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What is on a 5 panel drug test

When a person consumes drugs, it will eventually break down inside the body and release byproducts called metabolites. These byproducts are excreted in the body through the form of urine. This process is highly predictable, and once detected in the stat cup drug test, it can conclude that the patient has recently taken the drug. The most common sample type that 5-panel and 10-panel drug tests collect is urine. Compared to other samples, urine tests are the most affordable, painless, non-invasive, and have a longer time to detect drugs. What Illegal Drugs Does It Screen? The main difference between 5-panel and 10-panel drug tests is the number of drugs they test for. Therefore, the 5-panel drug test can detect the five most illicit substances, while the 10-panel spot ten different illegal substances in the patient's body. The 5-panel drug test screens the following illicit substances: Amphetamines Cocaine Metabolites Marijuana Metabolites (THC) Opiates Phencyclidine (PCP) The 10-panel drug test has a slightly more extensive range than the 5-panel. The following are the controlled substances that a 10-panel drug test can detect. Amphetamines Cocaine Metabolites Marijuana Metabolites (THC) Opiates Phencyclidine (PCP) Barbiturates Benzodiazepines Methadone Methaqualone Propoxyphene Who Takes the Test? The 5-panel drug test is a more common type of difficulty than the 10-panel since it covers the most usually abused drugs. It depends on the patient's employment industry that could determine the stat swab oral drug test type. 5-panel drug tests are usually used by private employers and government agencies like the Department of Transportation. Therefore, the 5-panel drug test takers are generally the potential hire and the current employee of these industries. On the other hand, a 10-panel drug test is commonly used by employers who want to detect a wider variety of illicit substances in their workforce. Professionals like law enforcement officials, medical professionals, federal employees, state employees, or local government workers are the usual takers of a 10-panel drug test. Drug Detection Windows Several factors could determine the time duration the drug can be detected. The drug itself, dose, type of sample, and individual metabolism can affect drug detection time. And since the drugs can be undetectable after a specific amount of time, the stat cup drug tests and samples are often ordered randomly. The following are the approximate detection time for each controlled substance. Amphetamines: one to two days. Cocaine Metabolites: one to four days. Marijuana Metabolites (THC): one day to five weeks. Opiates: one to four days. Phencyclidine: two to eight days. Barbiturates: two to ten days. Benzodiazepines: one to six weeks. Methadone: one to seven days. Methaqualone: two weeks Propoxyphene: six hours to two days. Drug Test Results All stat swab oral drug tests could have three outcomes: positive, negative, or inconclusive/invalid results. It will take about one to two business days to come back from the analysis. And if the patient tested positive for the controlled substance, it will take another two to four days for confirmation. Drug testing is often a routine step in the pre-employment screening process — and for a good reason. Drug use on the job can cause accidents, conflicts, and liability issues that could be avoided if companies are able to identify drug use early on. A 5-panel drug test is a type of drug test employers can use to detect prescription or illicit drugs. Read on to learn how the test works, what it screens for, and drug testing laws that candidates and employers should know. A 5-panel drug test screens for five types of illegal substances and commonly abused prescription drugs. Typically, a urine sample is collected for the 5-panel drug screening, but sweat, hair, blood, or saliva may also be tested. A 5-panel drug test may be used as part of a pre-employment background check, such as for positions regulated by the Department of Transportation (DOT). However, 5-panel drug tests are not typically used for other regulated industries, such as law enforcement, healthcare, or government, where more in-depth screenings are often required. Employers that use 5-panel drug testing can choose to work with a qualified background check provider, like GoodHire, for background checks. This not only helps ensure efficient and accurate testing, but can help employers stay compliant with all federal, state, and local drug testing laws. A 5-panel drug test identifies metabolites in an individual's system, which are chemicals that remain after your body metabolizes certain substances, such as food or drugs. They typically screen for metabolites linked to the following drug types: Amphetamines, such as ecstasy, crystal meth, or RitalinPhencyclidine, also called PCP or angel dustCocaine and crack cocaineOpiates, such heroin, oxycodone, or hydrocodoneMarijuana Although 5-panel tests do not screen for the presence of alcohol, labs may offer an add-on to include alcohol detection. Before using a 5-panel drug test, employers in states where marijuana is decriminalized or legalized should be aware of any local regulations regulating testing for marijuana – especially with rapidly changing legislation. How far back a 5-panel drug test can go depends on the type of sample taken, the type of drug used, and the frequency of use. Some types of drugs may have a longer detection period when used frequently. For example, people that smoke marijuana occasionally may have no detectable THC in their system after only a few days. However, in heavy marijuana smokers, THC may be detectable in urine for several weeks. Alcohol can also have an effect on the metabolism of drugs and vary drug detection time frames. Below is an approximate breakdown of how long a drug may be detectable in a urine drug test. SubstanceDetection Time FrameAmphetamines48 hoursCocaine2-4 daysMarijuana2-3 days for light smokers; 5 days for moderate smokers; 10 days for daily smokers; up to 28 days for chronic smokersOpiates2-3 daysPhencyclidine~8 days, or up to 30 days for chronic users The following are estimated time frames that drugs may be detectable in blood, saliva, and hair: SubstanceBloodSalivaHairAmphetamines~12 hours24-48 hoursup to 90 daysCocaine1-2 daysup to 72 hoursup to 90 daysMarijuanaup to 2 weeksup to 24 hoursup to 90 daysOpiatesup to 24 hoursup to 4 daysup to 90 daysPhencyclidine1-4 hours1-3 daysup to 90 days The results from a 5-panel drug test are typically ready a few business days after the sample is received by the lab. The final report will show a positive, negative, or inconclusive result for each of the five substances. Here is a closer look at how to read 5-panel drug test results: Positive: If the result shows "positive", this means