Pacific Behavioral Health
Collaborating Council (PBHCC)

IC&RC Alcohol and Drug Counselor (ADC)
Academy Curriculum

Day 1: Introduction to the IC&RC ADC
Performance Domains and Review of
Psychoactive Drugs

Trainer Guide

Developed in 2018 by the Pacific Southwest Addiction Technology Transfer Center and UCLA Integrated Substance Abuse Programs
# IC&RC Alcohol and Drug Counselor Academy, Day 1

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IC&RC Alcohol and Drug Counselor Academy, Day 1

Background Information
The IC&RC Alcohol and Drug Counselor (ADC) Academy curriculum is a weeklong training designed to prepare individuals based in the six U.S.-affiliated Pacific Jurisdictions to successfully pass the IC&RC ADC certification exam. The duration of the ADC Academy is forty hours of content spread across five full days of training. Funding for the development of the ADC Academy was provided by the Pacific Behavioral Health Collaborating Council (PBHCC). The curriculum is broken into five modules/days, which include:

- Day 1: Introduction to the IC&RC ADC Performance Domains and Review of Psychoactive Drugs
- Day 2: Core Competencies of Addiction Counselors – Knowledge and Skill Acquisition of Screening, Intake, Orientation, Assessment, Treatment Planning, and Counseling
- Day 3: Core Competencies of Addiction Counselors – Knowledge and Skill Acquisition of Case Management, Crisis Intervention, Client and Family Education, Referral, Report and Record Keeping, and Consultation
- Day 4: Core Competencies of Addiction Counselors – Prevention and Treatment of HIV/AIDS and Sexually Transmitted Infections
- Day 5: Course Review and Test-Taking Strategies

What Does the Training Package Contain?
- PowerPoint Training Slides (with notes)
- Trainer’s Guide with detailed instructions for how to convey the information and conduct the interactive exercises
What Does This Trainer’s Guide Contain?

- Slide-by-slide notes designed to help the trainer effectively convey the content of the slides themselves
- Supplemental information for select content to enhance the quality of instruction
- Suggestions for facilitating group discussions

How is This Trainer’s Guide Organized?

For this guide, text that is shown in bold italics is a “Note to the Trainer.” Text that is shown in normal font relates to the “Trainer’s Script” for the slide.

It is important for trainers to become acquainted with the slides and practice delivering the content of the presentation, ensuring a successful, live training experience.

General Information about Conducting the Training

The training is designed to be conducted in medium-sized groups (20-30 people). It is possible to use these materials with larger groups, but the trainer may have to adapt the small group exercises/case studies and discussions to ensure that there is adequate time to cover all of the content.

Materials Needed to Conduct the Training

- Computer with PowerPoint software installed (2010 or higher version recommended) and LCD projector to show the PowerPoint training slides.

- When making photocopies of the PowerPoint presentation to provide as a handout to training participants, it is recommended that you print the slides three slides per page with lines for notes. Select “pure black and white” as the color option. This will ensure that all text, graphs, tables, and images print clearly.

- Flip chart paper and easel/white board, and markers/pens to write down relevant information, including key case study discussion points.
Overall Trainer Notes

It is critical that, prior to conducting the actual training, the trainer practice using this guide while showing the slide presentation in Slideshow Mode in order to be prepared to use the slides in the most effective manner.

Icon Key

<table>
<thead>
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PBHCC Alcohol and Drug Counselor (ADC) Academy, Day 1

Introduction to the IC&RC ADC Performance Domains and Review of Psychoactive Drugs

Slide-By-Slide Trainer Notes

The notes below contain information that can be presented with each slide. This information is designed as a guidepost and can be adapted to meet the needs of the local training situation. Information can be added or deleted at the discretion of the trainer(s).

Slide 1: [Title Slide]

- Welcome participants to the training
- Orient participants to the training room/facility and to the nearest bathroom
- Introduce all trainers
- Be prepared to project the Center for Substance Abuse Treatment (2006) Technical Assistance Publication 21
- Briefly orient participants to the various handouts and resources they have received
Slide 1: [Title Slide]

All participants should receive the following:

Flash drive or link to:

- PDF copies of all slide decks
- CSAT Technical Assistance Publications:
Slide 1: [Title Slide]

- CSAT Treatment Improvement Protocol:
  

(Notes for Slide 1, continued)

Slide 1: [Title Slide]


- Paper copies of the following:
  - Candidate Guide for the International Certification and Reciprocity Consortium (IC&RC) Alcohol and Drug Counselor (ADC) Examination
Slide 2: Acknowledgements

- This training was developed by Drs. Thomas Freese and Christopher Rocchio from the University of California Los Angeles, Integrated Substance Abuse Programs (UCLA ISAP) and with Alex Ngiarangas, an addictions counselor and educator from the Republic of Palau. We would like to acknowledge and thank the Pacific Behavioral Health Collaborating Council (PBHCC) for their commitment to train individuals across the Pacific to effectively prevent, treat, and support individuals in their own recovery from substance use disorders, and for their financial support for the development and delivery of this curriculum. Additional resource provided by SAMHSA, grant number UR1TI080211.

Slide 3: Disclaimer

- [READ THE SLIDE]
Slide 4: Agenda for Day One

- Orient participants to the agenda
  - [ASK PARTICIPANTS] What questions do you have regarding today’s agenda?
  - [ASK PARTICIPANTS] What is missing from this agenda that you were hoping to get?

Slide 5: Introductions

- [ASK PARTICIPANTS] Please introduce yourself, where are you from, what do you hope to learn, and what were some of your expectations coming into this training?

Slide 6: Agenda (1)

- We will now review this week’s agenda.
Slide 7: Agenda for Day Two

- Orient participants to the agenda for day 2.

Slide 8: Agenda for Day Three

- Orient participants to the agenda for day 3.

Slide 9: Agenda for Day Four

- Orient participants to the agenda for day 4.
Slide 10: Agenda for Day Five

- Orient participants to the agenda for day 5.

Slide 11: Agenda (2)

- [ASK PARTICIPANTS] What questions do you have regarding this week’s agenda?
- [ASK PARTICIPANTS] What is missing in today’s agenda that you were hoping to get?
Slide 12: IC&RC Alcohol and Drug Counselor (ADC) Exam

- Ask participants to retrieve the Candidate Guide for the IC&RC ADC examination from their training packet.

- Please closely review the candidate guide for the International Certification and Reciprocity Consortium (IC&RC) Alcohol and Drug Counselor (ADC) Examination later today and prior to taking the exam. Besides the paper copy you are being provided today, we have also included an electronic copy of the candidate guide in your resource folder. The IC&RC ADC exam tests for competency of entry-level counselors.

- The ADC exam is comprised of 150 multiple choice questions. 125 of the test items are scored, and the other 25 are unweighted. These questions are referred to as pre-test items. Pretest items appear randomly throughout the examination. They are not identified and do not influence your final score.
Slide 12: IC&RC Alcohol and Drug Counselor (ADC) Exam

- You are given three hours to complete the test. Carefully read each question and choose the single best answer.

REFERENCE

Questions asked on the exam are based on the 2013 IC&RC job-task analysis. Results from the analysis identified specific tasks of entry-level counselors. Using factor analysis, tasks were organized into four factors known as performance domains. The 4 performance domains are:

- Screening, Assessment, and Engagement;
- Treatment Planning, Collaboration, and Referral;
- Counseling; and
- Professional and Ethical Responsibilities

The percentages following each performance domain refer to their relative weight on the exam.

Each performance domain is comprised of specific tasks.

Eight tasks are associated with screening, assessment, and engagement, 10 tasks associated with treatment planning, collaboration, and referral, 8 tasks associated with professional and ethical responsibilities, and 7 tasks associated with professional and ethical responsibilities.
(Notes for Slide 13, continued)

Slide 13: ADC Performance Domains

- The exam evaluates candidates’ proficiency and competence in the necessary knowledge and skills needed to perform specific tasks associated with each performance domain in the role of an entry-level addictions counselor.

- Please open the candidate guide and turn to page 11.

- Beginning on page 11, the four performance domains and their corresponding tasks can be found.

- Please only review the first task for screening, assessment, and engagement.

- [ASK PARTICIPANTS] What knowledge and skills would you need to perform this task?
Slide 13: ADC Performance Domains

• The 2013 Job-Task Analysis found and subject matter experts in addictions counseling agreed that the ability to demonstrate verbal and nonverbal communication to establish rapport and promote engagement requires knowledge of best practices for interviewing others, self-awareness, the stages of change, and how culture affects communication. Demonstration of this task would be evident in the counselor’s ability to engage and build trust with clients, and the ability and use of stage-appropriate interventions.

REFERENCES


Slide 14: The 12 Core Functions

- The four performance domains are a consolidated version of the IC&RC’s 12 core functions. Similar to tasks for each performance domain, the historic 12 core functions include a set of global criteria for each core function. As Herdman (2018) explains, global criteria are the necessary skills needed to perform a specific core function. Altogether, there are 46 global criteria. In days 2 and 3, we will review the 12 core functions.

REFERENCE
Herdman (2018) organized a crosswalk of the four performance domains to the 12 core functions. It is important to highlight here that the original core function of report and record keeping is applicable across all four performance domains. Herdman (2018) highlights that the original 12 core functions did not include a core function specific to ethics. He and others agree that this was a welcome addition to the IC&RC exam.

REFERENCE

Slide 16: Agenda (3)

- [ASK PARTICIPANTS] Do you have any questions regarding the 12 core functions of the 4 performance domains?

Slide 17: Technical Assistance Publication 21

- Since it was first published in 1998, the Substance Abuse and Mental Health Services Administration (SAMHSA), Center for Substance Abuse Treatment (CSAT), Addiction Counseling Competencies, Technical Assistance Publication (TAP) 21 has become a “benchmark by which curricula are developed and educational programs and professional standards are measured for the field of substance abuse treatment in the United States” (CSAT, 2006, p. 1).
Slide 17: Technical Assistance Publication 21

- TAP 21 identifies 123 competencies that are essential to the effective practice of counseling individuals in different stages of recovery from substance use disorders. In addition to the 123 competencies, “TAP 21 presents the knowledge, skills, and attitudes counselors need to become proficient in each competency.” Similar to the IC&RC performance domains, the core competencies were consolidated and organized into 4 transdisciplinary foundations and 8 practice dimensions. The four transdisciplinary foundations are comprised of discrete building blocks that are applicable to all disciplines working with individuals with substance use disorders. The eight practice domains are similar to the IC&RC’s 12 core functions and 4 performance domains. Each practice dimension specially addresses the professional practice needs and competencies of addiction counselors.
REFERENCE

After reviewing this slide, project page 9 of TAP 21 on screen. Review one or more competencies from each of the four foundations.

