS, North CS, Stein MB. Posttraumatic stress disorder: A state-of-the-science review. Journal of Psychiatric Research 2006; 40:1-21.

60. Bryant RA. Early predictors of posttraumatic stress disorder. Biol Psychiatry 2003; 53:789-795.

61. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995; 52(12):1048-1060.

62. Yehuda R. Risk and resilience in posttraumatic stress disorder. J Clin Psychiatry 2004; 65 (Suppl 1):29-36.

63. Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. Psychological Bulletin 2003; 129(1):52-73.

64. Davidson JRT. Long-term treatment and prevention of posttraumatic stress disorder. J Clin Psychiatry 2004; 65 (Suppl 1):44-48.

65. Phelan TW. All About Attention Deficit Disorder. Glen Ellyn, Illinois: Child Management Inc., 1993.

66. Barkley RA. Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York: Guilford Press; 1998.

67. Rinehart NJ, Bradshaw JL, Brereton AV, Tonge BJ. A clinical and neurobehavioural review of high functioning autism and Asperger's disorder. Australian and New Zealand Journal of Psychiatry 2002; 36:762-770.

68. Khouzam HR, El-Gabalawi F, Pirwani N, Priest F. Asperger's disorder: A review of its diagnosis and treatment. Comprehensive Psychiatry 2004; 45(3):184-191.

69. Davids E, Gastpar M. Attention deficit hyperactivity disorder and borderline personality disorder. Progress in Neuro-Psychopharmacology and Biological Psychiatry 2005; 29:865-877.

70. Kessler RC, Adler LA, Barkley R, Biederman J, Conners CK, Faraone SV, et al. Patterns and predictors of attention-deficit/hyperactivity disorder persistence into adulthood: Results from the National Comorbidity Survey replication. Biol Psychiatry 2005; 57(11):1442-1451.

71. Biederman J. Attention-Deficit/Hyperactivity Disorder: A selective overview. Biol Psychiatry 2005; 57:1215-1220.

72. Root II RW, Resnik RJ. An update on the diagnosis and treatment of attention-deficit/hepractivity disorder in children. Professional Psychology: Research and Practice 2003; 34(1):34-41.

73. Pitcher TM, Piek JP, Hay DA. Fine and gross motor ability in males with ADHD. Development Medicine & Child Neurology 2003; 45:525-535.

74. Blackman GL, Ostrander R, Herman KC. Children with ADHD and depression: A multisource, multimethod assessment of clinical, social, and academic functioning. Journal of Attention Disorders 2005; 8(4):195-207.

75. Tidmarsh L, Volkmar FR. Diagnosis and epidemiology of autism spectrum disorders. Can J Psychiatry 2003; 48(8):517-525.

76. Bryson SE, Rogers SJ, Fombonne E. Autism spectrum disorders: Early detection, intervention, education, and psychopharmacological management. Can J Psychiatry 2003; 48(8):506-516.

77. Bloch-Rosen S for the University of Delaware. Research Paper: Asperger's Syndrome, High Functioning Autism, and Disorders of the Autistic Continuum. 1999. Available at: http://www.aspergersyndrome.com/Paper040899.pdf. Accessed November 2010.

78. National Institute of Mental Health. Autism Spectrum Disorders (Pervasive Developmental Disorders). National Institutes of Health 2009. Available at: http://www.nimh.nih.gov/health/publications/autism/nimhautismspectrum.pdf. Accessed November 2010.

79. Fombonne E. Modern views of autism. Can J Psychiatry 2003; 48(8):503-505.

80. Wing L, Potter D. The epidemiology of autistic spectrum disorders: Is the prevalence rising? Mental Retardation and Developmental Disabilities Research Reviews 2002; 8:151-161.

81. Bailey AJ. The biology of autism. Psychological Medicine 1993; 23:7-11.

82. Nicolson R, Szatmari P. Genetic and neurodevelopmental influences in autistic disorder. Can J Psychiatry 2003; 48(8):526-537.

83. Hughes JR. A review of recent reports on autism: 1000 studies published in 2007. Epilepsy & Behavior 2008; 13:425-437.

84. Maimburg RD, Væth M. Perinatal risk factors and infantile autism. Acta Psychiatr Scand 2006; 114:257-264.

85. Happé F, Ronald A. The 'Fractionable Autism Triad': A Review of Evidence from Behavioural, Genetic, Cognitive and Neural Research. Neuropsychol Rev 2008; 18:287-304.

86. Parikh MH, Kolevson A, Hollander E. Psychopharmacology of Aggression in Children and Adolescents with Autism: A Critical Review of Efficacy and Tolerability. Journal of Child and Adolescent Psychopharmacology 2008; 18:157-178.

87. Ehlers S, Gillberg C. The epidemiology of Asperger Syndrome. A total population study. Journal of Child Psychology and Psychiatry and Allied Disciplines 1993; 34(8):1327-1350.

88. Mandell DS, Novak MM, Zubritsky CD. Factors associated with age of diagnosis among children with autism spectrum disorders. Pediatrics 2005; 116(6):1480-1486.

89. Schnur J. Asperger syndrome in children. Journal of the American Academy of Nurse Practitioners 2005; 17(8):302-308.

90. Rao PA, Beidel DC, Murray MJ. Social Skills Interventions for Children with Asperger's Syndrome or High-Functioning Autism: A Review and Recommendations. J Autism Dev Disord 2008; 38:353-361.

91. Solomon M, Goodlin-Jones BL, Anders TF. A social adjustment enhancement intervention for high functioning autism, Asperger's syndrome, and pervasive developmental disorder NOS. Journal of Autism and Developmental Disorders 2004; 34(6):649-668.

92. Beamont R, Sofronoff K. A multi-component social skills intervention for children with Asperger syndrome: The junior detective training program. The Journal of Child Psychology and Psychiatry 2008; 49(7):743-753.