that metabolites in the sample exceed the test cutoff concentration, or the amount of a substance that needs to be present to trigger a positive result. It could also mean a "false positive," which can occur if you have chemicals in your system similar to what the test is designed to detect. For example, some over-the-counter drugs — such as Sudafed or Robitussin — could potentially cause a positive test result. Negative: A "negative" means that the test did not detect drug use or the substance detected is below the test cutoff level. Inconclusive: If the result shows "inconclusive," this means that a result was not found. This can happen if there was a testing error. It can also occur if the sample was watered down or there is a reason to believe the test was faulty. For example, this may happen if a candidate drinks a lot of water or uses another method to dilute their urine to try and pass the test. Before pre-employment test results are sent to an employer, they are typically reviewed by a Medical Review Officer (MRO) to confirm positive test results or ensure that it was not a false positive. MROs are licensed physicians who are trained and certified to review and validate lab test results. When results are questionable, the MRO may reach out to the candidate to discuss if they have taken any physician-directed prescription drugs or eaten any foods that could affect results. Or, they may order confirmatory testing to rule out a false positive. After the MRO has completed the review, employers are given the positive or negative test certification. At this time, the candidate can choose to dispute the findings or request further explanation of their test results. While federal law does not prohibit employer drug testing of candidates and existing employees, employers are still required to stay compliant with federal, state, and local fair hiring practices, which include drug testing regulations. Here is a closer look at the key laws employers may need to know: Title VII of the Civil Rights Act: Under this law, employers are required to ensure that drug tests are administered in a manner that does not discriminate against candidates and employees. The Act also prohibits employers from treating employees differently due to their race, sex, religion, or nationality, including singling out protected classes for drug testing. Americans with Disabilities Act (ADA): The ADA protects the rights of persons with disabilities. Under this Act, employers cannot enforce different policies for people recovering from drug abuse who are not currently using drugs. They also cannot punish individuals who have a history of drug abuse or those that have gone through a rehabilitation program. While the Act does not explicitly require employers to provide reasonable accommodation for legal medical cannabis users, more than 20 states currently prohibit discrimination against workers on the basis of their marijuana use. Fair Credit Reporting Act (FCRA): When employers use a consumer reporting agency (CRA), like GoodHire, to conduct drug screenings as part of a background check, the employer must follow certain regulations. These include providing notice of your intent to conduct a background check, receiving written consent from the candidate, and following the adverse action process should you choose not to hire a candidate due to the results of a drug test. Candidates also have the right to review and dispute errors under the FCRA.State and local laws: States and major cities may have their own drug testing regulations. For example, in some states, employers must make a conditional offer of employment before asking a candidate to take a drug test or may not use a candidate's refusal to submit to testing as a basis for not hiring. If you are unsure of the laws in your jurisdiction, consider seeking legal counsel before implementing a drug testing program. Marijuana laws: The legalization and decriminalization of marijuana may have an impact on drug testing guidelines, depending on your state. Some states have already barred employers from making employment decisions solely based on marijuana use. In New York City, for example, employers may not test for marijuana on pre-employment and current employee drug tests, with a few narrow exceptions depending on the nature of the role.. Philadelphia has a similar law in place for pre-employment testing as well. Employers that are unable to perform a 5-panel drug test due to marijuana laws may choose to consider running a 4-panel drug test instead.   A 5-panel drug test screens for commonly abused prescription medications and drugs, including amphetamines, phencyclidine, cocaine, opiates, and marijuana. Urine lab tests can detect the presence of some drugs from two to 28 days, depending on the substance and frequency of use. Hair samples can detect drugs for up to 90 days, while saliva and blood tests may only be able to detect the presence of drugs for just a few hours or up to a few days, depending on the substance. Urine testing is often the industry standard for conducting drug tests. However, blood, saliva, and hair may also be used for 5-panel drug tests. Yes. 5-panel drug tests are typically conducted using a candidate's urine sample, however sweat, hair, blood, or saliva may also be tested. No, a 5-panel drug test does not screen for or detect alcohol. However, if the test taker drinks alcohol, it could affect the results. Yes. A 5-panel drug test screen includes marijuana. When conducted using a urine lab test, a 5-panel drug test can detect marijuana for two to three days for light smokers; five days for moderate smokers; 10 days for daily smokers; and up to 28 days for chronic smokers. No. 5-panel drug tests do not test for steroids. They only test for the presence of amphetamines, phencyclidine, cocaine, opiates, and marijuana. Drug testing can help employers make informed hiring decisions, while promoting a safer work environment, and mitigating risk. Partnering with a professional employment screening provider, like GoodHire, can streamline the entire background check process, including drug testing, while supporting compliance with applicable federal, state, and local laws. GoodHire offers a suite of drug testing options, including a 5-panel test, with fast, accurate test results verified by a Medical Review Officer. Get started. The resources provided here are for educational purposes only and do not constitute legal advice. We advise you to consult your own counsel if you have legal questions related to your specific practices and compliance with applicable laws. Technical analysis of a biological specimen "Toxicology screen" and "tox screen" redirect here. For testing for toxic substances on organisms in general, see Toxicology testing. For other uses, see Drug testing (disambiguation). This article needs more reliable medical references for verification or