The transdisciplinary foundations are comprised of the following four sets of competencies: understanding addiction, treatment knowledge, application to practice, and professional readiness. The 23 competencies identified within these four foundations should be considered as prerequisites to the remaining 100 competencies identified in the 8 practice dimensions.
Slide 18: Four Transdisciplinary Foundations

REFERENCE

After reviewing this slide, project page 39 of TAP 21 onto the screen. Review one or more competencies from each of the eight practice dimensions.

The eight practice dimensions include: clinical evaluation; treatment planning; referral; service coordination; counseling; client, family, and community education; documentation; and professional and ethical responsibilities. Three of the eight practice dimensions (i.e., clinical evaluation, service coordination, and counseling) are subdivided into elements.

The CSAT asserts that knowledge effective performance in these competencies are necessary for counseling individuals in recovery from substance use disorders.
Slide 19: Eight Practice Dimensions

REFERENCE


Slide 20: Agenda (4)

- [ASK PARTICIPANTS] What questions do you have about the IC&RC 12 core functions and 4 performance domains or the TAP 21 addiction counselor competencies?
Slide 21: Clarifying Terms

- All treatment providers, as highlighted in TAP 21 “must have a basic understanding of addiction that includes knowledge of current models and theories, appreciation of the multiple contexts within which substance use occurs, and awareness of the effects of psychoactive drug use” (CSAT, 2006, p.5).

- We would like to establish and clarify terms that will be used throughout this training.

- [READ BULLETED LIST ON SLIDE]

[ASK PARTICIPANTS]: What thoughts or comments do you have regarding these 2 definitions?
Slide 21: Clarifying Terms

REFERENCE


Slide 22: Addiction Is a Brain Disease, and It Matters

In the late 1990s Dr. Alan Leschner, former Director of the National Institute on Drug Abuse (NIDA), did a review of the research of substance related conditions and began an education campaign about substance use disorders, demonstrating that, based on the best science, substance use disorders were best characterized as a brain disease. In this article he demonstrated that like other chronic diseases (e.g., diabetes, hypertension), this chronic brain diseases needed ongoing care and support.
(Notes for Slide 22, continued)

Slide 22: Addiction Is a Brain Disease, and It Matters

- [ADVANCE ANIMATION] Read quote from Alan Leschner.
- [ASK PARTICIPANTS]: What thoughts or comments do you have regarding these 2 definitions?

Slide 23: How Do We Conceptualize “Addiction”?

- The conceptualization of substance use disorders as a chronic brain disease is controversial.

- [SUMMARIZE/READ BULLETED LIST ON SLIDE]
- [ASK PARTICIPANTS]: What thoughts or comments do you have regarding this “brain disease” concept?
Slide 24: Why do people take drugs?

• [ASK PARTICIPANTS]: Why do people take drugs?

• [CLICK TO ADVANCE ANIMATION]

• [READ BULLETED LIST UNDER “To Feel Good.”]

• [CLICK TO ADVANCE ANIMATION]

• [READ BULLETED LIST UNDER “To Feel Better.”]

Slide 25: In other words

• [READ SLIDE]
Slide 26: What is Addiction?

- Decades of research have revealed addiction to be a disease that alters the brain. We now know that while the initial decision to use drugs is voluntary, drug addiction is a disease of the brain that compels a person to become singularly obsessed with obtaining and abusing drugs despite their many adverse health and life consequences.

REFERENCE


IMAGE CREDIT

NIDA website.
Slide 27: Addiction is Like Other Diseases...

- It is preventable
- It is treatable
- It changes biology
- If untreated, it can last a lifetime

Slide 28: Imaging Chronic Disease

- Addiction is similar to other chronic diseases. Using imaging technology to measure metabolism (in this case, glucose uptake) in the brain and heart, one can see that both addiction and heart disease produce observable changes in organ function. In each pair of images shown above, the healthy organ shows greater activity (reds and yellows) than the diseased organ. In addiction, the frontal cortex, which is a part of the brain associated with judgment and decision-making, is significantly affected. Like heart disease, drug addiction can be prevented and treated successfully. If left untreated, however, its effects can last a lifetime.
Slide 28: Imaging Chronic Disease

REFERENCE

IMAGE CREDIT
NIDA website.

Slide 29: Agenda (5)

- [ASK PARTICIPANTS] Are there any questions regarding addiction as a brain disease?
Slide 30: Science of Addiction

- Advances in medicine and scientific techniques have given researchers a clearer idea of what addiction is:
  - Magnetic resonance imaging (MRI)
  - Positron emission tomography (PET) scan
  - Advanced genetic research

Slide 31: Brain Areas Affected by Drugs

- This slide depicts a cartoon image of the human brain
- The Prefrontal Cortex is associated with our judgement/executive level decision making
- The Nucleus accumbens is associated with our cognitive processing of aversion, motivation, pleasure, reward, and reinforcement learning
- The Limbic system is associated with our center of emotions, learning, and memory; cingulate gyri, hypothalamus, amygdala (emotional reactions), and hippocampus (memory)
- The Cerebellum is associated with coordination
Slide 31: Brain Areas Affected by Drugs

REFERENCE

IMAGE CREDIT
NIDA website.

Slide 32: Neurotransmitters Definition

REFERENCE
Slide 33: Major Neurotransmitters Involved in SUD

- [READ BULLETED LIST] Note: This is to introduce the neurotransmitters. Do not review here as the full definitions come in the following slides.

Slide 34: Basic Neurotransmitters

- [READ BULLETED LIST] Note: This is to introduce the neurotransmitters. Do not review here as the full definitions come in the following slides.

Slide 35: Neurotransmitters

- [SUMMARIZE/READ EACH BULLET]

Major Neurotransmitters Involved in SUD
- Dopamine
- Serotonin
- Norepinephrine
- GABA
- Glutamate

Basic Neurotransmitters
- Norepinephrine
- Dopamine
- Serotonin
- Glutamate
- Gamma-aminobutyric acid or GABA
- Enkephalins or Endorphins

Neurotransmitters
- Norepinephrine, a neurotransmitter found in the brain, associated with arousal reactions & mood.
- Dopamine – a neurotransmitter found in the brain associated with body movement and pleasure.
- Serotonin is normally involved in temperature regulation, sensory perception, and mood control. However, it plays a major role in emotional disorders such as depression, suicide, impulsive behavior, and aggression. The hallucinogenic drug LSD acts on Serotonin receptors; so do some antidepressant drugs.
Neurotransmitters (continued)

- Glutamate and GABA (gamma-amino butyric acid) are amino acids that act as neurotransmitters. The majority of synapses within the brain use glutamate or GABA. They also have other functions in the body such as making energy-rich molecules in cells. The fact that GABA and glutamate are so widely present makes it likely that they will be altered during drug addiction. This also makes it difficult to treat addiction with drug therapy without causing side effects.
- Enkephalins and Endorphins: first discovered in 1975, both compounds have pain killing properties that are more powerful than morphine, with endorphins being 40 times more powerful than enkephalins, and 100 times more powerful than morphine.

Neurotransmitters (continued 2)

- Current research seems to suggest that the presence or absence of such compounds as endorphins & especially dopamine may explain several conditions including compulsive drug abuse, chemical dependence, pain management, sexual activity, schizophrenia, and the natural "high" of exercise that many people experience.
- Further research is needed to help us identify exactly how this knowledge may be applied to successful treatment and recovery issues.

Slide 37: Neurotransmitters (continued 2)

- Research demonstrates that these neurotransmitters are important in many behaviors and conditions. Changes in levels can impact both experience and expression of disease. But we need more research in order to understand how we can use this in providing treatment and recovery to an individual client/patient.
This slide shows how neurotransmission works specifically for dopamine. What is schematically illustrated in this slide is a nerve terminal (top), the synaptic cleft or space between the neurons, and the post-synaptic or receiving portion of a dendrite on a neighboring neuron. Dopamine is contained in vesicles (round storage sites) in the nerve terminal; dopamine receptors are present on the receiving (bottom) neuron.

When a signal comes down the axon, dopamine (shown in orange) is released into the synapse. It then crosses the synaptic cleft to the second neuron, where it binds to and stimulates dopamine receptors (shown in blue), generating a signal in the second neuron. The dopamine is then released from the receptor and crosses back to the first neuron where it is picked up by dopamine transporters (reuptake molecules; shown in purple) for re-use.
Slide 38: Normal Dopamine Transmission

• [ADVANCE SLIDE TO PLAY VIDEO]

VIDEO SOURCE
Natural Rewards Elevate Dopamine Levels

- Natural rewards stimulate dopamine neurotransmission. Eating something that you enjoy or being stimulated sexually can cause dopamine levels to increase. In these graphs, dopamine is being measured inside the brains of animals. Its increase is shown in response to food or sex cues. This basic mechanism of controlled dopamine release and reuptake has been carefully shaped and calibrated by evolution to reward normal activities critical for our survival.

REFERENCE

Methamphetamine and Dopamine Transmission

- **[ADVANCE SLIDE TO PLAY VIDEO]**
- When meth is added to the system, it pushes dopamine from the descending neuron into the synapse, causing a flood of dopamine.
- Meth then blocks the reuptake transporters so that it remains in the synapse for an extended period of time. This is what causes the intense euphoric feelings that meth produces. This can go on for an extended time with a single dose of methamphetamine producing this effect for 10+ hours.

**VIDEO SOURCE**

Slide 41: Effects of Drugs on Dopamine Release

- When scientists looked at the impact of drugs used by humans on dopamine output in rats, they found that all of them had a direct impact.

- With alcohol, also called ethanol, dopamine output doubled from baseline; a spike similar to what was seen with the sex response.

- With nicotine, a similar spike was seen.

- With cocaine, dopamine output increased about 3.5 times over baseline.

- With methamphetamine the increase was about 1200 times baseline.