93. Owens G, Granader Y, Humphrey A, Baron-Cohen S. Lego® therapy and the social use of language programme: An evaluation of two social skills interventions for children with high functioning autism and Asperger syndrome. J Autism Dev Disord 2008; 38:1944-1957.

94. Toth K, King BH. Asperger's Syndrome: Diagnosis and Treatment. Am J Psychiatry 2008; 168(8):958-963.

95. Masi G, Mucci M, Millepiedi S. Separation anxiety disorder in children and adolescents: Epidemiology, diagnosis and management. CNS Drugs 2001; 15(2):93-104.

96. Hanna GL, Fischer DJ, Fluent TE. Separation anxiety disorder and school refusal in children and adolescents. Pediatrics in Review 2006; 27(2):56-63.

97. Miller MC., ed. Separation anxiety. Harvard Mental Health Letter 2007; 23(7):1-3.

98. Shear K, Jin R, Meron Ruscio A, Walters EE, Kessler RC. Prevalence and correlates of estimated DSM-IV child and adult separation anxiety disorder in the National Comorbidiy Survey replication. Am J Psychiatry 2006; 163(3):1074-1083.

99. Foley DL, Pickles A, Maes HM, Silberg JL, Eaves LJ. Course and short-term outcomes of separation anxiety disorder in a community sample of twins. J Am Acad Child Adolesc Psychiatry 2004; 43(9):1107-1114.

100. Kearney CA, Sims KE, Pursell CR, Tillotson CA. Separation anxiety disorder in young children: A longitudinal and family analysis. Journal of Child and Adolescent Psychology 2003; 32(4):593-598.

101. Hoek H, van Hoeken D. Review of prevalence and incidence of eating disorders. Int J Eat Disord 2003;160:248.

102. Palmer B. Epidemiology, diagnosing and assessing eating disorders. Psychiatry 2008; 7(4):143-146.

103. Ackard DM, Fulkerson JA, Neumark-Sztainer D., Prevalence and Utility of DSM-IV Eating Disorder Diagnostic Criteria among Youth. Int J Eat Disord 2007; 40:409-417.

104. Bulik CM, Reba L, Siega-Riz A-M, Reichborn-Kjennerud T. Anorexia nervosa: Definition, epidemiology, and cycle of risk. International Journal of Eating Disorders 2005; 37:52-59.

105. Chavez M., Insel T.R. Eating Disorders. National Institute of Mental Health's Perspective. American Psychologist 2007; 62(3):159-166.

106. Hsu GLK. Epidemiology of the eating disorders. Psychiatric Clinics of North America 1996; 19(4):681-697.

107. Franko DL, Keel PK, Dorer DJ, Blais MA, Delinsky SS, Eddy KT, Charat V, Renn R, Herzog DB. What predicts suicide attempts in women with eating disorders? Psychological Medicine 2004; 34:843-853.

108. Bulik CM, Sullivan PF, Fear J, Pickering A. Predictors of the development of bulimia nervosa in women with anorexia nervosa. The Journal of Nervous and Mental Disease 1997; 185(11):704-707.

109. Wonderlich SA, Lilenfeld LR, Riso LP, Engel S, Mitchell JE. Personality and anorexia nervosa. International Journal of Eating Disorders 2005; 37:S68-S71.

110. Slade R. The Anorexia Nervosa Reference Book. London: Harper & Row; 1984.

111. Fichter MM, Quadflieg N, Hedlund S. Twelve-year course and outcome predictors of anorexia nervosa. International Journal of Eating Disorders 2006; 39(2):87-100.

112. Bates T, Zlopasa O, Gasparović V. Anorexia nervosa: stanje zivotne ugrozenosti. Lijecnicki vjesnik 2007; 129(1-2):11-6.

113. Carter JC, Blackmore E, Sutandar-Pinnock K, Woodside DB. Relapse in anorexia nervosa: A survival analysis. Psychological Medicine 2004; 34:671-679.

114. Herzog DB, Dorer DJ, Keel PK, Selwyn SE, Ekeblad ER, Flores AT, Greenwood DN, Burwell RA, Keller MB. Recovery and relapse in anorexia nervosa and bulimia nervosa: a 7.5-year follow-up study. Journal of the American Academy of Child and Adolescent Psychiatry 1999; 38:829–837.

115. Bulik CM, Sullivan PF, Fear J, Pickering A. Outcome of anorexia nervosa: Eating attitudes, personality, and parental bonding. International Journal of Eating Disorders 2000; 28(2):139-147.

116. Hay PJ, Bacaltchuk J. Extracts from "Clinical Evidence": Bulimia nervosa. BMJ 2001; 323:33-37.

117. National Alliance on Mental Illness. Bulimia nervosa. Available at: http://www.nami.org/Template.cfm?Section=By_Illness&template=/ContentManagement/ContentDisplay.cfm&ContentID=65839. Accessed November 2010.

118. O'Brien KM, Vincent NK. Psychioatric comorbidity in anorexia and bulimia nervosa: nature, prevalence, and causal relationships. Clinical Psychology Review 2003; 23:57-74.

119. Pearlstein T. Eating disorders and comorbidity. Arch Womens Ment Health 2002; 4:67-68.

120. Lo Russo L, Campisi G, Di Fede O, Di lIberto C, Panzarella V, Lo Muzio L. Oral manifestations of eating disorders: a critical review. Oral Diseases 2008; 14:479-484.

121. Merck. Mental Retardation/Intellectual Disability. The Merck Manual Home Edition; Section 23: Children's Health Issues; Chapter 285: Mental Retardation/Intellectual Disability [Online]. Available at: http://www.merck.com/mmhe/sec23/ch285/ch285a.html. Accessed November 2010.