relies too heavily on primary sources, specifically: detection periods seem to lack medical sources. Please review the contents of the article and add the appropriate references if you can. Unsourced or poorly sourced material may be challenged and removed. Find sources: "Drug test" – news · newspapers · books · scholar · JSTOR (March 2025) Medical diagnostic test Drug testMeSHD015813 A drug test (also often toxicology screen or tox screen) is a technical analysis of a biological specimen, for example urine, hair, blood, breath, sweat, or oral fluid/saliva—to determine the presence or absence of specified parent drugs or their metabolites. Major applications of drug testing include detection of the presence of performance enhancing steroids in sport, employers and parole/probation officers screening for drugs prohibited by law (such as cocaine, methamphetamine, and heroin) and police officers testing for the presence and concentration of alcohol (ethanol) in the blood commonly referred to as BAC (blood alcohol content). BAC tests are typically administered via a breathalyzer while urinalysis is used for the vast majority of drug testing in sports and the workplace. Numerous other methods with varying degrees of accuracy, sensitivity (detection threshold/cutoff), and detection periods exist. A drug test may also refer to a test that provides quantitative chemical analysis of an illegal drug, typically intended to help with responsible drug use.[1] The detection windows depend upon multiple factors: drug class, amount and frequency of use, metabolic rate, body mass, age, overall health, and urine pH. For ease of use, the detection times of metabolites have been incorporated into each parent drug. For example, heroin and cocaine can only be detected for a few hours after use, but their metabolites can be detected for several days in urine. The chart depicts the longer detection times of the metabolites. In the case of hair testing, the metabolytes are permanently embedded into hair, and the detection time is determined by the length of the hair sample used in the analysis. The standard length of head hair used in the test is 1.5", which corresponds to about 3 months. Body/pubic hair grows slower, and the same 1.5" would result in a longer detection time.[2] Oral fluid or saliva testing results for the most part mimic that of blood. The only exceptions are THC (tetrahydrocannabinol) and benzodiazepines. Oral fluid will likely detect THC from ingestion up to a maximum period of 6–12 hours. This continues to cause difficulty in oral fluid detection of THC and benzodiazepines.[3] Breath air for the most part mimics blood tests as well. Due to the very low levels of substances in the breath air, liquid chromatography—mass spectrometry has to be used to analyze the sample according to a recent publication wherein 12 analytes were investigated. Rapid oral fluid products are not approved for use in workplace drug testing programs and are not FDA cleared. Using rapid oral fluid drug tests in the workplace is prohibited in only:[4] California Kansas Maine Minnesota New York Vermont The following chart gives approximate detection periods for each substance by test type. Approximate values for detection periods Substance Urine Hair Blood / Oral Fluid Alcohol 6–24 hours[5] Note: Alcohol tests may measure ethyl glucuronide, which can stay in urine for up to 80 hours up to 90 days[6] 12 to 24 hours Amphetamines (except methamphetamine) 1 to 4 days[7] up to 90 days 12 hours Methamphetamine 5 days- up to a week[8] up to 90 days 1 to 3 days[8] MDMA (Ecstasy) 3 to 5 days up to 90 days 3 to 4 days Barbiturates (except phenobarbital) 1–4 days or 2–3 weeks up to 90 days 1 to 2 days Phenobarbital 2 to 3 weeks[9] up to 90 days 4 to 7 days Benzodiazepines Therapeutic use: up to 7 days. Chronic use (over one year): 4 to 6 weeks up to 90 days 6 to 48 hours Cannabis Passive inhalation: up to 22 minutes** Infrequent users: 7–10 Days Heavy users: 30 to 100 days[10][11][12] up to 90 days[10] 2 to 3 days in blood of infrequent users, up to 2 weeks in blood of heavy users[10] However, it depends on whether actual THC or THC metabolites are being tested for, the latter having a much longer detection time than the former. THC (found in marijuana) may only be detectable in saliva/oral fluid for 2 to 24 hours in most cases, though in rare cases has been detected up to 28 days after consumption.[13] Cocaine 2 to 4 days[14] (with exceptions for heavy users who can test positive up to 4/6 weeks, and individuals with certain kidney disorders) up to 90 days 2–10 days, heavy users or individuals with previous substance use 6/8 weeks Codeine 2 to 3 days up to 90 days 1 to 4 days Cotinine (a breakdown product of nicotine) 2 to 4 days up to 90 days 2 to 4 days Morphine 2 to 3 days[14] up to 90 days 1 to 3 days Tricyclic antidepressants (TCA's) 7 to 10 days Detectable but dose relationship not established. LSD 1–4 days (including metabolites) up to 90 days[15] 2 to 4 days Methadone 3 days[14] up to 90 days 24 hours Steroids 3 to 30 days PCP 3 to 7 days for single use; up to 30 days in chronic users[16] up to 90 days 1 to 3 days[16] Example of a device used to screen urine for drugs. There is no line present at the benzodiazepine area, indicating a positive screen for this class of drugs. Other drugs, and the negative control (labelled "C"), are negative. Urine analysis is primarily used because of its low cost. Urine drug testing is one of the most common testing methods used. The enzyme-multiplied immune test is the most frequently used urinalysis. Complaints have been made about the relatively high rates of false positives using this test.[17] Urine drug tests screen the urine for the presence of a parent drug or its metabolites. The level of drug or its metabolites is not predictive of when the drug was taken or how much the patient used.[18] Urine drug testing is an immunoassay based on the principle of competitive binding. Drugs which may be present in the urine specimen compete against their respective drug conjugate for binding sites on their specific antibody. During testing, a urine specimen migrates upward by capillary action. A drug, if present in the urine specimen below its cut-off concentration, will not saturate the binding sites of its specific antibody. The antibody will then react with the drug-protein conjugate and a visible colored line will show up in the test line region of the specific drug strip.[19] A common misconception is that a drug test that is testing for a class of drugs, for example, opioids, will detect all drugs of that class. However, most opioid tests will not reliably detect oxycodone, oxymorphone, meperidine, or fentanyl.[20] Likewise, most benzodiazepine drug tests will not reliably detect lorazepam.