REFERENCE

Slide 42: Decreased dopamine transporter binding in METH users

- The scan on the far left of the screen depicts the brain of a non-methamphetamine using control. Notice the bright colors in the reward center. The center scan is the brain of a methamphetamine user who has been matched to the control in terms of age, race, etc. Notice that there are no bright red and orange colors in the reward center. This indicates far less dopamine activity in the meth brain.

REFERENCE

Slide 43: But Dopamine is only Part of the Story

- Scientific research has shown that other neurotransmitter systems are also affected:
  - Serotonin
    - Regulates mood and sleep
  - Glutamate
    - Regulates learning and memory

Slide 44: Prolonged Drug Use Changes The Brain In Fundamental and Long-Lasting Ways

- [READ/PARAPHRASE CONTENT]

IMAGE CREDIT
NIDA website.
Slide 45: We Have Evidence That These Changes Can Be Both Structural and Functional

• [READ/PARAPHRASE CONTENT]

IMAGE CREDIT
NIDA website.

Slide 46: Brain Changes

• While in the short term, dopamine output is enhanced through use of drugs/alcohol, long-term use of substances can contribute to depletions and deficits in activity in the brain. This slide shows what normal activity looks like in a PET scan (left half of brain image), as compared to the activity of a chronic substance user (right half of brain image).

IMAGE CREDIT
NIDA website.
Dopamine D2 Receptors are Lower in Addiction

- Repeated drug exposure also changes brain function. Positron emission tomography (PET) images show similar changes in brain dopamine receptors resulting from addiction to different substances. Dopamine D2 receptors are one of five types of receptors that bind dopamine in the brain. The brain images on the left are those of controls, while those on the right are from individuals addicted to cocaine, methamphetamine, alcohol, or heroin. The striatum (which contains the reward and motor circuitry) shows up as bright red and yellow in the controls (in the left column), indicating numerous D2 receptors. Conversely, the brains of addicted individuals (in the right column) show a less intense signal, indicating lower levels of D2 receptors. This reduction likely stems from repeated over-stimulation of the dopamine receptors. Brain adaptations such as this contribute to the compulsion to abuse drugs.
Slide 47: Dopamine D2 Receptors are Lower in Addiction

REFERENCE

Changes in the Prefrontal Cortes

- The limbic system lies between the brain stem and the cerebral cortex. It is involved with learning, motivation, memory, and emotion. Methamphetamine does much of its work in two key structures in the limbic system: the amygdala and the hippocampus.

- In this comparative scan of a control and methamphetamine abuser, you see a deactivation in the prefrontal cortex of the meth abuser, the part of the brain responsible for high-order decision making. This means that methamphetamine abusers are less able to make higher-order decisions (like stopping their drug use).
Slide 49: Changes in the Amygdala

- In this comparative scan of a control and methamphetamine abuser, you see a hyper-activation in the amygdala of the methamphetamine abuser, the part of the brain responsible for emotional processing (and the fight or flight reaction). Stimulation of the amygdala is associated with increased emotionality – specifically fear, anxiety, irritability, and anger. Methamphetamine stimulation of this brain area would explain the extreme emotionality and occasionally violent reactions that chronic methamphetamine users sometimes exhibit.

Slide 50: “Go” and “Stop” Circuits

- GO pathway – ventral tegmental area, nucleus accumbens, the lateral hypothalamus, and the amygdala
- STOP pathway – prefrontal and orbitofrontal cortices, and the connective fibers: fasciculus retroflexus and lateral habenula
Slide 51: Cognitive and Memory Effects

- The next portion of the training reviews the cognitive and memory effects of psychoactive substances on the user.

Slide 52: Dopamine Transporters in Methamphetamine Abusers

- Methamphetamine use decreases dopamine transporter activity and compromises mental function. The brain image at the top left is a PET image from a normal control subject. The striatum is brightly lit in red and yellow, indicating the presence of many dopamine transporters, which contrasts with the brain of a methamphetamine abuser (bottom left). What does this mean functionally? The graphs on the right show the relationship between performance on a motor (upper right) and a memory task (lower right) and methamphetamine-driven decreases in dopamine transporters.
Slide 52: Dopamine Transporters in Methamphetamine Abusers

- The magnitude of the decline in the dopamine transporter binding is positively correlated with the extent of motor and memory impairment; thus the greater the decline, the greater the impairment in memory and motor reaction time.

REFERENCE
In this study, researchers examined the PET scans of chronic methamphetamine users who had achieved two years of abstinence from methamphetamine. The scans showed a return to virtually normal dopamine levels. While this is good news, and suggests that the brain has an amazing ability to repair itself, the subjects in the study did not regain all of the lost cognitive function associated with the damage, which could suggest an incomplete recovery.

While the fact that the brain recovers is good news, the not-so-good news is that the recovery takes months, not days. Treatment and recovery are long-term processes.
Slide 53: Partial Recovery of Brain Dopamine Transporters in Methamphetamine Abuser After Protracted Abstinence

REFERENCE

Slide 54: Cocaine Impact on Glucose Metabolism

• Brain glucose metabolism with PET – chronic cocaine abusers vs. normal controls; chronic cocaine abusers were tested 1-6 weeks after last use of cocaine and again after 3-month drug-free period.

• Cocaine abusers had significantly lower metabolic activity in 16 of the 21 left frontal regions and 8 of the 21 right frontal regions. These decreases persisted after 3-4 months of detoxification and were correlated with dose and years of use.

• Conclusion – reduced frontal metabolism in neurologically intact cocaine abusers that persist even after 3-4 months of detoxification.
Slide 54: Cocaine Impact on Glucose Metabolism

REFERENCE

Slide 55: What does this mean for clients?

- This next portion of the training reviews discusses addiction as a brain disease.

IMAGE CREDIT
Purchased image.
Addiction is, Fundamentally, a *Brain Disease*

...BUT

It’s Not *Just* a Brain Disease

Slide 56: Addiction is, Fundamentally a Brain Disease

• READ SLIDE
Environmental factors

- “No single factor determines whether a person will become addicted to drugs. The overall risk for addiction is impacted by the biological makeup of the individual – it can even be influenced by gender or ethnicity, his or her developmental stage, and the surrounding social environment (e.g., conditions at home, at school, and in the neighborhood (NIDA, 2010).”

REFERENCE

Slide 58: Vulnerability to addiction differs from person to person

- “As with any other disease, vulnerability to addiction differs from person to person. In general, the more risk factors an individual has, the greater the change that taking drugs will lead to abuse and addiction. ‘Protective’ factors reduce a person’s risk of developing addiction. Scientists estimate that genetic factors account for between 40 and 60 percent of a person’s vulnerability to addiction, including the effects of environment on gene expression and function (NIDA, 2010).”

REFERENCE


IMAGE CREDIT

Purchased image.
Slide 59: Drug addiction is a chronic brain disorder

- The brain changes that occur as a result of ongoing drug use can be long lasting, and can persist long after the user has stopped using drugs. Studies show the brain can recover and return towards normal, but this recovery process takes time (exact time depends upon a number of factors).

IMAGE CREDIT
Purchased image.

Slide 60: Cognitive Effects of Chronic Substance Use

- General effects of most substances:
  - Episodic memory
  - Emotional processing
  - Executive functions

[SUMMARIZE/READ BULLETED LIST]
Cognitive Effects of Chronic Substance Use (continued)

- Specific effects:
  - Alcohol and Psychostimulants: impulsive action and cognitive flexibility
  - Alcohol and MDMA: spatial processing, perceptual speed, and selective attention
  - Cannabis and Methamphetamine: prospective memory
  - Cannabis and MDMA: processing speed and decision making

REFERENCE

Strategies for Cognitive Impairment

- Reducing substance use may be more acceptable than total abstinence
  - Any reduction in use is progress
  - Affirm early successes to enhance self-efficacy
  - When beginning treatment and during early recovery, clients often feel worse before they feel better
  - Educate client to anticipate changes in mood, symptoms, lifestyle, and peer relations
REFERENCE

Strategies for Cognitive Impairment 2

MODIFY TREATMENT PROTOCOLS
- Decrease length of sessions (attention, memory)
- Take structured breaks (attention, focus, memory)
- Increase session frequency (practice)
- Repeat presentations of therapeutic information (daily, every week, every month, every three months, etc.)
- Multi-modal presentations—audio, visual, experiential, verbal, hot/cold situations, etc.

How could you do this at your clinic?

REFERENCES


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Strategies for Cognitive Impairment 3

- Use memory aids—calendars, planners, phone apps, diagrams
- Teach stress management, breathing, relaxation, and mindfulness meditation skills
- Provide immediate feedback and corrective experiences
- Repeat instructions, put things in writing, provide short/direct instructions

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Slide 64: Strategies for Cognitive Impairment 3

- [SUMMARIZE/READ BULLETED LIST]
Slide 65: Impact of Alcohol and Other Drugs on Adolescent Brain Development

This next portion of the training reviews the impact that alcohol and other drugs have on adolescent brain development.

Slide 66: Addiction is a Developmental Disease: It Starts Early

- Addiction is a developmental disease that usually begins in adolescence. If a person can make it through young adulthood without being diagnosed with and SUD, they are very unlikely to develop one. For example, 67 percent of those who try marijuana for the first time are between the ages of 12 and 17. Prevention efforts are therefore of primary importance—to stop drug abuse before it ever starts.
Slide 67: Continuing Brain Development

- This drawing illustrates the pruning process.
- Between birth and 6 years of age...
- ...there is a tremendous proliferation of neural connections.
- This is followed by sustained thinning starting around puberty. Scientists think this process reflects greater organization of the brain as it prunes redundant connections, and increases in myelin, which enhance transmission of brain messages.

REFERENCE

Slide 68: Brain Development Ages 5-20 years

- Constructed from MRI scans of healthy children and teens, the time-lapse movie, from which the above images were extracted, compresses 15 years of brain development (ages 5–20) into just a few seconds. Red indicates more gray matter, blue less gray matter. Gray matter wanes in a back-to-front wave as the brain matures and neural connections are pruned. Areas performing more basic functions mature earlier; areas for higher order functions mature later. The prefrontal cortex, which handles reasoning and other "executive" functions, emerged late in evolution and is among the last to mature. Studies in twins are showing that development of such late-maturing areas is less influenced by heredity than areas that mature earlier.