122. Bhasin TK, Brocksen S, Avchen RN, Braun KVN. Prevalence of four developmental disabilities among children aged 8 years – Metropolitan Atlanta Developmental Disabilities Surveillance Program, 1996 and 2000. In: Surveillance Summaries, January 27, 2006. MMWR 2006; 55(SS-1):1-9.

123. Batshaw ML, Perret YM. Children With Disabilities. U.S.A.: Paul H. Brookes Publishing Company; 1992.

124. Stephens DL, Collins MD, Dodder RA. A longitudinal study of employment and skill acquisition among individuals with developmental disabilities. Research in Developmental Disabilities 2005; 26:469-486.

125. Bradley R, Conklin CZ, Westen D. The borderline personality diagnosis in adolescents: Gender differences and subtypes. Journal of Child Psychology and Psychiatry 2005; 46(9):1006-1019.

126. Ekselius L, Tillfors M, Furmark T, Fredrikson M. Personality disorders in the general population: DSM-IV and ICD-10 defined prevalence as related to sociodemographic profile. Personality and Individual Differences 2001; 30:311-320.

127. Davidson KM. Cognitive-behavioural therapy for personality disorders. Psychiatry 2008; 7(3):117-120.

128. Reichborn-Kjennerud T, Czajkowski N, Neale MC, Ørstavik RE, Torgersen S, Tambs K, Røysamb E, Harris JR, Kendler KS. Genetic and environmental influences on dimensional representations of DSM-IV cluster C personality disorders: a population-based multivariate twin study. Psychological Medicine 2007; 37:645-653.

129. Skodol AE, Siever LJ, Livesley WJ, Gunderson JG, Pfohl B, Widiger TA. The borderline diagnosis II: Biology, Genetics, and Clinical Course. Biol Psychiatry 2002; 51:951-963.

130. Huber G. The heterogeneous course of schizophrenia. Schizophrenia Research 1997; 28:177-185.

131. Gerbaldo H, Georgi K, Pieschl D. The deficit syndrome in first-admission patients with psychotic and non-psychotic disorders. Eur Psychiatry 1997; 12:53-57.

132. Hafner H, an der Heiden W. Epidemiology of schizophrenia. Canadian Journal of Psychiatry 1997; 42:139-151.

133. Jablensky A. Epidemiology of schizophrenia: The global burden of disease and disability. Eur Arch Psychiatry Clin Neurosci 2000; 250:274-285.

134. Maki P, Veijola J, Jones PB, Murray GK, Koponen H, Tienari P, Miettunen J, Tanskanen P, Wahlberg K-E, Koskinen J, Lauronen E, Isohanni M. Predictors of schizophrenia—a review. British Medical Bulletin 2005; 73-74:1-15.

135. Buskist W, Gerbing DW. Psychology: Boundaries and Frontiers. Glenview, Illinois: Brown Higher Education; 1990.

136. Siris SG. Suicide and schizophrenia. Journal of Psychopharmacology 2001; 15(2):127-135.

137. Marwaha S, Johnson S. Schizophrenia and employment: A review. Social Psychiatry and Psychiatric Epidemiology 2004; 39:337-349.

138. Marwaha S, Johnson S, Bebbington P, Stafford M, Angermeyer MC, Brugha T, Azorin J-M, Kilian R, Hansen K, Toumi M. Rates and correlated of employment in people with schizophrenia in the UK, France, and Germany. British Journal of Psychiatry 2007; 191:30-37.

139. Rehm J, Taylor B, Room R. Global burden of disease from alcohol, illicit drugs and tobacco. Drug and Alcohol Review 2006; 25:503-513.

140. Merck. Alcohol. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/print/sec15/ch198/ch198g.html. Accessed November 2010.

141. Doweiko HE. Concepts of Chemical Dependency. U.S.A.: Brooks/Cole Publishing Company; 1993.

142. Grant BF, Stinson FS, Dawson DA, Choi P, Dufour MC, Compton W, Pickering RP, Kaplan K. Prevalence and co-occurrence of substance use disorders and independent mood and anxiety disorders: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. Archives of General Psychiatry 2004; 61(8):807-816.

143. Hingson RW, Heeren T, Winter MR. Age at drinking onset and alcohol dependence. Arch Pediatr Adolesc Med 2006; 160:739-746.

144. Hales RE, Yudofsky SC, Talbot JA. (Eds.) Textbook of Psychiatry. Second edition. Washington, DC: The American Psychiatric Press; 1994.

145. Becker WC, Fiellin DA, Merrill JO, Schulman B, Finkelstein R, Olsen Y, Busch SH. Opiod use disorders in the United States: Insurance status and treatment access. Drug and Alcohol Dependence 2008; 94:207-213.

146. Brands B, Spraule B, Marshman J. (Eds.) Drugs and Drug Abuse. Third edition. Toronto: Addiction Research Foundation; 1998.

147. National Institute on Drug Abuse. Research Report Series: Heroin Abuse and Addiction. NIH Publication No.: 05-4165. 2005. Available at: http://www.nida.nih.gov/PDF/RRHeroin.pdf. Accessed November 2010.

148. Merck. Opioids. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/sec15/ch198/ch198f.html. Accessed November 2010.

149. Merck. Marijuana (Cannabis). The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/print/sec15/ch198/ch198i.html. Accessed November 2010.

150. Barnes GE, Barnes MD, Patton D. Prevalence and predictors of "heavy" marijuana use in a Canadian youth sample. Substance Use and Misuse 2005; 40:1849-1863.

151. Kalont H, Corrigal W. The Health Effects of Cannabis. Canada: Center for Addiction and Mental Health; 1999.

The following references were also consulted to develop the health state descriptions and text in this document:

Abbate-Daga G, Piero A, Gramaglia C, Fassino S. Factors related to severity of vomiting behaviors in bulimia nervosa. Psychiatry Research 2005; 134:75-84.