[21][22] However, urine drug screens that test for a specific drug, rather than an entire class, are often available. When an employer requests a drug test from an employee, or a physician requests a drug test from a patient, the employee or patient is typically instructed to go to a collection site or their home. The urine sample goes through a specified 'chain of custody' to ensure that it is not tampered with or invalidated through lab or employee error. The patient or employee's urine is collected at a remote location in a specially designed secure cup, sealed with tamper-resistant tape, and sent to a testing laboratory to be screened for drugs (typically the Substance Abuse and Mental Health Services Administration 5 panel). The first step at the testing site is to split the urine into two aliquots. One aliquot is first screened for drugs using an analyzer that performs immunoassay as the initial screen. To ensure the specimen integrity and to detect possible adulterants, additional parameters are tested for.[23] Some test the properties of normal urine, such as, urine creatinine, pH, and specific gravity. Others are intended to catch substances added to the urine to alter the test result, such as, oxidants (including bleach),[24] nitrites,[25] and gluteraldehyde.[26] If the urine screen is positive then another aliquot of the sample is used to confirm the findings by gas chromatography—mass spectrometry (GC-MS) or liquid chromatography - mass spectrometry methodology. If requested by the physician or employer, certain drugs are screened for individually; these are generally drugs part of a chemical class that are, for one of many reasons, considered more habit-forming or of concern. For instance, oxycodone and diamorphine may be tested, both sedative analgesics. If such a test is not requested specifically, the more general test (in the preceding case, the test for opioids) will detect most of the drugs of a class, but the employer or physician will not have the benefit of the identity of the drug. Employment-related test results are relayed to a medical review office (MRO) where a medical physician reviews the results. If the result of the screen is negative, the MRO informs the employer that the employee has no detectable drug in the urine, typically within 24 hours. However, if the test result of the immunoassay and GC-MS are non-negative and show a concentration level of parent drug or metabolite above the established limit, the MRO contacts the employee to determine if there is any legitimate reason—such as a medical treatment or prescription.[27] On-site instant drug testing is a more cost-efficient method of effectively detecting substance use amongst employees, as well as in rehabilitation programs to monitor patient progress.[28] These instant tests can be used for both urine and saliva testing. Although the accuracy of such tests varies with the manufacturer, some kits have rates of accuracy correlating closely with laboratory test results.[29] Main article: Breathalyzer Breath test being used on a volunteer. Breath test is a widespread method for quickly determining alcohol intoxication. A breath test measures the alcohol concentration in the body by a deep-lung breath. There are different instruments used for measuring the alcohol content of an individual though their breath. Breathalyzer is a widely known instrument which was developed in 1954 and contained chemicals unlike other breath-testing instruments.[30] More modernly used instruments are the infrared light-absorption devices and fuel cell detectors, these two testers are microprocessor controlled meaning the operator only has to press the start button. To get accurate readings on a breath-testing device the individual must blow for approximately 6 seconds and need to contain roughly 1.1 to 1.5 liters of breath. For a breath-test to result accurately and truly an operator must take steps such as avoiding measuring "mouth alcohol" which is a result from regurgitation, belching, or recent intake of an alcoholic beverage.[31] To avoid measuring "mouth alcohol" the operator must not allow the individual that's taking the test to consume any materials for at least fifteen minutes before the breath test. When pulled over for a driving violation if an individual in the United States refuses to take a breath test that individual's driver's license can be suspended for a 6 to 12 months time period. Main article: Hair analysis Hair analysis to detect addictive substances has been used by court systems in the United States, United Kingdom, Canada, and other countries worldwide. In the United States, hair testing has been accepted in court cases as forensic evidence following the Frye Rule, the Federal Rules of Evidence, and the Daubert Rule. As such, hair testing results are legally and scientifically recognized as admissible evidence.[32] Hair testing is commonly used in the USA as pre-employment drug test. The detection time for this test is roughly 3 months, which is the time, that takes head hair to grow ca. 1.5 inches, that are collected as a specimen. Longer detection times are possible with longer hair samples. A 2014 collaborative US study of 359 adults with moderate-risk drug use found, that a large number of participants, who reported drug use in the last 3 months, had negative hair tests. The tests were done using an immunoassay followed by a confirmatory GC-MS. For marijuana, only about half of self-disclosed users had a positive hair test. Under-identification of drug use by hair testing (or over-reporting) was also widespread for cocaine, amphetamines, and opioids. Because such under-identification was more common among participants, who self-reported an infrequent use, the authors suggested, that the immunoassay did not have the sensitivity required for such infrequent uses.[33] It is worth noting, that most earlier studies reported, that hair tests found ca. 50-fold higher prevalence of illicit drug use, than self reports.[34] In late 2022 the US Federal Motor Carrier Safety Administration denied a petition to recognize hair samples as an alternative (to the currently used urine samples) drug-testing method for truckers. The agency did not comment on the test validity, but rather stated, that it lacks the statutory authority to adopt new analytical methods.