- [ANIMATION: This slide has complex animations and the trainer should practice prior to training. A step-by-step guide is provided below]
Slide 68: Brain Development Ages 5-20 years

- [The first bullet comes in automatically at the beginning of the slide. Provide the following description]
  
  - This slide demonstrates the neural pruning through animations. This is a series of MRI scans from healthy children showing brain development as they age from 5 to 20 years.

- [Move forward to reveal the next bullet, and present the information]
  
  - Red indicates more gray matter and blue indicates less gray matter.
Slide 68: Brain Development Ages 5-20 years

• [Move forward and a small brain image will briefly appear on the lower right and then a short movie will automatically play full screen showing brain maturation. Once it stops, the small image of the brain will appear again on the lower right of the slide. Move forward to reveal the next bullet]

• As you can see, the pruning occurs from the back of the brain toward the front.

• [Move forward to reveal the last bullet]
Slide 68: Brain Development Ages 5-20 years

- This means that the prefrontal cortex (responsible for executive functioning, like decision-making) is the last to mature.

REFERENCES


In reality, the exact impact of substance use on the developing brain is not known. However, when we look at the impact on the adult brain and understand normal development, several things seems true about this interaction, including that it may lead to difficulties in decision making and understanding the consequences of behavior (Fiellin, 2013). Additionally, it may increase the risk of memory and attention problems. These impairments, in turn, may lead to increased experimentation across a variety of behaviors; and increase the risk of addiction to a variety of substances (Fiellin, 2013).

REFERENCE
A variety of studies have been conducted to examine the impact of marijuana on the adolescent brain. The following are descriptions of the three such studies:

- **Lisdahl & Price, 2011**: Increased MJ use was associated with slower psychomotor speed/sequencing ability ($p < .01$), less efficient sustained attention ($p < .05$), and increased cognitive inhibition errors ($p < .03$). Gender significantly moderated the effects of MJ on psychomotor speed/sequencing ability ($p < .003$) in that males had a more robust negative relationship. The current study demonstrated that MJ exposure was associated with poorer psychomotor speed, sustained attention and cognitive inhibition in a dose-dependent manner in young adults, findings that are consistent with other samples of adolescent MJ users. Male MJ users demonstrated greater cognitive slowing than females.
• Ehrenreich et al, 1999: Of the potential predictors of test performance within the user group, including present age, age of onset of cannabis use, degree of acute intoxication (THC+THCOH plasma levels), and cumulative toxicity (estimated total life dose), an early age of onset turned out to be the only predictor, predicting impaired reaction times exclusively in visual scanning. Early-onset users (onset before age 16; n = 48) showed a significant impairment in reaction times in this function, whereas late-onset users (onset after age 16; n = 51) did not differ from controls (n = 49). These data suggest that beginning cannabis use during early adolescence may lead to enduring effects on specific attentional functions in adulthood. Apparently, vulnerable periods during brain development exist that are subject to persistent alterations by interfering exogenous cannabinoids.
Slide 70: Marijuana and the Adolescent Brain (1)

Fontes et al., 2011: The early-onset group showed significantly poorer performance compared with the controls and the late-onset group on tasks assessing sustained attention, impulse control and executive functioning. Early-onset chronic cannabis users exhibited poorer cognitive performance than controls and late-onset users in executive functioning. Chronic cannabis use, when started before age 15, may have more deleterious effects on neurocognitive functioning.

REFERENCES


REFERENCES, continued

People who began using marijuana in their teenage years and then continued to use marijuana for many years lost about 8 IQ points from childhood to adulthood, whereas those who never used marijuana did not lose any IQ points. The amount people smoked also made a difference. Those who smoked the most – at least every day – saw the greatest drop in IQ, the full 8 points. And the younger they were when they started using cannabis, the greater the IQ decline. It wasn’t just IQ. Adults who smoked marijuana as teenagers did worse in tests of memory and decision-making than adults who hadn’t smoked yet.

New research makes this finding more controversial as there may be confounding variables associated with socioeconomics.
REFERENCES


Slide 72: Marijuana and the Adolescent Brain (3)

- Summarize bulleted list

  - Neuronal efficiency is a measure of the level of activation of the brain in a particular situation. Poor neuronal efficiency is associated with increased difficulty in memory recall.

  - Poorer cognitive functioning factors are listed on the slide.

REFERENCES

Slide 72: Marijuana and the Adolescent Brain (3)

REFERENCES, continued

Slide 73: Drug addiction is a chronic relapsing disorder similar to other chronic diseases

• Transition slide

• [SUMMARIZE/READ CONTENT]

Slide 74: Why are we comparing SUD to these particular illnesses

• [SUMMARIZE/READ CONTENT]
Slide 75: Relapse Rates Are Similar for Drug Dependence and Other Chronic Illnesses

Graph: Percent of Relapse by Chronic Condition

Graph shows Percentage of people who relapse on the Y axis. In this case relapse indicates return to behaviors that negatively impact cognition.

- Chronic disease management requires both medical and behavioral interventions to manage them successfully. Relapse in these conditions could be define as cessation/inconsistent medication adherence, lack of monitoring of the condition (e.g., not monitoring blood sugar in diabetes), and/or return of behaviors that negatively impact the chronic condition.

- If we compare relapse across these chronic conditions, we see rates of 30-50% for Type 1 Diabetes, 50-70% for Hypertension and Asthma, and 40-60% for Substance Use Disorders (called drug dependence here according to DSM-IV guidelines).
Slide 75: Relapse Rates Are Similar for Drug Dependence and Other Chronic Illnesses

In other words, individuals with substance use disorders relapse at about the same rate as people with other chronic diseases, supporting the idea that we should be managing substance use disorders in ways that are similar to other chronic conditions.

REFERENCE

Slide 76: Treating a Behavioral Disorder Must Go Beyond Just Fixing the Chemistry

- Addiction requires treatment that addresses its complexity. Substance abuse treatment should address the whole person and can include medications, health care services, behavioral therapies, spiritual wellbeing, and ancillary support services.

IMAGE CREDIT
Purchased image.

Slide 77: Four Legs of Addiction Treatment

- Alcohol and drug addiction is affected by many factors, including development, physiology, genetics, social influence, personality, coping discrepancies, spiritual values, reinforcement, conditioning, abuse, self-regulated use, and dependence. All of these factors point to initial use, and can be linked to one or more other factors.
(Notes for Slide 77, continued)

**Slide 77: Four Legs of Addiction**

**Treatment**

- A person’s treatment plan should be holistic in nature and address the multiple needs of the individual, such as sexual orientation, gender differences, homelessness, family dynamics, children/prenatal care, legal issues, disabilities, employment issues, developmental needs, co-occurring disorders, and cultural, racial/ethnic, and religious norms.

**Slide 78: Full recovery is a challenge but it is possible...**

- [READ SLIDE]
- People do recover from addiction. Research bears this out.
Extended abstinence is predictive of sustained recovery. The odds of remaining abstinent rise if patients have been abstinent for 1 to 3 years. After 3 years, the recovery odds remain high and stable. Therefore, as with other chronic diseases, addiction requires an ongoing and active disease management strategy.

REFERENCE

Slide 80: Dopamine Transporter Recovery with Prolonged Abstinence from Methamphetamine

- It takes time, but the brain can recover. This slide shows images of dopamine transporter (DAT) binding in three brains: (1) a healthy control (top); (2) a methamphetamine abuser one month after discontinuing drug abuse (middle); and (3) a methamphetamine abuser after 14 months of abstinence (bottom). The control brain shows a robust concentration of dopamine transporters in the striatum (red and yellow), while the methamphetamine abuser has a dramatic drop in DAT binding, even a month after drug abuse has stopped. Sustained abstinence, however, allows a near-full return of DAT binding to normal levels. Still, some of the behavioral effects of methamphetamine do not completely return to normal (not shown). This means that it can take a long time to recover from methamphetamine abuse, but recovery is possible.
Slide 80: Dopamine Transporter Recovery with Prolonged Abstinence from Methamphetamine

**REFERENCES**


**Slide 81: Treatment Reduces Drug Use and Recidivism**

- SUD treatment works and brings about reductions not just in drug use, but also in criminal recidivism. A therapeutic community approach (CREST) was tested in prison and continued during participants’ transition back to the community. Among those who completed treatment and aftercare programs, 35% remained drug free, and 69% were not arrested within three years of release from incarceration. These reductions in turn yield significant cost savings and provide hope for families and communities devastated by addiction.
Slide 81: Treatment Reduces Drug Use and Recidivism

REFERENCE


Slide 82: Lessons from Chronic Illness

- [SUMMARIZE/READ CONTENT]

Slide 83: Lessons from Chronic Care

- [SUMMARIZE/READ CONTENT]
- *Yellow words are most important points in this slide*
Slide 84: Summary

- [SUMMARIZE/READ CONTENT]
- Yellow words are most important points in this slide

Slide 85: Agenda (6)

- Yellow bullet is the current agenda item
- Check-in with participants to see if they have any questions.
1. Explain that drug classifications provide a useful reference tool for approximating relative drug effects, possible harms, and potential withdrawal features.

2. Explain that there are limitations to classifications and that classifications are intended as a general guide only, as variations in effects and intensity may occur for drugs within the same class, e.g., although ecstasy produces similar effects to amphetamines, it is not as intense and may have additional hallucinogenic effects for some people.

3. Explain that for the purposes of this training, cannabis has been placed in the central nervous system (CNS) depressants category because of its primary effects as a CNS depressant, and in the hallucinogens category because at high doses, cannabis may produce hallucinogenic effects. Opioids have been classified as CNS depressants as a result of their primary effect on the CNS.
Slide 86: Classifying Psychoactive Drugs

GENERAL NOTES FOR TRAINERS

Abbreviations in slide

- DMT: N,N-dimethyltryptamine
- PCP: phencyclidine
- MDMA: 3,4 methylenedioxymethamphetamine

REFERENCE


Slide 87: Alcohol

- Now we’re going to talk about alcohol.