American Academy of Child and Adolescent Psychiatry. Asperger's disorder. Available at: http://www.aacap.org/publications/factsfam/69.htm. Accessed November 2010.

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington, DC: American Psychiatric Association; 1995.

Angermeyer MC, Holzinger A, Matschinger H, Stengler-Wenzke K. Depression and quality of life: Results of a follow-up study. Int J Soc Psychiatry 2002; 48(3):189-199.

Angst J, Gamma A, Endrass J, Goodwin R, Ajdacic V, Elch D, et al. Obsessive-compulsive severity spectrum in the community: prevalence, comorbidity, and course. Eur Arch Psychiatry Clin Neurosci 2004; 254:156-164.

Ballanger JC. Overview of different pharmacotherapies for attaining remission in generalized anxiety disorder. J Clin Psychiatry 2001; 62[Suppl 19]:11-19.

Barkley RA. Attention deficit hyperactivity disorder: A handbook for diagnosis and treatment. New York: Guilford Press; 1998.

Barkley RA. Taking Charge of ADHD: The Complete Authoritative Guide for Parents. U.S.A.: The Guilford Press; 1995.

Barnes GE, Barnes MD, Patton D. Prevalence and predictors of "heavy" marijuana use in a Canadian youth sample. Substance Use and Misuse 2005; 40:1849-1863.

Baron, Earhard & Ozier. Psychology. Pearson Education Canada Inc., 2001.

Beamish PM, Granello DH, Belcastro AL. Treatment of panic disorder: Practical guidelines. Journal of Mental Health Counseling 2002; 24(3):224-246.

Beaudet MP. Depression. Health Reports 1996; 7(4):11-24.

Beaudet MP. Psychological health – depression. Health Reports 1999; 11(3):63-75.

Bizzarri J, Rucci P, Vallotta A, Girelli M, Scandolari A, Zerbetto E, et al. Dual diagnosis and quality of life in patients in treatment for opioid dependence. Substance Use and Misuse 2005; 40:1765-1776.

Blackman GL, Ostrander R, Herman KC. Children with ADHD and depression: A multisource, multimethod assessment of clinical, social, and academic functioning. Journal of Attention Disorders 2005; 8(4):195-207.

Bloch-Rosen S for the University of Delaware. Research Paper: Asperger's Syndrome, High Functioning Autism, and Disorders of the Autistic Continuum. 1999. Available at: http://www.aspergersyndrome.com/Paper040899.pdf. Accessed November 2010.

Bobes J, Gonzalez MP, Bascaran MT, Arango C, Saiz PA, Bousono M. Quality of life and disability in patients with obsessive-compulsive disorder. Eur Psychiatry 2001; 16:239-245.

Bradley R, Conklin CZ, Westen D. The borderline personality diagnosis in adolescents: Gender differences and subtypes. Journal of Child Psychology and Psychiatry 2005; 46(9):1006-1019.

Brands B, Spraule B, Marshman J. Drugs and Drug Abuse. Canada: Addiction Research Foundation; 1998.

Bryson SE, Rogers SJ, Fombonne E. Autism spectrum disorders: Early detection, intervention, education, and psychopharmacological management. Can J Psychiatry 2003; 48(8):506-516.

Buonopane A, Petrakis IL. Pharmacotherapy of alcohol use disorders. Substance Use and Misuse 2005; 40:2001-2020.

Buskist W, Gerbing DW. Psychology: Boundaries and Frontiers. Glenview, Illinois: Brown Higher Education; 1990.

Candilis PJ, McLean RYS, Otto MW, Manfro GG, Worthington JJ, Penava SJ, et al. Quality of life in patients with panic disorder. J Nerv Ment Dis 1999; 187(7):429-434.

Clarkin JF, Yeomans FE, Kernberg O. Psychotherapy for borderline personality disorder. New York: Wiley, 1999.

Cohen AS, Dinzeo TJ, Nienow TM, Smith DA, Singer B, Docherty NM. Diminished emotionality and social functioning in schizophrenia. The Journal of Nervous and Mental Disease 2005; 193(12):796-802.

Cramer V, Torgersen S, Kringlen E. Quality of life and anxiety disorders: A population study. The Journal of Nervous and Mental Disease 2005; 193(3):196-202.

Dahl AA, Ravindran A, Allgulander C, Kutcher SP, Austin C, Burt T. Setraline in generalized anxiety disorder: Efficacy in treating the psychic and somatic anxiety factors. Acta Psychiatr Scand 2005; 111:429-435.

D'Ardenne P, Capuzzo N, Fakhoury WKH, Jankovic-Gavrilovic J, Priebe S. Subjective quality of life and posttraumatic stress disorder. The Journal of Nervous and Mental Disease 2005; 193(1):62-65.

Davids E, Gastpar M. Attention deficit hyperactivity disorder and borderline personality disorder. Progress in Neuro-Psychopharmacology and Biological Psychiatry 2005; 29:865-877.

Davidson JRT. Long-term treatment and prevention of posttraumatic stress disorder. J Clin Psychiatry 2004; 65(Suppl 1):44-48.

De Bildt A, Sytema S, Kraijer D, Sparrow S, Minderaa R. Adaptive functioning and behaviour problems in relation to level of education in children and adolescents with intellectual disability. Journal of Intellectual Disability Research 2005; 49(9):672-681.

Doll HA, Petersen SE, Stewart-Brown SL. Eating disorders and emotional and physical well-being: Associations between student self-reports of eating disorders and quality of life as measured by the SF-36. Quality of Life Research 2005; 14:705-717.

Doweiko HE. Concepts of Chemical Dependency. U.S.A.: Brooks/Cole Publishing Company; 1993.

Dykens EM. Happiness, well-being, and character strengths: Outcomes for families and siblings of persons with mental retardation. Mental Retardation 2005; 43(5):360-364.