[35] Although some lower courts may have accepted hair test evidence, there is no controlling judicial ruling in either the federal or any state system declaring any type of hair test as reliable. Hair testing is now recognized in both the UK and US judicial systems. There are guidelines for hair testing that have been published by the Society of Hair Testing (a private company in France) that specify the markers to be tested for and the cutoff concentrations that need to be tested. Addictive substances that can be detected include Cannabis, Cocaine, Amphetamines and drugs new to the UK such as Mephedrone. In contrast to other drugs consumed, alcohol is deposited directly in the hair. For this reason the investigation procedure looks for direct products of ethanol metabolism. The main part of alcohol is oxidized in the human body. This means it is released as water and carbon dioxide. One part of the alcohol reacts with fatty acids to produce esters. The sum of the concentrations of four of these fatty acid ethyl esters (FAEEs: ethyl myristate, ethyl palmitate, ethyl oleate and ethyl stearate) are used as indicators of the alcohol consumption. The amounts found in hair are measured in nanograms (one nanogram equals only one billionth of a gram), however with the benefit of modern technology, it is possible to detect such small amounts. In the detection of ethyl glucuronide, or EtG, testing can detect amounts in picograms (one picogram equals 0.001 nanograms). However, there is one major difference between most drugs and alcohol metabolites in the way in which they enter into the hair: on the one hand like other drugs FAEEs enter into the hair via the keratinocytes, the cells responsible for hair growth. These cells form the hair in the root and then grow through the skin surface taking any substances with them. On the other hand, the sebaceous glands produce FAEEs in the scalp and these migrate together with the sebum along the hair shaft (Auwärter et al., 2001, Pragst et al., 2004). So these glands lubricate not only the part of the hair that is just growing at 0.3 mm per day on the skin surface, but also the more mature hair growth, providing it with a protective layer of fat. FAEEs (nanogram = one billionth of a gram) appear in hair in almost one order of magnitude lower than (the relevant order of magnitude of) EtG (picogram = one trillionth of a gram). It has been technically possible to measure FAEEs since 1993, and the first study reporting the detection of EtG in hair was done by Sachs in 1993.[36] In practice, most hair which is sent for analysis has been cosmetically treated in some way (bleached, permed etc.). It has been proven that FAEEs are not significantly affected by such treatments (Hartwig et al., 2003a). FAEE concentrations in hair from other body sites can be interpreted in a similar fashion as scalp hair (Hartwig et al., 2003b). Presumptive substance tests attempt to identify a suspicious substance, material or surface where traces of drugs are thought to be, instead of testing individuals through biological methods such as urine or hair testing. The test involves mixing the suspicious material with a chemical in order to trigger a color change to indicate if a drug is present. Most are now available over-the-counter for consumer use, and do not require a lab to read results. Duquenois reagent Benefits to this method include that the person who is suspected of drug use does not need to be confronted or aware of testing. Only a very small amount of material is needed to obtain results, and can be used to test powder, pills, capsules, crystals, or organic material. There is also the ability to detect illicit material when mixed with other non-illicit materials. The tests are used for general screening purposes, offering a generic result for the presence of a wide range of drugs, including Heroin, Cocaine, Methamphetamine, Amphetamine, Ecstasy/MDMA, Methadone, Ketamine, PCP, PMA, DMT, MDPV, and may detect rapidly evolving synthetic designer drugs. Separate tests for Marijuana/Hashish are also available.[37] There are five primary color-tests reagents used for general screening purposes. The Marquis reagent turns into a variety of colors when in the presence of different substances. Dille-Koppanyi reagent uses two chemical solutions which turns a violet-blue color in the presence of barbiturates. Duquenois-Levine reagent is a series of chemical solutions that turn to the color of purple when the vegetation of marijuana is added. Van Urk reagent turns blue-purple when in the presence of LSD. Scott test's chemical solution shows up as a faint blue for cocaine base.[38] In recent years, the use of presumptive test kits in the criminal justice system has come under great scrutiny due to the lack to forensic studies, questioned reliability, rendering of false positives with legal substances, and wrongful arrests.[39][40][41] Saliva / oral fluid-based drug tests can generally detect use during the previous few days. It is better at detecting very recent use of a substance. THC may only be detectable for 2–24 hours in most cases. On site drug tests are allowed per the Department of Labor.[42] Detection in saliva tests begins almost immediately upon use of the following substances, and lasts for approximately the following times: Alcohol: 6-12 h[43] Marijuana: 1-24h A disadvantage of saliva based drug testing is that it is not approved by FDA or SAMHSA for use with DOT / Federal Mandated Drug Testing.[44] Oral fluid is not considered a bio-hazard unless there is visible blood; however, it should be treated with care. Sweat patches are attached to the skin to collect sweat over a long period of time (up to 14 days).[45] These are used by child protective services, parole departments, and other government institutions concerned with drug use over long periods, when urine testing is not practical.[46] There are also surface drug tests that test for the metabolite of parent drug groups in the residue of drugs left in sweat. An example of a rapid, non-invasive, sweat-based drug test is fingerprint drug screening.[47] This 10 minute fingerprint test[48] is in use by a variety of organisations in the UK and beyond, including within workplaces,[49][50] drug treatment and family safeguarding services[51][52] at airport border control (to detect drug mules)[53] and in mortuaries to assist in investigations into cause of death.[54] Drug-testing a blood sample measures whether or not a drug or a metabolite is in the body at a particular time. These types of tests are considered to be the most accurate way of telling if a person is intoxicated. Blood drug tests are not used very often because they need specialized equipment and medically trained administrators. Depending on how much marijuana was consumed, it can usually be detected in blood tests within six hours of consumption. After six hours has passed, the concentration of marijuana in the blood decreases significantly. It generally disappears completely within 30 days. Can occur at any time, usually when the investigator has reason to believe that a substance is possibly being used by the subject by behavior or immediately after an employee-related incident occurs during work hours. Testing protocol typically conforms to the national medical standard, candidates are given up to 120 minutes to reasonably produce a urine sample from the time of commencement (in some instances this time frame may be extended at the examiner's discretion). In the case of life-threatening symptoms, unconsciousness, or bizarre behavior in an emergency situation, screening for common drugs and toxins may help find the cause, called a toxicology test or tox screen to denote the broader area of possible substances beyond just self-administered drugs. These tests can also be done post-mortem during an autopsy in cases where a death was not expected. The test is usually done within 96 hours (4 days) after the desire for the test is realized. Both a urine sample and a blood sample may be tested.