IMAGE CREDIT

Purchased image.
Alcohol: Basic facts

- **Description**: Alcohol or ethyl alcohol (ethanol) is present in varying amounts in beer, wine, and liquors
- **Route of administration**: Oral
- **Acute Effects**: Sedation, euphoria, lower heart rate and respiration, slowed reaction time, impaired coordination, coma, death

Alcohol: More Basic facts

**Withdrawal Symptoms:**
- Tremors, chills
- Cramps
- Hallucinations
- Convulsions
- Delirium tremens
- Death

**GENERAL NOTES FOR TRAINERS**

Delirium tremens is a medical emergency associated with untreated alcohol withdrawal. It occurs 3-14 days after drinking is stopped. Delirium tremens include agitation, restlessness, gross tremor, disorientation, fluid and electrolyte imbalance, sweating and high fevers, visual hallucinations, and paranoia. The prevalence is < 5% of patients, and it may lead to death.
Slide 89: Alcohol: More Basic facts

REFERENCE

Slide 90: Long-term effects of alcohol use

- [READ THE BULLETED LIST ON SLIDE]

- Point to the areas of the body that are affected by the use of alcohol when reading the content to participants.
Slide 90: Long-term effects of alcohol use

REFERENCE

Slide 91: Important terminology

- **Explain to your audience that you will review 3 important concepts related to addiction.**
- **Read these important terms, but do not explain them at this point.**
Slide 92: Psychological craving

- Explain to your audience what psychological craving is by reading the slide.

- Provide some examples. For instance, a woman who quit smoking years ago, but who still feels cravings when exposed to certain situations (friends who smoke, parties, coffee time).

IMAGE CREDIT

Purchased image.
For our purposes, there are four main neurotransmitters relevant to alcohol:

- Two processes occur simultaneously when alcohol is consumed. One process acts on the naturally occurring opioids in the brain (which deaden pain and cause feelings of euphoria), and dopamine (which makes things feel good). The other process operates on glutamate (the excitatory neurotransmitter that wakes you up) and GABA (the inhibitory neurotransmitter that slows you down).
- Let’s look at each separately.

REFERENCE


IMAGE CREDITS

Purchased images.
Alcohol Neuronal Activity #1: The Party

- In the first process, a person drinks alcohol.
- This causes the release of the endogenous, or naturally occurring, opioids to be released in the pleasure centers of the brain.
- This in turn, caused the release of dopamine.
- Since dopamine makes the person feel good, drinking is reinforced and this increases the likelihood of repeated use.
- As we will see later, two medications used to treat alcohol directly addresses these pleasurable effects of alcohol.
Tolerance

- a condition of cellular adaptation to a pharmacologically active substance so that increasingly larger doses are required to produce the same physiologic or psychological effect obtained earlier with smaller doses.
At the same time...Alcohol Neuronal Activity #2

- GABA is increased, slowing the brain down
- Over time, the brain reacts to the over-abundance of GABA by creating more receptors for Glutamate—increasing the effect of Glutamate, energizing the system and restoring balance

Slide 96: At the same time...Alcohol Neuronal Activity #2

- At the same time that alcohol is making the person feel good, a second process occurs.

When the person drinks, GABA (the inhibitory neurotransmitter) is released and this slows the brain down. This results in the symptoms of intoxication from alcohol like difficulty with coordination, drowsiness, slurring speech, etc.

- With repeated use, the brain attempts to correct for the overabundance of GABA by creating more receptors for Glutamate. This increases the effectiveness of glutamate, the system is energized and balance is restored.

IMAGE CREDITS

Purchased images.
• Now if the person drinks the same amount of alcohol, the intoxicating effects are not seen. It takes more alcohol in order to overcome the newly upregulated glutamate system. As this process occurs again and again, the individual must drink more and more to get the intoxicating effect. This process is known as tolerance.

IMAGE CREDITS
Purchased images.
Another Neuronal Activity

Slide 98: Another Neuronal Activity

- To Review...
- Normally, GABA and Glutamate are well balanced. Each takes precedence as required for normal functioning throughout the day.
- Repeated alcohol use overwhelms the glutamate system.
- The brain responds by making more receptors for glutamate, making it more effective.
- Once the brain has made this adaptation to the regular presence of alcohol, what do you think will happen if the person, abruptly stops drinking?
The answer is, of course, withdrawal.

Withdrawal is dangerous (potentially fatal) for the individual and the person should get medical support using medications (often with benzodiazepines and others) to assist with this process. Once the acute crisis is over, however, the glutamate system continues to be overactive since it takes time for the receptor levels to go back to normal. This “post acute withdrawal” often leads to the individual to feel anxious and agitated—a frequent cause of relapse.

One of the medications that we will explore in module 2 directly addresses this post acute withdrawal issues to assist the individual in maintaining abstinence.
Slide 100: Withdrawal Definition

- [ASK PARTICIPANTS] What are some examples of withdrawal symptoms?
- [READ THE BULLETED LIST ON THE SLIDE]

Slide 101: Withdrawal

- [READ THE SLIDE]

Slide 102: Opioids – What Are They?

- Point to the different pictures showing the various forms of opioids, including prescription drugs.

IMAGE CREDITS

Abbot Laboratories website.
Slide 103: What are Opioids?

- Heroin is a semisynthetic derived from morphine. Semisynthetic is derived by altering chemicals contained in opium.

REFERENCE


Slide 104: Opioids

- [SUMMARIZE/READ CONTENT]
Slide 105: Effects of Opioids

- [SUMMARIZE/READ CONTENT]

REFERENCE

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Slide 106: Effects of Opioids (continued)

- [SUMMARIZE/READ CONTENT]

REFERENCE
Slide 107: Opioids: Long-term Effects

- [SUMMARIZE/READ CONTENT]

REFERENCE


Slide 108: Opioids: Heroin

- [SUMMARIZE/READ CONTENT]

REFERENCE

(Notes for Slide 108, continued)

Slide 108: Opioids: Heroin

IMAGE CREDITS
NIDA website.

Slide 109: Opioids: Heroin Patterns of Use

REFERENCE
Slide 110: Opioids: Prescription Drugs

- Fentanyl (Duragesic®)
- Hydrocodone (Vicodin®)
- Oxycodone (OxyContin®)
- Oxymorphone (Opana®)
- Propoxyphene (Darvon®)
- Hydromorphone (Dilaudid®)
- Meperidine (Demerol®)
- Diphenoxylate (Lomotil®)

REFERENCE

Slide 111: Opioids: Basic facts

- [SUMMARIZE/READ CONTENT]

REFERENCE
Slide 112: What Causes Overdose?
• [SUMMARIZE/READ CONTENT]

REFERENCE

Slide 113: Who is at Risk?
• [SUMMARIZE/READ CONTENT]

REFERENCE
Slide 114: Sedative-Hypnotics

- [SUMMARIZE/READ CONTENT]

REFERENCE


IMAGE CREDITS

Purchased images.
Slide 115: Sedative-Hypnotics (continued)

- [SUMMARIZE/READ CONTENT]

REFERENCE


IMAGE CREDIT

Purchased image.

Slide 116: Sedative-Hypnotics

- [SUMMARIZE/READ CONTENT]

- Soma is muscle relaxant, metabolized to mebprobamate (Miltown)
Slide 116: Sedative-Hypnotics

- Non-benzo hypnotics bind to alpha 1 subunit of GABA-A receptor; thus sedating without anxiolytic or anticonvulsant properties

REFERENCE

Slide 117: Sedative-Hypnotic Effects

- [SUMMARIZE/READ CONTENT]

REFERENCE
(Notes for Slide 117, continued)

Slide 117: Sedative-Hypnotic Effects

IMAGE CREDIT
Purchased image.

Slide 118: Sedating Drugs and Overdose

• [SUMMARIZE/READ CONTENT]

REFERENCE
Slide 119: Other Sedative-Hypnotic Risks

- [SUMMARIZE/READ CONTENT]

REFERENCE


Slide 120: Cannabinoids

- [ASK PARTICIPANTS] Do you have any questions regarding sedative hypnotics.

- We are now transitioning to cannabinoids.

IMAGE CREDITS

Purchased images.
Marijuana is a dry, shredded green/brown mix of flowers, stems, seeds, and leaves of the hemp plant Cannabis sativa. It is usually smoked as a cigarette (joint, nail) or in a pipe (bong). It also is smoked in blunts, which are cigars that have been emptied of tobacco and refilled with marijuana, often in combination with another drug. It might also be mixed in food or brewed as a tea. As a more concentrated, resinous form it is called hashish, and as a sticky black liquid, it is called hash oil. Marijuana smoke has a pungent and distinctive, usually sweet-and-sour odor.

The main active chemical in marijuana is THC (delta-9-tetrahydrocannabinol). The membranes of certain nerve cells in the brain contain protein receptors that bind to THC. Once securely in place, THC sets off a series of cellular reactions.

Ask participants for examples of the acute effects of marijuana.
REFERENCES


THC works by acting on specialized cells called neurons in the brain (refer to illustration). Neurons do not touch each other, and the gap between them—called the synaptic space—needs to be bridged for messages to get from one neuron to the next. To get messages across the space, neurons release chemicals, or neurotransmitters. The receiving neuron contains special proteins called receptors that neurotransmitters will bind to, similar to the way a key fits into a lock. After a neurotransmitter has bound to a receptor, proteins called transporters or reuptake pumps will carry neurotransmitters back to the neurons that released them. The reason this process is important is that certain neurotransmitters and receptors are associated with specific emotional and functions. Any changes to these steps—the way neurotransmitters are released, the way receptors work, or the way transporters or reuptake pumps work—can have profound effects on sensation, perception, thought, mood, and behavior.
(Notes for Slide 122, continued)

Slide 122: Marijuana: Neurotransmitter System

- When people take drugs, these processes are altered, leading to changes in the way they feel and behave.

REFERENCE

Marijuana: How Does it Work?

- Marijuana gets its effects because it contains over 60 chemicals called cannabinoids. The main active chemical is a cannabinoid called delta-9-tetrahydorocannabinol, often referred to as THC. Cannabinoids trigger cannabinoid receptors, which are particularly dense in parts of the brain that affect pleasure, memory, thinking, concentration, and coordination.
- The effects of marijuana generally last 1-4 hours.

REFERENCE

Slide 124: Marijuana and the Brain

[ADVANCE SLIDE TO PLAY VIDEO]

VIDEO SOURCE

Slide 125: Cannabis: Basic facts (2)

[SUMMARIZE/READ CONTENT]

REFERENCE
Slide 126: Neurologic Impact of Marijuana in Adults

- [SUMMARIZE/READ CONTENT]

REFERENCE


Slide 127: Neurologic Impact of Marijuana in Adults (continued)

- [SUMMARIZE/READ CONTENT]

REFERENCE

• Explain that the negative short-term effects of marijuana may include problems with memory and learning; distorted perception; difficulty in thinking and problem solving; loss of coordination; and increased heart rate.