Ehlers S, Gillberg C. The epidemiology of Asperger Syndrome. A total population study. Journal of Child Psychology and Psychiatry and Allied Disciplines 1993; 34(8):1327-1350.

Emerson E, Robertson J, Wood J. Emotional and behavioural needs of children and adolescents with intellectual disabilities in an urban conurbation. Journal of Intellectual Disability Research 2005; 49(1):16-24.

Ettigi P, Meyerhoff A, Chirban J, Jacobs RJ, Wilson RR. The quality of life and employment in panic disorder. J Nerv Ment Dis 1997; 185(6):368-372.

Fairburn CG, Agras WS, Walsh BT, Wilson GT, Stice E. Prediction of outcome in bulimia nervosa by early change in treatment. American Journal of Psychiatry 2004; 161:2322-2324.

Ferriman A. The stigma of schizophrenia. BMJ 2000; 320(7233):522.

Fichter MM, Quadflieg N. Twelve-year course and outcome of bulimia nervosa. Psychological Medicine 2004; 33:1395-1406.

Fichter MM, Quadflieg N, Hedlund S. Twelve-year course and outcome predictors of anorexia nervosa. International Journal of Eating Disorders 2006; 39(2):87-100.

Foley DL, Pickles A, Maes HM, Silberg JL, Eaves LJ. Course and short-term outcomes of separation anxiety disorder in a community sample of twins. J Am Acad Child Adolesc Psychiatry 2004; 43(9):1107-1114.

Frith U. Autism: Explaining the Enigma. Oxford: Blackwell; 1989.

Fombonne E. Modern views of autism. Can J Psychiatry 2003; 48(8):503-505.

Franko DL, Keel PK, Dorer DJ, Blais MA, Delinsky SS, Eddy KT et al. What predicts suicide attempts in women with eating disorders? Psychological Medicine 2004; 34:843-853.

Ghaziuddin M, Mountain-Kimchi K. Defining the intellectual profile of Asperger syndrome: Comparison with high-functioning autism. Journal of Autism and Developmental Disorders 2004; 34(3):279-284.

Gliatto MF. Generalized anxiety disorder. American Family Physician 2000; 62(7):1591-1600, 1602. Available at: http://www.aafp.org/afp/20001001/1591.html. Accessed November 2010.

Goldney RD, Fisher LJ, Wilson DH, Cheok F. Major depression and its associated morbidity and quality of life in a random, representative Australian community sample. Australian and New Zealand Journal of Psychiatry 2000; 34:1022-1029.

Goodwin FK, Jamison KR. Manic-Depressive Illness. Oxford: Oxford University Press; 1990.

Gorissen M, Sanz JC, Schmand B. Effort and cognition in schizophrenia patients. Schizophrenia Research 2005; 78:199-208.

Gottesfield H. Abnormal Psychology. U.S.A.: Science Research Associates Inc.; 1979.

Gourlay J, Ricciardelli L, Ridge D. Users' experiences of heroin and methadone treatment. Substance Use and Misuse 2005; 40:1875-1882.

Government of Canada. The Human Face of Mental Health and Mental Illness in Canada, 2006. Minister of Public Works and Government Services Canada. Catalogue no.: HP5-19/2006E.

Grant BF, Hasin DS, Stinson FS, Dawson DA, Chou SP, Ruan WJ et al. Prevalence, correlates, and disability of personality disorders in the United States: Results from the National Epidemiologic Survey on Alcohol and Related Conditions. Journal of Clinical Psychiatry 2004; 65: 948-958.

Gualtieri CT. Brain Injury and Mental Retardation: Psychopharmacology and Neuropsychiatry. PA, U.S.A.: Lippincott Williams & Wilkins; 2002.

Gupta S, Kulhara P, Verma SK. Quality of life in schizophrenia and dysthymia. Acta Psychiatrica Scandinavica 1998; 97:290-296.

Hales RE, Yudofsky SC, Talbot JA. Textbook of Psychiatry. Washington, DC: The American Psychiatric Press; 1994.

Hanna GL, Fischer DJ, Fluent TE. Separation anxiety disorder and school refusal in children and adolescents. Pediatrics in Review 2006; 27(2):56-63.

Hansson L. Quality of life in depression and anxiety. International Review of Psychiatry 2002; 14:185-189.

Hay PJ, Bacaltchuk J. Extracts from "Clinical Evidence": Bulimia nervosa. BMJ 2001; 323:33-37.

Hayden MF, Kim SH, DePaepe P. Health status, utilization patterns, and outcomes of persons with intellectual disabilities: Review of the literature. Mental Retardation 2005; 43(3):175-195.

Health Canada. Economic Burden of Illness in Canada, 1998. Ottawa: Health Canada, 2002. Available at: http://www.phac-aspc.gc.ca/publicat/ebic-femc98/pdf/ebic1998.pdf. Accessed November 2010.

Hollander E. Obsessive-compulsive disorder: The hidden epidemic. J Clin Psychiatry 1997; 58(Suppl 12):3-6.

Hollifield M, Katon W, Skipper B, Chapman T, Ballenger JC, Mannuzza S, Fyer AJ. Panic disorder and quality of life: Variables predictive of functional impairment. The American Journal of Psychiatry 1997; 154(6):766-772.

Howgego IM, Owen C, Meldrum L, Yellowlees P, Dark F, Parslow R. Posttraumatic stress disorder: An exploratory study examining rates of trauma and PTSD and its effect on client outcomes in community mental health. BMC Psychiatry 2005; 5:21-37.

Johansen VA, Wahl AK, Eilertsen DE, Weisaeth L, Hanestad BR. The predictive value of post-traumatic stress disorder

symptoms for quality of life: A longitudinal study of physically injured victims of non-domestic violence. Health and Quality of Life Outcomes 2007; 5:26-36.