[55] A blood sample is routinely used to detect ethanol/methanol and ASA/paracetamol intoxication. Various panels are used for screening urine samples for common substances, e.g. triage 8 that detects amphetamines, benzodiazepines, cocaine, methadone, opiates, cannabis, barbiturates and tricyclic antidepressants.[56] Results are given in 10–15 min. Similar screenings may be used to evaluate the possible use of date rape drugs. This is usually done on a urine sample.[55] Drug checks/tests (also known as pill testing) are provided at some events such as concerts and music festivals. Attendees can voluntarily hand over a sample of any drug or drugs in their possession to be tested to check what the drug is and its purity. The scheme is used as a harm reduction technique so people are more aware of what they are taking and the potential risks.[57][58][59] Drug and alcohol impairment while at work increases the risk of work-place accidents and decreases productivity. Employers such as the commercial driving and airline industry may conduct random drug tests on employees with the goal of deterring use to improve safety.[60] There is some evidence that increasing the use of random drug testing in the airline industry reduces the percentage of people who test positive, however, it is unclear if this decrease is associated with a corresponding decrease in fatal or non-fatal injuries, other accidents, number of days absent from work.[61] It is also not clear if there are other unwanted side effects that may result from random drug and alcohol testing in the workplace.[61] Anabolic steroids are used to enhance performance in sports and as they are prohibited in most high-level competitions drug testing is used extensively in order to enforce this prohibition. This is particularly so in individual (rather than team) sports such as athletics and cycling. Before testing samples, the tamper-evident seal is checked for integrity. If it appears to have been tampered with or damaged, the laboratory rejects the sample and does not test it. Next, the sample must be made testable. Urine and oral fluid can be used "as is" for some tests, but other tests require the drugs to be extracted from urine. Strands of hair, patches, and blood must be prepared before testing. Hair is washed in order to eliminate second-hand sources of drugs on the surface of the hair, then the keratin is broken down using enzymes. Blood plasma may need to be separated by centrifuge from blood cells prior to testing. Sweat patches are opened and the sweat collection component is removed and soaked in a solvent to dissolve any drugs present. Laboratory-based drug testing is done in two steps. The first step is the screening test, which is an immunoassay based test applied to all samples. The second step, known as the confirmation test, is usually undertaken by a laboratory using highly specific chromatographic techniques and only applied to samples that test positive during the screening test.[62] Screening tests are usually done by immunoassay (EMIT, ELISA, and RIA are the most common). A "dipstick" drug testing method which could provide screening test capabilities to field investigators has been developed at the University of Illinois.[63] After a suspected positive sample is detected during screening, the sample is tested using a confirmation test. Samples that are negative on the screening test are discarded and reported as negative. The confirmation test in most laboratories (and all SAMHSA certified labs) is performed using mass spectrometry, and is precise but expensive. False positive samples from the screening test will almost always be negative on the confirmation test. Samples testing positive during both screening and confirmation tests are reported as positive to the entity that ordered the test. Most laboratories save positive samples for some period of months or years in the event of a disputed result or lawsuit. For workplace drug testing, a positive result is generally not confirmed without a review by a Medical Review Officer who will normally interview the subject of the drug test. Urine drug test kits are available as on-site tests, or laboratory analysis. Urinalysis is the most common test type and used by federally mandated drug testing programs and is considered the Gold Standard of drug testing. Urine based tests have been upheld in most courts for more than 30 years. However, urinalysis conducted by the Department of Defense has been challenged for reliability of testing the metabolite of cocaine. There are two associated metabolites of cocaine, benzoylecgonine (BZ) and ecgonine methyl ester (EME), the first (BZ) is created by the presence of cocaine in an aqueous solution with a pH greater than 7.0, while the second (EME) results from the actual human metabolic process. The presence of EME confirms actual ingestion of cocaine by a human being, while the presence of BZ is indicative only. BZ without EME is evidence of sample contamination, however, the US Department of Defense has chosen not to test for EME in its urinalysis program.[64][relevant?] A number of different analyses (defined as the unknown substance being tested for) are available on Urine Drug Screens. Spray (sweat) drug test kits are non-invasive. It is a simple process to collect the required specimen, no bathroom is needed, no laboratory is required for analysis, and the tests themselves are difficult to manipulate and relatively tamper-resistant. The detection window is long and can detect recent drug use within several hours. There are also some disadvantages to spray or sweat testing. There is not much variety in these drug tests, only a limited number of drugs can be detected, prices tend to be higher, and inconclusive results can be produced by variations in sweat production rates in donors. They also have a relatively long specimen collection period and are more vulnerable to contamination than other common forms of testing.[45] Hair drug testing is a method that can detect drug use over a much longer period of time than saliva, sweat or urine tests. Hair testing is also more robust with respect to tampering. Thus, hair sampling is preferred by the US military[65] and by many large corporations, which are subject to Drug-Free Workplace Act of 1988. Head hair normally growth at the rate of 0.5 inches per month.[66] Thus, the most common hair sample length of 1.5" from the scalp would detect drug use within the last 90-100 days. 80-120 strands of hair are sufficient for the test.