• Ask participants to provide examples of the acute effects of cannabis from their professional experience.

REFERENCE

Explain to participants that many people have believed that marijuana is not physically addictive. However, there appears to be a characteristic withdrawal profile indicating that physical dependence does actually occur.
Slide 130: Negative Effects on Behavior and Mental Health

- The purpose of this slide and the two slides that follow is to highlight that marijuana use can have serious negative effects. Introduce these slides by saying that people often say that marijuana is “harmless.” The information presented on these slides highlights that it is not at all a “harmless” drug.

- Marijuana use can have very negative effects on behavior and mental health. Since marijuana is a psychoactive drug, it causes significant impairment, just like alcohol and other drugs. This means that when experiencing a marijuana “high” people are impaired, both physically and mentally. It is unsafe to drive, operate heavy machinery, or do other things that require concentration and physical coordination when under the influence of marijuana. Long-term marijuana use has a negative impact on learning and memory.
(Notes for Slide 130, continued)

Slide 130: Negative Effects on Behavior and Mental Health

• Long-term marijuana use also causes amotivational syndrome, as it makes regular users less motivated to do things. Marijuana use is also associated with mental health problems and mental illness, particularly mood disorders. It is unclear if marijuana is what causes these problems, or if people who have mood disorders are more likely to use marijuana to self-medicate. Research also shows that heavy marijuana use is associated with serious mental illness, particularly among people who are at risk for serious mental illness because of family history.

• Additional Information for the Trainer(s)

• Serious mental illness differs from “mental illness” in general in that it lasts longer and is more disabling, often preventing people from working or functioning in their day to day lives. Among individuals who meet diagnostic criteria for marijuana abuse, 36% have had a mood disorder in their life, and 25% have had an anxiety disorder in their life.
Slide 130: Negative Effects on Behavior and Mental Health

- Among individuals who meet diagnostic criteria for marijuana dependence, 60.5% have had a mood disorder in their life, and 48.5% have had an anxiety disorder in their lifetime. Overall, marijuana dependence increases the odds of a co-occurring mood disorder by 6.5 times, and of an anxiety disorder by 4.6 times. Further, there is a significant gender difference with regard to major depression, with marijuana dependence increasing the odds for men by 4.6 times and 7.2 times for women.

REFERENCE

Slide 131: Long-term effects of cannabis use

- [SUMMARIZE/READ CONTENT]
- Point to the areas of the body that are affected by the use of this drug.

GENERAL NOTES FOR TRAINERS

Research findings on long-term marijuana abuse indicate some changes in the brain similar to those seen after long-term abuse of other major drugs. For example, cannabinoid (THC or synthetic forms of THC) withdrawal in chronically exposed animals leads to an increase in the activation of the stress-response system and changes in the activity of nerve cells containing dopamine. Dopamine neurons are involved in the regulation of motivation and reward, and are directly or indirectly affected by all drugs of abuse.

REFERENCE

Butane Hash Oil

- **Description:** a resinous matrix of cannabinoids obtained from the cannabis plant by solvent extraction.
- **Hash oil is the most potent of these main cannabis products, which are herb (marijuana), resin (hashish), and oil (hash oil).**
- **Dabs, honey oil, shatter, wax, or earwax**
- **Route of administration:** smoking, vaporization, orally or topically. Typically, it is vaporized in what is known as a "rig," a small water pipe designed for hash oil vaporization.

REFERENCE


IMAGE CREDIT

Purchased image.
Butane Has Oil (continued)

- Very little smell, either in its solid form or when vaporized
- Portable
- Intense effects with small amounts

REFERENCE


IMAGE CREDIT

Purchased image.
Slide 134: Synthetic Drugs

- This slide summarizes the dilemma we are faced with because these drugs have many different “names” that are given to mask the fact they are chemical substances that have been created to produce some sort of a “high.” Although the chemical composition of some of them is known, the rogue chemists producing them are constantly changing the formulations so they can stay ahead of the latest federal and state legal definitions and laws to avoid prosecution.

- Synthetic cannabinoids in herbal incense products were first detected in the United States in November 2008, by the Drug Enforcement Administration’s (DEA) forensic laboratory. These products were first encountered by U.S. Customs and Border Protection. Spice and Bath Salts are advertised as being “all natural,” safe to use, and legal but, in fact, they are none of those things. The packages often say “not for human consumption,” “for novelty use,” or “use as directed” (but without any directions for use on the package).
Slide 134: Synthetic Drugs

- The colorful and professional packaging and wording often changes as the laws are amended. In some jurisdictions, depending on how the laws are written, the prosecutor must prove the person intended to use the product (not just possess it), which makes it even more difficult to reduce availability of these substances.

REFERENCE


IMAGE CREDIT

Purchased images.
Slide 135: Spice vs. “Spice”

- Spices that we cook with have nothing to do with Spice the drug.
- Spice the drug are synthetic chemicals that are highly toxic and dangerous.
- Calling them Spice is a marketing strategy.

Slide 136: Synthetic Cannabinoids (a.k.a. Spice)

- Spice, sometimes known as an “herbal marijuana alternative,” looks like plant material or potpourri and is similarly coated in chemicals that mimic the effects of marijuana when it is smoked or steeped for a hot drink. These synthetic drugs are attractive to users because the chemicals used to create them defy detection in traditional drug tests. Labels on Spice products often claim that they contain “natural” psychoactive material taken from a variety of plants.
• Spice products do contain dried plant material, but chemical analyses show that their active ingredients are synthetic (or designer) cannabinoid compounds.

• Spice and K2 is known by a variety of brand names, including: Zohai, Genie, K3, Bliss, Nice, Black Mamba, Incense, Yucatan Fire, Skunk, Smoke, ChillX, Highdi’s Almdróhner, Sence, Earth Impact, Gorillaz, Galaxy Gold, Space Truckin, Solar Flare, Moon Rocks, Blue Lotus, Aroma, Scope, Sky, OG, Potpourri, Bombay Blue, and even fake weed. Spice is sold in colorful three-ounce plastic pouches decorated with psychedelic designs. Experts point out that due to the variation in chemical additives used in Spice, K2, and other synthetics, users don’t know exactly what they’re getting in each packet and the effects can therefore be unpredictable. (SOURCE: Logan et al., 2012, *Journal of Forensic Sciences*). According to the U.S. Drug Testing Laboratories (2011), over 250 synthetic cannabis compounds have been identified to date.
Slide 136: Synthetic Cannabinoids (a.k.a. Spice)

REFERENCE

IMAGE CREDIT
Purchased image.

Slide 137: Synthetic Cannabinoids (Spice)

- Manufacturers of Spice products attempt to evade legal restrictions by substituting different chemicals in their mixtures. The DEA continues to monitor the situation and evaluate the need for updating the list of banned cannabinoids. The five banned active chemicals are JWH-018, JWH-073, JWH-200, CP-47, 497, and CP-47, 497-C8.
Slide 137: Synthetic Cannabinoids (Spice)

- Most states have also banned the products, but the list of banned substances varies by state and the states keep revising the lists to try to control new products that are developed to get around the current laws. Toxicology laboratories are developing tests for these drugs, but as of September 2012, only 17 of all the synthetic cannabis variations can be identified in urine tests developed by one lab and most of the blood and oral fluid tests only identify 12.

REFERENCE

Many factors are associated with the recent popularity of Spice products, especially among younger users. Spice smokers find the effects similar to those of marijuana; Spice is often referred to as a “legal high;” regulatory mechanisms are difficult to enforce when products are available on the Internet; and Spice is marketed as a natural herb and intuitive language on packaging makes it attractive to young and drug-naïve individuals.

REFERENCE

Short-Term Effects of Synthetic Cannabinoids

- Loss of control
- Lack of pain response
- Increased agitation
- Pale skin
- Seizures
- Vomiting
- Profuse sweating

In addition to physical signs of use, users may experience severe paranoia, delusions, and hallucinations.

Slide 139: Short-Term Effects of Synthetic Cannabinoids

- Short term effects include loss of control, lack of pain response, increased agitation, pale skin, seizures, vomiting, profuse sweating, uncontrolled spastic body movements, elevated blood pressure, heart rate and palpitations. In addition to physical signs of use, users may experience severe paranoia, delusions, hallucinations and increased agitation.

REFERENCE

Slide 140: Cannabis vs. Cannabinoids: Effects Seen in Clinical Cases

- Due to the paucity of medical literature and research regarding synthetic cannabinoids, the clinical effects are primarily known from case reports and case series. Effects of synthetic cannabinoids are similar to those produced by marijuana, such as: elevated mood, relaxation, and altered perception. In some cases, the effects are even stronger than those reported for marijuana. Some users have reported psychotic effects, including extreme anxiety, paranoia, and hallucinations.

- So far, there have been no scientific studies of Spice’s effects on the human brain, but it is known that the cannabinoid compounds found in Spice products act on the same cell receptors as THC, the primary psychoactive component of marijuana.

- Some compounds found in Spice, however, bind more strongly to those receptors, which could lead to a much more powerful and unpredictable effect.
Because the chemical composition of many products sold as Spice is unknown, it is likely that some varieties also contain substances that could cause dramatically different effects than the user might expect.

Spice abusers who have been taken to Poison Control Centers report symptoms that include rapid heart rate, vomiting, agitation, confusion, and hallucinations. Spice can also raise blood pressure and cause reduced blood supply to the heart (myocardial ischemia), and in a few cases it has been associated with heart attacks. Regular users may experience withdrawal and addiction symptoms. Based on the more unpleasant effects of cannabinoid intoxication, one would wonder why anyone would voluntarily ingest any of these substances, except perhaps to avoid testing positive for cannabis (1-5).
GENERAL NOTES FOR TRAINERS

Tachycardia = heart rate that exceeds the normal range

Hypokalemia = a lower than normal amount of potassium in the blood

REFERENCES


REFERENCES, continued


Slide 141: Bath Salts vs. “Bath Salts”

- Bath salts that are used as a soothing addition to a bath have nothing to do with “bath salts” the drug.
- Bath salts the drug are synthetic chemicals that are highly toxic and dangerous.
- Calling them bath salts is another marketing strategy.