Kalont H, Corrigal W. The Health Effects of Cannabis. Canada: Center for Addiction and Mental Health; 1999.

Kasper S. Social phobia: The nature of the disorder. Journal of Affective Disorders 1998; 50:S3-S9.

Kearney CA, Sims KE, Pursell CR, Tillotson CA. Separation anxiety disorder in young children: A longitudinal and family analysis. Journal of Child and Adolescent Psychology 2003; 32(4):593-598.

Keeley ML, Storch EA, Dhungana P, Geffken GR. Pediatric obsessive-compulsive disorder: A guide to assessment and treatment. Issues in Mental Health Nursing 2007; 28:555-574.

Kessler RC. The impairments caused by social phobia in the general population: Implications for intervention. Acta Psychiatr Scand 2003; 108(Suppl 417):19-27.

Kessler RC, Adler LA, Barkley R, Biederman J, Conners CK, Faraone SV, et al. Patterns and predictors of attention-deficit/hyperactivity disorder persistence into adulthood: Results from the National Comorbidity Survey replication. Biol Psychiatry 2005; 57:1442-1451.

Kessler RC, Berglund P, Demler O, Jin R, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry 2005; 62:593-602.

Kessler RC, Chiu WT, Demler O, Walters EE. Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. Archives of General Psychiatry 2005; 62:617-627.

Kessler RC et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. Archives of General Psychiatry 1994; 51:8-19.

Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. Arch Gen Psychiatry 1995; 52(12):1048-1060.

Kirisci L, Vanyukov M, Tarter R. Detection of youth at high risk for substance use disorders: A longitudinal study. Psychology of Addictive Behaviors 2005; 19(3):243-252.

Koran LM, Thienemann ML, Davenport R. Quality of life for patients with obsessive-compulsive disorder. The American Journal of Psychiatry 1996; 153(6):783-788.

Lader M. The clinical relevance of treating social phobia. Journal of Affective Disorders 1998; 50: S29-S34.

LeBlanc N, Morin D. Depressive symptoms and associated factors in children with attention deficit hyperactivity disorder. Journal of Child and Adolescent Psychiatric Nursing 2004; 17(2):49-55.

Lecrubier Y. Posttraumatic stress disorder in primary care: A hidden diagnosis. J Clin Psychiatry 2004; 65 (Suppl 1):49-54.

Lepine J-P. Epidemiology, burden, and disability in depression and anxiety. Journal of Clinical Psychiatry 2001; 62(suppl 13):4-10.

Le Roux H, Gatz M, Wetherell JL. Age at onset of generalized anxiety disorder in older adults. Am J Geriatr Psychiatry 2005; 13(1):23-30.

Lim K-L, Jacobs P, Ohinmaa A, Schopflocher D, Dewa CS. A new population-based measure of the economic burden of mental illness in Canada. Chronic Diseases in Canada 2008; 28(3):92-98.

Lipton MI. Posttraumatic Stress Disorder – Additional Perspectives. Springfield, IL: C.C. Thomas; 1994.

Livesley J. Integrated therapy for complex cases of personality disorder. Journal of Clinical Psychology: In Session 2008; 64(2):207-221.

Lochner C, Hemmings SMJ, Kinnear CJ, Moolman-Smook JC, Corfield VA, Knowles JA, et al. Gender in obsessive-compulsive disorder: Clinical and Genetic Findings. European Neuropsychopharmacology 2004; 14:105-113.

Mandell DS, Walrath CM, Manteuffel B, Sgro G, Pinto-Martin J. Characteristics of children with autistic spectrum disorders served in comprehensive community-based mental health setting. Journal of Autism and Developmental Disorders 2005; 35(3):313-321.

Mandell DS, Novak MM, Zubritsky CD. Factors associated with age of diagnosis among children with autism spectrum disorders. Pediatrics 2005; 116(6):1480-1486.

Marwaha S, Johnson S. Schizophrenia and employment: A review. Social Psychiatry and Psychiatric Epidemiology 2004; 39:337-349.

Marwaha S, Johnson S, Bebbington P, Stafford M, Angermeyer MC, Brugha T, Azorin J-M, Kilian R, Hansen K, Toumi M. Rates and correlated of employment in people with schizophrenia in the UK, France, and Germany. British Journal of Psychiatry 2007; 191:30-37.

Masellis M, Rector NA, Richter MA. Quality of life in OCD: Differential impact of obsessions, compulsions, and depression comorbidity. Can J Psychiatry 2003; 48(2):72-77.

Masi G, Mucci M, Millepiedi S. Separation anxiety disorder in children and adolescents: Epidemiology, diagnosis and management. CNS Drugs 2001; 15(2):93-104.

McCance-Katz EF, Carroll KM, Rounsaville BI. Gender differences in treatment seeking cocaine abusers – implications for prognosis and treatment. American Journal on Addictions 1999; 8:300-311.

McCance-Katz EF, Hart CL, Boyarsky B, Kosten T, Jatlow P. Gender effects following repeated administration of cocaine and alcohol in humans. Substance Use and Misuse 2005; 40:511-528.

Merck. Alcohol. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/print/sec15/ch198/ch198g.html. Accessed November 2010.

Merck. Amphetamines. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 18: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/sec15/ch198/ch198k.html. Accessed November 2010.

Merck. Cocaine. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/sec15/ch198/ch198j.html. Accessed November 2010.

Merck. Marijuana (Cannabis). The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/print/sec15/ch198/ch198i.html. Accessed November 2010.

Merck. Mental Retardation/Intellectual Disability. The Merck Manual Home Edition; Section 23: Children's Health Issues; Chapter 285: Mental Retardation/Intellectual Disability [Online]. Available at: http://www.merck.com/mmhe/sec23/ch285/ch285a.html. Accessed November 2010.