[67] In the absence of hair on the head, body hair can be used as an acceptable substitute. This includes facial hair, the underarms, arms, and legs or even pubic hair. Because body hair usually grows slower than head hair, drugs can often be detected in body hair for longer periods, e.g. up to 12 months. Currently, most entities that use hair testing have prescribed consequences for individuals removing hair to avoid a hair drug test. Most drugs are analysed in hair samples not as the original psychoactive molecules, but rather as their metabolytes. For example, ethanol is determined as ethyl glucuronide, while cocaine use is confirmed using ecgonine. Testing for metabolytes reduces the likelihood of false positive results due to contamination. One disadvantage of hair testing is, that it cannot detect recent drug use, because it takes at least a week after a drug intake for the metabolytes to show up in a growing hair above the skin.[65] Urine tests are better suited for detecting recent (within a week) drug use. In a practical test, hair sample is usually washed with a low polarity solvent (such as dichloromethane) to remove surface contaminations. Then, the sample is pulverized and extracted with a more polar solvent, such as methanol.[68] Although thousand different substances can be determined in a single gas chromatography–mass spectrometry or liquid chromatography–mass spectrometry experiment, due to the low concentration of analytes, practical measurements (see selective ion monitoring) are limited to a smaller number (10-20) of analytes. Designer drugs are usually missed in such measurements, because the analyst must know in advance what chemicals to look for. Most hair testing laboratories use the aforementioned chromato-mass-spectrometry methods for confirmation or for rarely tested drugs only. Mass screening (preliminary or final) is usually done with immunoassays, because of their lower cost. The results of federally mandating drug testing were similar to the effects of simply extending to the trucking industry the right to perform drug tests, and it has been argued that the latter approach would have been as effective at lower cost.[69] Psychologist Tony Buon has criticized the use of workplace drug testing on a number of grounds, including: Flawed technology: The real world performance of testing is much lower than that claimed by its promoters. Buon suggest that tests are probably adequate for rehabilitation and treatment situations, possibly adequate for pre-employment situations, but not for dismissing employees. Ethical Issues: Because of the fairly simple ways that an employee can invalidate the test, drug testing must be strictly monitored. This means that the specimen must be observed leaving the body. Many legal objections currently being raised in the courts about drug testing are pointing to legal requirements of prior notice, consent, due process, and cause.[70][71] Wrong focus: As has been shown with Employee Assistance Programs, the focus of management concern should be on work performance decline. Buon suggests effective management practices are an infinitely better approach to managing workplace alcohol and other drug issues.[72] Tony Buon has also reported by the CIPD as stating that "drug testing captures the stupid—experienced drug users know how to beat the tests".[73] From a penological standpoint, one purpose of drug testing is to help classify the people taking the drug test within risk groups so that those who pose more of a danger to the public can be incapacitated through incarceration or other restrictions on liberty. Thus, the drug testing serves a crime control purpose even if there is no expectation of rehabilitating the drug user through treatment, deterring drug use through sanctions, or sending a message that drug use is a deviant behavior that will not be tolerated.[74] A study in 2004 by the Independent Inquiry into Drug Testing at Work found that attempts by employers to force employees to take drug tests could potentially be challenged as a violation of privacy under the Human Rights Act 1998 and Article 8 of the European Convention of Human Rights.[75] However, this does not apply to industries where drug testing is a matter of personal and public safety or security rather than productivity. In consultation with Dr. Carlton Turner, President Ronald Reagan issued Executive Order 12564. In doing so, he instituted mandatory drug-testing for all safety-sensitive executive-level and civil-service Federal employees. This was challenged in the courts by the National Treasury Employees Union. In 1988, this challenge was considered by the US Supreme Court.[76] A similar challenge resulted in the Court extending the drug-free workplace concept to the private sector.[77] These decisions were then incorporated into the White House Drug Control Strategy directive issued by President George H.W. Bush in 1989.[78] All defendants serving on federal probation or federal supervised release are required to submit to at least three drug tests. Failing a drug test can be construed as possession of a controlled substance, resulting in mandatory revocation and imprisonment.[79] There have been inconsistent evaluation results as to whether continued pretrial drug testing has beneficial effects.[80] Testing positive can lead to bail not being granted, or if bail has already been granted, to bail revocation or other sanctions. Arizona also adopted a law in 1987 authorizing mandatory drug testing of felony arrestees for the purpose of informing the pretrial release decision, and the District of Columbia has had a similar law since the 1970s. It has been argued that one of the problems with such testing is that there is often not enough time between the arrest and the bail decision to confirm positive results using GC/MS technology. It has also been argued that such testing potentially implicates the Fifth Amendment privilege against self-incrimination, the right to due process (including the prohibition against gathering evidence in a manner that shocks the conscience or constitutes outrageous government conduct), and the prohibition against unreasonable searches and seizures contained in the Fourth Amendment.[81] According to Henriksson, the anti-drug appeals of the Reagan administration "created an environment in which many employers felt compelled to implement drug testing programs because failure to do so might be perceived as condoning drug use. This fear was easily exploited by aggressive marketing and sales forces, who often overstated the value of testing and painted a bleak picture of the consequences of failing to use the drug testing product or service being offered."[82] On March 10, 1986, the Commission on Organized Crime asked all U.S. companies to test employees for drug use. By 1987, nearly 25% of the Fortune 500 companies used drug tests.