IMAGE CREDITS

Purchased images.
Slide 142: Synthetic Cathinones: “Bath Salts”

- Synthetic cathinones known as Bath Salts are one of the latest additions to a growing list of substances young people can use to get high. Bath Salts is a powder laced with a cocktail of chemicals that comes in either capsules or in loose form. A user can either swallow the capsule whole or use the powder, mixed with liquid, and injected. Sometimes it is snorted directly up the nose. It is said to replicate a cocaine or ecstasy high (1).

- Synthetic cathinones are related to the parent compound cathinone, one of the psychoactive principals in khat. The synthetic powder is sold legally online and in drug paraphernalia stores under a variety of names, such as Ivory Wave, Purple Wave, Red Dove, Blue Silk, Zoom, Bloom, Cloud Nine, Ocean Snow, Lunar Wave, Vanilla Sky, White Lightning, Scarface, and Hurricane Charlie.
Slide 142: Synthetic Cathinones: “Bath Salts”

- Because these products are relatively new to the drug abuse scene, knowledge about their precise chemical composition and short- and long-term effects is limited, yet the known information warrants a proactive stance to understand and minimize any potential dangers to the public’s health.

- These products often contain various amphetamine-like chemicals, such as methylenedioxypyrovalerone (MPDV), mephedrone and pyrovalerone. These drugs are typically administered orally, by inhalation, or by injection, with the worst outcomes apparently associated with snorting or intravenous administration. Mephedrone is of particular concern because, according to the United Kingdom experience, it presents a high risk for overdose. These chemicals act in the brain like stimulant drugs (indeed they are sometimes touted as cocaine substitutes); thus they present a high abuse and addiction liability.
Consistent with this notion, these products have been reported to trigger intense cravings not unlike those experienced by methamphetamine users, and clinical reports from other countries appear to corroborate their addictiveness. They can also confer a high risk for other medical adverse effects. Some of these may be linked to the fact that, beyond their known psychoactive ingredients, the contents of "bath salts" are largely unknown, which makes the practice of abusing them, by any route, that much more dangerous (2).

**GENERAL NOTES FOR TRAINERS**

The list of synthetic cathinones is long: butylone, dimethylcathinone, ethcathinone, ethylone, 3- and 4-flouromethcathinone, methadone, mephedrone, methlenedioxyxpyrovalerone (MDPV), methylone and pyrovalerone, among others. Bupropion is the only cathinone derivative that has a medical indication in the U.S. and Europe. The first synthetic cathinone, methcathinone, was produced in 1928.
Slide 142: Synthetic Cathinones: “Bath Salts”

REFERENCES


Slide 143: Bath Salts: Acute Effects

REFERENCES

Slide 144: Stimulants

• Tell participants that you will now discuss stimulants, and point out the photographs of methamphetamine and cocaine.

IMAGE CREDITS
Drug Enforcement Administration website.

Slide 145: Types of Stimulants:
Methamphetamine

• [SUMMARIZE/READ CONTENT]

Methamphetamine

Description:
• A synthetic drug (ATS) that affects the central nervous system. It takes the form of a white, odorless, bitter-tasting crystalline powder that easily dissolves in water or alcohol

Route of administration:
• Smoked, injected, snorted, or administered by mouth or rectum

• Cocaine is a strong central nervous system stimulant that interferes with the re-absorption process of dopamine, a chemical messenger associated with pleasure and movement. The build-up of dopamine causes continuous stimulation of receiving neurons, which is associated with the euphoria commonly reported by cocaine users.
Slide 145: Types of Stimulants: Methamphetamine

- Methamphetamine has strong effects on the central nervous system, even when small amounts of the drug are used.

REFERENCE

Methamphetamine (continued)

- Methamphetamine is an addictive stimulant drug that strongly activates certain systems in the brain. Methamphetamine is chemically related to amphetamine, but the effects of methamphetamine on the central nervous system are greater. Both drugs have some limited therapeutic uses, primarily in the treatment of obesity.

- Methamphetamine is made in illegal laboratories and has a high potential for abuse and addiction.

- Methamphetamine is taken orally or intranasally (snorting the powder), by intravenous injection, and by smoking. Immediately after smoking or intravenous injection, the methamphetamine user experiences an intense sensation, called a “rush” or “flash,” that lasts only a few minutes and is described as extremely pleasurable. Oral or intranasal use produces euphoria—a high, but not a rush.
Slide 146: Types of Stimulants: Methamphetamine (continued)

- Users may become addicted quickly and use the drug with increasing frequency and in increasing doses.

GENERAL NOTES FOR TRAINERS

Abbreviations in slide: ADD – Attention Deficit Disorder; ADHD-Attention Deficit and Hyperactivity Disorder

REFERENCE


IMAGE CREDITS

Drug Enforcement Administration website.
Methamphetamine: Patterns of Use

- Smoking or injecting causes an immediate, intense “rush” which lasts a few minutes.
- Snorting or oral ingestion produces euphoria—a high, but not an intense rush.
  - Snorting produces effects within 3 to 5 minutes.
  - Oral ingestion produces effects within 15 to 30 minutes.
- Often abused in “binge & crash” pattern.
  - “Binge,” foregoing food and sleep while continuing to take the drug for up to several days.

REFERENCE

Methamphetamine: Acute effects

- Increased attention and decreased fatigue
- Increased activity and wakefulness
- Decreased appetite
- Euphoria and rush
- Increased respiration
- Rapid/irregular heartbeat
- Hyperthermia

[SUMMARIZE/READ CONTENT]

- Explain that effects in the central nervous system (CNS) of methamphetamine include increased wakefulness, increased physical activity, decreased appetite, increased respiration, hyperthermia, and euphoria. Other CNS effects include irritability, insomnia, confusion, tremors, convulsions, anxiety, paranoia, and aggressiveness. Hyperthermia and convulsions can result in death.

REFERENCE

Methamphetamine: Long-term Effects

• Psychosis, including:
  – paranoia
  – hallucinations
  – repetitive motor activity
• Changes in brain structure and function
• Deficits in thinking and motor skills
• Increased distractibility
• Memory loss
• Aggressive or violent behavior
• Mood disturbances
• Severe dental problems
• Weight loss

REFERENCE

Cocaine is a powerfully addictive stimulant drug. The powdered hydrochloride salt form of cocaine can be snorted or dissolved in water and injected.

“Crack” is cocaine that has not been neutralized by an acid to make the hydrochloride salt. This form of cocaine comes in a rock crystal that can be heated and its vapours smoked. The term “crack” refers to the crackling sound that is made when it is heated.

REFERENCE


IMAGE CREDITS

- Drug Enforcement Administration website.
Types of Stimulants
Cocaine: Basic facts

**Description:**
A plant-derived compound that increases alertness and arousal by stimulating the central nervous system.

**Route of administration:**
Smoked, injected, snorted, or administered by mouth or rectum.

**[SUMMARIZE/READ CONTENT]**

- Cocaine is a strong central nervous system stimulant that interferes with the re-absorption process of dopamine, a chemical messenger associated with pleasure and movement. The build-up of dopamine causes continuous stimulation of receiving neurons, which is associated with the euphoria commonly reported by cocaine users.

- Methamphetamine has strong effects on the central nervous system, even when small amounts of the drug are used.

**REFERENCE**
Cocaine: Patterns of Use

- Injecting or smoking cocaine delivers the drug rapidly into the bloodstream and brain, producing a quicker and stronger but shorter-lasting high than snorting.
  - The high from snorting cocaine may last 15 to 30 minutes.
  - The high from smoking may last 5 to 10 minutes.
- Often used in a binge pattern—taking the drug repeatedly within a relatively short period of time, at increasingly higher doses.
- Stays in system for 1-2 days.

REFERENCE


Cocaine: Acute effects

- Constricted blood vessels
- Dilated pupils
- Increased body temperature, HR, and blood pressure
- Bizarre, erratic, and violent behavior
- Restlessness
- Irritability
- Anxiety, panic
- Tremors, vertigo, and muscle twitches
- Cardiovascular effects (heart rhythm and heart attacks)
- Neurological effects (stroke, seizures, headaches, coma)

REFERENCE


The physical effects of cocaine use include constricted blood vessels, dilated pupils, and increased temperature, heart rate, and blood pressure. The duration of cocaine’s immediate euphoric effects, which include hyperstimulation, reduced fatigue, and mental alertness, depends on the route of administration. The faster the absorption, the more intense the high.
Slide 153: Cocaine Acute effects

- On the other hand, the faster the absorption, the shorter the duration of action. The high from snorting may last 15 to 30 minutes, while that from smoking may last 5 to 10 minutes. Increased use can reduce the period of time a user feels high and increases the risk of addiction.

REFERENCE

Slide 154: Cocaine: Long-term Effects

- [SUMMARIZE/READ CONTENT]
Slide 154: Cocaine: Long-term Effects

REFERENCE


Slide 155: Types of Stimulants;

Prescription Stimulants

- [SUMMARIZE/READ CONTENT]
- Common amphetamines include Dexedrine (d-amphetamine), methamphetamine, Ritalin, and Adderall (dl-amphetamine).

REFERENCE

Slide 156: Street Names for Stimulants

- [SUMMARIZE/READ CONTENT]

REFERENCE


Slide 157: Short-Term Effects

- [SUMMARIZE/READ CONTENT]

- The short-term effects plus ease of manufacture and less stigma are what makes meth appealing to use among different groups. Also used to self-medicate depression/ADHD.

- Risk of HIV/Hepatitis B&C transmission is increased with increased libido/impulsivity.
REFERENCES


Methylphenidate (Ritalin, Concerta)

- When used to treat ADHD, patients may experience increased attention, decreased impulsiveness, and decreased hyperactivity.
- Milder stimulant that works by affecting the levels of neurotransmitters in the nervous system.
- May also be used to augment treatment of depression in some cases.