Merck. Opioids. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 198: Drug Use and Dependence [Online]. Available at: http://www.merckmanuals.com/professional/sec15/ch198/ch198f.html. Accessed November 2010.

Merck. Personality disorders. The Merck Manual of Diagnosis and Therapy; Section 15: Psychiatric Disorders; Chapter 201: Personality Disorders. Available at: http://www.merckmanuals.com/professional/sec15/ch201/ch201a.html. Accessed November 2010.

Miller MC., ed. Panic disorder. Harvard Mental Health Letter 2001; 17(9):1-5.

Miller MC., ed. Social phobia – Part 1. Harvard Mental Health Letter 1994; 11(4):1-3.

Mogotsi M, Kaminer D, Stein DJ. Quality of life in the anxiety disorders. Harvard Rev Psychiatry 2000; 8:273-282.

Monnier J, Brawman-Mintzer O. Generalized anxiety disorder. In: Nutt DJ, Ballenger JC, Editors. Anxiety Disorders. Malden, MA, USA: Blackwell Science Ltd.; 2003. pp. 51-64.

Moritz S, Rufer M, Fricke S, Karow A, Morfeld M, Jelinek L, et al. Quality of life in obsessive-compulsive disorder before and after treatment. Comprehensive Psychiatry 2005; 46:453-459.

Murray CJL, Lopez AD, eds. Summary: The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020. Cambridge, MA: Published by the Harvard School of Public Health on behalf of the World Health Organization and the World Bank, Harvard University Press, 1996.

National Institute of Mental Health. Anxiety Disorders. Rockville, Maryland. NIMH. NIH Publication No. 98-4268. www.nimh.nih.gov/publicat/anxiety.cfm. Accessed December 2010.

Nicolson R, Szatmari P. Genetic and neurodevelopmental influences in autistic disorder. Can J Psychiatry 2003; 48(8):526-537.

Ninan PT. Dissolving the burden of generalized anxiety disorder. J Clin Psychiatry 2001; 62[Suppl 19]:5-10.

Ninan PT. Generalized anxiety disorder: Why are we failing our patients? J Clin Psychiatry 2001; 62[Suppl 19]:3-4.

Olmsted MP, Kaplan AS, Rockert W. Defining remission and relapse in bulimia nervosa. International Journal of Eating Disorders 2005; 38:1-6.

Owen RR, Rost K, Hollenberg J, Humphrey JB, Lazoritz M, Bartlett J, et al. Effectiveness of care and improvement in quality of life in patients with panic disorder. Evaluation Review 1997; 21(3):405-416.

Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. Psychological Bulletin 2003; 129(1):52-73.

Phelan TW. All About Attention Deficit Disorder. Glen Ellyn, Illinois: Child Management Inc., 1993.

Poulton R, Milne BJ, Craske MG, Menzies RG. A longitudinal study of the etiology of separation anxiety. Behavior Research and Therapy 2001; 39:1395-1410.

Powers MD (Ed.). Children with Autism: A Parent's Guide. MD, U.S.A.: Woodbine House; 1989.

Rasmussen SA, Eisen JL. Clinical features and phenomenology of obsessive compulsive disorder. Psychiatric Annals 1989; 19(2):67-73.

Rauch SAM, Foa EB. Post-traumatic stress disorder. In: Nutt DJ, Ballenger JC, Editors. Anxiety Disorders. Malden, MA, USA: Blackwell Science Ltd.; 2003. pp. 65-82

Rehm J, Patra J, Taylor B. Harm, benefits, and net effects on mortality of moderate drinking of alcohol among adults in Canada in 2002. Ann Epidemiol 2007; 17:S81-6.

Reichenberg A, Rieckmann N, Harvey PD. Stability in schizophrenia symptoms over time: Findings from the Mount Sinai Pilgrim Psychiatric Center longitudinal study. Journal of Abnormal Psychology 2005; 114(3):363-372.

Rosenfeld L. 'I can't hear the music'. Leenaars AA, Wenckstern S, Sakinofsky I, Dyck RJ, Kral MJ, Blanc RC, ed. Suicide in Canada. Toronto. University of Toronto Press. 1998:376.

Rubin HC, Rapaport MH, Levine B, Gladsjo JK, Rabin A, Auerbach M, et al. Quality of well being in panic disorder: The assessment of psychiatric and general disability. Journal of Affective Disorders 2000; 57:217-221.

Russell E, Sofronoff K. Anxiety and social worries in children with Asperger syndrome. Australian and New Zealand Journal of Psychiatry 2005; 39:633-638.

Russell S, Mammen P, Russell PSS. Emerging trends in accepting the term intellectual disability in the world disability literature (Editorial). Journal of Intellectual Disabilities 2005; 9(3):187-192.

Saarijarvi S, Salminen JK, Toikka T, Raitasalo R. Health-related quality of life among patients with major depression. Nord J Psychiatry 2002; 56(4):261-264.

Safren SA, Heimberg RG, Brown EJ, Holle C. Quality of life in social phobia. Depression and Anxiety 1996/1997; 4:126-133.

Sartorius N. The economic and social burden of depression. Journal of Clinical Psychiatry 2001; 62(suppl 15):8-11.

Sasson Y, Zohar J, Chopra M, Lustig M, Iancu J, Hendler T. Epidemiology of obsessive-compulsive disorder: A world view. J Clin Psychiatry 1997; 58(Suppl 12):7-10.

Schnur J. Asperger syndrome in children. Journal of the American Academy of Nurse Practitioners 2005; 17(8):302-308.

Shear K, Jin R, Meron Ruscio A, Walters EE, Kessler RC. Prevalence and correlates of estimated DSM-IV child and adult separation anxiety disorder in the National Comorbidiy Survey replication. Am J Psychiatry 2006; 163(3):1074-1083.