REFERENCE


IMAGE CREDIT

Purchased image.
Amphetamine/dextroamphetamine (Adderall)

- Used for attention deficit hyperactivity disorder (ADHD)
- Increases ability to concentrate and reduces hyperactivity
- May also be used to treat certain sleep disorders (narcolepsy)
- Original form taken 2-3 x/day
- Adderall XR taken once/day

REFERENCE


IMAGE CREDIT

Purchased image.
Lisdexamphetamine (Vyvanse)

- Dextroamphetamine prodrug
- Enters the body in an inactive form
- As digested, slowly converts into active form; effects can last for up to 14 hours
- Lower abuse potential
- Also approved for binge eating disorder
- Capsule dose 30-70 mg per day

REFERENCE


IMAGE CREDIT

Purchased image.
Slide 161: Medical Risks

- Norepinephrine release causes constriction of blood vessels, elevated blood pressure and rapid heart rate
- Increased activity levels
- Elevated body temperatures
- Increased risk of seizures
- Potentially fatal arrhythmias, heart attack, or stroke

REFERENCE

IMAGE CREDIT
Purchased image.

Slide 162: Psychiatric Symptoms

- Psychiatric symptoms associated with use of larger doses of amphetamines include depression, anxiety, psychosis, and suicidal ideation
- Symptoms may depend on differences in sensitivity, frequency and quantity of use, and method of administration
- Abstinence syndrome may occur (dysphoria, anhedonia, irritability, insomnia/hypersomnia, anxiety, low energy)

- Users may appear manic (grandiosity, psychomotor agitation)
Slide 162: Psychiatric Symptoms

- Stimulant users: up to 60% with affective d/os, 40-50% with anxiety disorders

REFERENCE


Slide 163: Stimulants: Basic facts

- [SUMMARIZE/READ CONTENT]

REFERENCE

Slide 164: Long-term effects of stimulants

- [SUMMARIZE/READ CONTENT]

- Explain the long-term effects of cocaine: Regardless of how cocaine is used or how frequently, a user can experience acute cardiovascular or cerebrovascular emergencies, such as a heart attack or stroke, which could result in sudden death. Cocaine-related deaths are often a result of cardiac arrest or seizure followed by respiratory arrest. Use of cocaine in a binge, during which the drug is taken repeatedly and at increasingly high doses, may lead to a state of increasing irritability, restlessness, and paranoia. This can result in a period of full-blown paranoid psychosis, in which the user loses touch with reality and experiences auditory hallucinations.
Other complications associated with cocaine use include disturbances in heart rhythm and heart attacks, chest pain, respiratory failure, strokes, seizures, and headaches, as well as gastrointestinal complications such as abdominal pain and nausea. Because cocaine has a tendency to decrease appetite, many chronic users can become malnourished.

• Explain the long-term effects of methamphetamine:
  Methamphetamine releases high levels of the neurotransmitter dopamine, which stimulates brain cells, enhancing mood and body movement. It also appears to have a neurotoxic effect, damaging brain cells that contain dopamine as well as serotonin, another neurotransmitter. Over time, methamphetamine appears to cause reduced levels of dopamine, which can result in symptoms like those of Parkinson’s disease, a severe movement disorder.
Slide 164: Long-term effects of stimulants

REFERENCES


Slide 165: Tobacco

- Now we’re going to transition to talking about tobacco.

REFERENCE


IMAGE CREDITS

Purchased images.
In addition to nicotine, cigarette smoke is primarily composed of a dozen gases (mainly carbon monoxide) and tar. The tar in a cigarette, which varies from about 15 mg for a regular cigarette to 7 mg in a low-tar cigarette, exposes the user to an increased risk of lung cancer, emphysema, and bronchial disorders.

Nicotine is absorbed readily from tobacco smoke in the lungs, and it does not matter whether the tobacco smoke is from cigarettes, cigars, or pipes. Nicotine also is absorbed readily when tobacco is chewed. With regular use of tobacco, levels of nicotine accumulate in the body during the day and persist overnight. Thus, daily smokers or chewers are exposed to the effects of nicotine for 24 hours each day. Adolescents who chew tobacco are more likely than nonusers to eventually become cigarette smokers.
Slide 166: Tobacco: Basic facts (1)

- Nicotine provides an almost immediate “kick” because it causes a discharge of epinephrine from the adrenal cortex. This stimulates the central nervous system and endocrine glands, which causes a sudden release of glucose. Stimulation is then followed by depression and fatigue, leading the user to seek more nicotine.

- *Ask participants for examples of the acute effects of tobacco.*

REFERENCE

Explain that research has found that when chronic smokers are deprived of cigarettes for 24 hours, they have increased anger, hostility, and aggression, and loss of social cooperation. Persons suffering from withdrawal also take longer to regain emotional equilibrium following stress. During periods of abstinence and/or craving, smokers have shown impairment across a wide range of psychomotor and cognitive functions, such as language comprehension.

REFERENCE

Slide 167: Tobacco: Basic facts (2)

(Notes for Slide 167, continued)

Long-term effects of tobacco use

• Aneurysm
• Cataracts
• Cancer (lung and other types)
• Chronic bronchitis
• Emphysema
• Asthma symptoms
• Obstructive pulmonary diseases
• Heart disease (stroke, heart attack)
• Vascular disease
• Harm to a fetus during pregnancy, low weight at birth
• Death

Slide 168: Long-term effects of tobacco use

• [SUMMARIZE/READ CONTENT]
• Point to the areas of the body that are affected by the use of tobacco when reading the content to your audience.

• Explain that some of the long-term effects of tobacco use are an increased risk of lung cancer, emphysema, and bronchial disorders. The carbon monoxide in tobacco smoke increases the chance of cardiovascular diseases. The U.S. Environmental Protection Agency has concluded that secondhand smoke causes lung cancer in adults and greatly increases the risk of respiratory illnesses in children and sudden infant death.
Emphysema is a lung disease in which tissue deterioration results in increased air retention and reduced exchange of gases. The result is breathing difficulties and shortness of breath.

REFERENCE
The main psychoactive ingredients in khat are cathine and cathinone, chemicals that are structurally similar to, but less potent than amphetamine, yet result in similar psychomotor stimulant effects. Chewing khat leaves can induce a state of euphoria and elation as well as feelings of increased alertness and arousal. The user can also experience an increase in blood pressure and heart rate. The effects begin to subside after about 90 minutes to 3 hours, but can last 24 hours. At the end of a khat session, the user may experience a depressive mood, irritability, loss of appetite, and difficulty sleeping. Chewing the leaves and twigs of the plant produces amphetamine-like euphoric effects. In 2006, there were 10 million khat users worldwide.
Slide 169: Khat

(Notes for Slide 169, continued)

REFERENCE


IMAGE CREDIT

Purchased image.
Slide 170: Hallucinogens

**REFERENCES**


**IMAGE CREDITS**

Purchased images.

Slide 171: Hallucinogens: Basic facts

**[SUMMARIZE/READ CONTENT]**
Slide 171: Hallucinogens: Basic facts

REFERENCE


Slide 172: Hallucinogens: More Basic facts

REFERENCE

Slide 173: Hallucinogens: Acute effects

- [SUMMARIZE/READ CONTENT]

REFERENCE


Slide 174: Hallucinogens: Acute effects (continued)

- [SUMMARIZE/READ CONTENT]

REFERENCE

Slide 175: Hallucinogens: Long-term Effects

- Flashbacks, or recurrences of certain aspects of the drug experience
- Risk of psychiatric illness
- Impaired memory
- Tolerance

REFERENCE


Slide 176: Inhalants

- Deliberate inhalation of fumes, vapors or gases to “get high”
- “Sniffing” or “Huffing”
- More than 1,400 household products
- Not a specific drug—but a method of drug delivery
- Whippets, Poppers

REFERENCE

More on Inhalants

Route of administration:
- Huffing (breathe them in through the nose or mouth)
- Sniff or snort fumes from a container or dispenser (such as a glue bottle or a marking pen)
- Spray aerosols (such as computer cleaning dusters) directly into nose or mouth
- Place a chemical-soaked rag in mouth
- Inhale fumes from a balloon or a plastic or paper bag

REFERENCE

Commonly Abused Inhalants - Solvents

- Volatile solvents—liquids that vaporize at room temperature
  - Industrial or household products, including paint thinners or removers, degreasers, dry-cleaning fluids, gasoline, and lighter fluid
  - Art or office supply solvents, including correction fluids, felt-tip marker fluid, electronic contact cleaners, and glue

REFERENCE
Slide 179: Commonly Abused Inhalants – Aerosols

- Aerosols—sprays that contain propellants and solvents
  - Household aerosol propellants in items such as spray paints, hair or deodorant sprays, fabric protector sprays, aerosol computer cleaning products, and vegetable oil sprays

REFERENCE

Slide 180, Commonly Abused Inhalants – Gases

- Gases—found in household or commercial products and used as medical anesthetics
  - Household or commercial products, including butane lighters and propane tanks, whipped cream aerosols or dispensers (whippets), and refrigerant gases
  - Medical anesthetics, such as ether, chloroform, halothane, and nitrous oxide ("laughing gas")

REFERENCE
Slide 181: Commonly Abused Inhalants – Nitrites

- Nitrites—used primarily as sexual enhancers
  - Organic nitrites are volatiles that include cyclohexyl, butyl, and amyl nitrites, commonly known as “peppers.”
  - Amyl nitrite is still used in certain diagnostic medical procedures.
  - When marketed for illicit use, organic nitrites are often sold in small brown bottles labeled as “video head cleaner,” “room odorizer,” “leather cleaner,” or “liquid aroma.”

REFERENCE

Slide 182: Inhalants Patterns of Use

- **SUMMARIZE/READ CONTENT**

  - “Sudden sniffing death” sniffing highly concentrated amounts of the chemicals in solvents or aerosol sprays can directly cause heart failure within minutes.
  - High concentrations of inhalants may also cause death from suffocation, especially when inhaled from a paper or plastic bag or in a closed area.

**REFERENCE**

Inhalants: Acute Effects

- Headache
- Muscle weakness
- Abdominal pain
- Severe mood swings
- Violent behavior
- Slurred speech
- Numbness, tingling in hands and feet
- Visual disturbances

Inhalants: Acute Effects (continued)

- Fatigue
- Lack of coordination
- Apathy
- Impaired judgment
- Dizziness
- Lethargy
- Stupor
- Loss of consciousness
- Limb spasms

REFERENCE


Slide 184: Inhalants: Acute Effects (continued)

- [SUMMARIZE/READ CONTENT]

REFERENCE

Slide 185: Inhalants Long-Term Effects

- [SUMMARIZE/READ CONTENT]

REFERENCE


Slide 186: Questions

- *Ask participants if they have any final questions.*
Acknowledgements

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