Sherbourne CD, Wells KB, Judd LL. Functioning and well-being of patients with panic disorder. Am J Psychiatry 1996; 153(2):213-218.

Siris SG. Suicide and schizophrenia. Journal of Psychopharmacology 2001; 15(2):127-135.

Skodol AE, Gunderson JG, Pfohl B, Widiger TA, Livesley WJ, Siever LJ. The borderline diagnosis I: Psychopathology, Comorbidity, and Personality Structure. Biol Psychiatry 2002; 51:936-950.

Skodol AE, Siever LJ, Livesley WJ, Gunderson JG, Pfohl B, Widiger TA. The borderline diagnosis II: Biology, Genetics, and Clinical Course. Biol Psychiatry 2002; 51:951-963.

Slade R. The Anorexia Nervosa Reference Book. London: Harper & Row; 1984.

Sousa MB, Isolan LR, Oliveira RR, Manfro GG, Cordioli AV. A randomized clinical trial of cognitivie-behavioral group therapy and sertraline in the treatment of obsessive-compulsive disorder. J Clin Psychiatry 2006; 67(7):1133-1139.

Springhouse Corporation. Diseases: Causes and Diagnosis, Current Therapy, Nursing Management. Nurses Reference Library, 1987.

Stein MB, Heimberg RG. Well-being and life satisfaction in generalized anxiety disorder: Comparison to major depressive disorder in a community sample. Journal of Affective Disorders 2004; 79:161-166.

Stein MB, Kean YM. Disability and quality of life in social phobia: Epidemiologic findings. Am J Psychiatry 2000; 157:1606-1613.

Stengler-Wenzke K, Kroll M, Matschinger H, Angermeyer MC. Subjective quality of life of patients with obsessive-compulsive disorder. Soc Psychiatry Psychiatr Epidemiol 2006; 41:662-668.

Stephens DL, Collins MD, Dodder RA. A longitudinal study of employment and skill acquisition among individuals with developmental disabilities. Research in Developmental Disabilities 2005; 26:469-486.

Stephens T, Dulberg C, Joubert N. Mental health of the Canadian population: A comprehensive analysis. Chronic Diseases in Canada 2000; 20(3):118-126.

Tidmarsh L, Volkmar FR. Diagnosis and epidemiology of autism spectrum disorders. Can J Psychiatry 2003; 48(8):517-525.

Touyz SW. The treatment of bulimia. (Chapter VIII). Eating Disorders: Prevalence, Treatment. Australia: Williams & Williams; 1985.

Vanin JR, Vanin SK. Blocking the cycle of panic disorder. Postgraduate Medicine Online May 1, 1999; 105(5).

Visser SN, Lesesne CA for the Centers for Disease Control and Prevention. Prevalence of diagnosis and medication treatment for attention-deficit/hyperactivity disorder – United States, 2003. MMWR Morbidity and Mortality Weekly Report 2005; 54: 842-847.

Walker JR, Norton GR, Ross CA. Panic Disorder and Agoraphobia. Ottawa, Canada: Brooks/Cole Publishing Company; 1991.

Waraich P, Goldner EM, Somers JM, Hsu L. Prevalence and incidence studies of mood disorders: A systematic review of the literature. Can J Psychiatry 2004; 49(2):124-138.

Weisbrot DM, Gadow KD, DeVincent CJ, Pomeroy J. The presentation of anxiety in children with pervasive developmental disorders. Journal of Child and Adolescent Psychopharmacology 2005; 15(3):477-496.

Wetherell JL, Thorp SR, Patterson TL, Golshan S, Jeste DV, Gatz M. Quality of life in geriatric generalized anxiety disorder: A preliminary investigation. Journal of Psychiatric Research 2004; 38:305-312.

Wittchen H-UI, Carter RM, Pfister H, Montgomery SA, Kessler RC. Disabilities and quality of life in pure and comorbid generalized anxiety disorder and major depression in a national survey. International Clinical Psychopharmacology 2000; 15(6):319-328.

Wittchen H-U, Fehm L. Epidemiology and natural course of social fears and social phobia. Acta Psychiatr Scand 2003; 108(Suppl 417):4-18.

Wittchen H-U, Fehm L. Epidemiology, patterns of comorbidity, and associated disabilities of social phobia. Psychiatric Clinics of North America 2001; 24(4):617-641.

Wittchen H-U, Yeh E-K. Cross-national epidemiology of major depression and bipolar disorder. JAMA 1996; 276(4):293-299.

WHO (World Health Organization). The global burden of disease: 2004 update. Switzerland: WHO Press; 2008.

WHO (World Health Organization). International Classification of Diseases, Ninth revision. Basic Tabulation List with Alphabetical Index. Geneva: World Health Organization; 1978.

WHO (World Health Organization). International Statistical Classification of Diseases and Related Health Problems, Tenth revision (2nd ed.). Geneva: World Health Organization; 2004. Available online at: ww.who.int/classifications/icd/en.

Yehuda R. Risk and resilience in posttraumatic stress disorder. J Clin Psychiatry 2004; 65 (Suppl 1):29-36.

Zohar J, Sasson Y, Chopra M, Amiaz R, Nakash N. Obsessive-compulsive disorder. In: Nutt DJ, Ballenger JC, Editors. Anxiety Disorders. Malden, MA, USA: Blackwell Science Ltd.; 2003. pp. 83-94.

The following websites were also consulted:

www.adaa.org
Anxiety Disorders Association of America

www.cmha.ca
Canadian Mental Health Association

www.mayoclinic.com
Mayo Clinic

www.mentalhealthamerica.net
Mental Health America

www.nami.org
National Alliance on Mental Illness

www.nimh.nih.gov
National Institute of Mental Health

www.nida.nih.gov
National Institute on Drug Abuse