[83] According to an uncontrolled self-report study done by DATIA and Society for Human Resource Management in 2012 (sample of 6,000 randomly selected human resource professionals), human resource professionals reported the following results after implementing a drug testing program: 19% of companies reported a subjective increase in employee productivity, 16% reported a decrease in employee turnover (8% reported an increase), and unspecified percentages reported decreases in absenteeism and improvement of workers' compensation incidence rates.[84] According to US Chamber of Commerce 70% of all illicit drug users are employed.[85] Some industries have high rates of employee drug use such as construction (12.8%), repair (11.1%), and hospitality (7.9-16.3%).[86] A person conducting a business or undertaking (PCBU—the new term that includes employers) has duties under the work health and safety (WHS) legislation to ensure a worker affected by alcohol or other drugs does not place themselves or other persons at risk of injury while at work. Workplace policies and prevention programs can help change the norms and culture around substance use.[87] All organisations—large and small—can benefit from an agreed policy on alcohol and drug misuse that applies to all workers. Such a policy should form part of an organisations overall health and safety management system. PCBUs are encouraged to establish a policy and procedure, in consultation with workers, to constructively manage alcohol and other drug related hazards in their workplace. A comprehensive workplace alcohol and other drug policy should apply to everyone in the workplace and include prevention, education, counselling and rehabilitation arrangements. In addition, the roles and responsibilities of managers and supervisors should be clearly outlined.[88] All Australian workplace drug testing must comply with Australian standard AS/NZS4308:2008.[89] In Victoria, roadside saliva tests detect drugs that contain:[90] THC (Delta-9 tetrahydrocannabinol), the active component in cannabis. methamphetamine, also known as "ice", "crystal" and "crank". MDMA (Methylenedioxymethamphetamine), which is known as ecstasy. In February 2016 a New South Wales magistrate "acquitted a man who tested positive for cannabis". He had been arrested and charged after testing positive during a roadside drug test, despite not having smoked for nine days. He was relying on advice previously given to him by police.[91] In the United States federal criminal system, refusing to take a drug test triggers an automatic revocation of probation or supervised release.[92][93] In Victoria, Australia the driver of the car has the option to refuse the drug test. Refusing to undergo a drug test or refusing to undergo a secondary drug test after the first one, triggers an automatic suspension and disqualification for a period of two years and a fine of AUD$1000. The second refusal triggers an automatic suspension and disqualification for a period of four years and an even larger fine. In 2000, an Australian Mining Company South Blackwater Coal Ltd with 400 employees, imposed drug-testing procedures, and the trade unions advised their members to refuse to take the tests, partly because a positive result does not necessarily indicate present impairment; the workers were stood-down by the company without pay for a week.[94] In 2003, sixteen members of the Chicago White Sox considered refusing to take a drug test, in hopes of making steroid testing mandatory.[95] In 2006, Levy County, Florida, volunteer librarians resigned en masse rather than take drug tests.[96] In 2010, Iranian super heavyweight class weightlifters refused to submit to a drug test authorized by the Iran Weightlifting League.[97] Cannabis drug tests Detoxification Drug checking Drug testing welfare recipients Drug-Free Workplace Act of 1988 Equine drug testing Forensic toxicology Lacing (drugs) Occupational health concerns of cannabis use Poppy seed defence Presumptive and confirmatory tests Reagent testing School district drug policies Urinalysis

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Pharmaceutical compound 2C-B2C-B structure3D representation of a 2C-B moleculeClinical dataOther names4-Bromo-2,5-dimethoxyphenethylamine; 2,5-Dimethoxy-4-bromophenethylamine; Nexus; Venus; Bromo; Bees; Erox; Synergy; Performax; Toonies[1]Routes ofadministrationBy mouth, insufflation, rectalDrug classSerotonergic psychedelic; Hallucinogen; Serotonin 5-HT2A receptor agonistATC codeNoneLegal statusLegal status AU: S9 (Prohibited substance) BR: Class F2 (Prohibited psychotropics) CA: Schedule III DE: Anlage I (Authorized scientific use only) UK: Class A US: Schedule I UN: Psychotropic Schedule II Pharmacokinetic dataMetabolismLiver (MAO and CYP450)[5][1]MetabolitesBDMPE, BDMPAA, BDMBA, and others[2]Onset of actionOral: 20–90 min[2]Elimination half-life1.2–2.5 hours[3][4]Duration of actionOral: 2–8 hours[5][2][1]ExcretionUrine[2][1]Identifiers IUPAC name 2-(4-Bromo-2,5-dimethoxyphenyl)ethanamine CAS Number66142-81-2PubChem CID98527DrugBankDB01537ChemSpider88978UNIIV77772N32HKEGGC22775ChEBICHEBI:189669ChEMBLChEMBL292821CompTox Dashboard (EPA)DTXSID10216332 ECHA InfoCard100.164.088 Chemical and physical dataFormulaC10H14BrNO2Molar mass260.131 g·mol−13D model (JSmol)Interactive image SMILES COc1cc(CCN)c(OC)cc1Br InChI InChI=1S/C10H14BrNO2/c1-13-9-6-8(11)10(14-2)5-7(9)3-4-12/h5-6H,3-4,12H2,1-2H3Key:YMHOBZXQZVXHBM-UHFFFAOYSA-N 2C-B, also known as 4-bromo-2,5-dimethoxyphenethylamine or by the slang name Nexus, is a synthetic psychedelic drug of the 2C family, mainly used as a recreational drug.[2][1][6] It was first synthesized by Alexander Shulgin in 1974 for use in psychotherapy. To date, there is limited scientific information regarding the drug's pharmacokinetics and pharmacological effects in humans. The existing studies primarily classify 2C-B as a stimulant and hallucinogen, and less commonly an entactogen.[7] 2C-B is also known by a number of slang names and appears on the illicit market in multiple forms:[8][9] as a powder, in capsules or pills. For recreational use, the substance is generally consumed orally or nasally. 1000mg of 2C-B 2C-B became briefly popular in the United States as substitute for the street drug ecstasy (MDMA) when the latter became illegal in 1985.[10] Many 2C-B users are young adults who attend raves.[8] Although 2C-B is still used in the rave subculture (commonly mistaken for and/or sold as ecstasy), more knowledgeable use has become more widespread in the 2000s.[11] There are claims that 2C-B was used as entheogen by the Sangoma, Nyanga, and Amagqirha people in place of their traditional plants; they refer to the chemical as Ubulawu Nomathotholo, which roughly translates to "Medicine of the Singing Ancestors".[12][13][14] In Shulgin's book PiHKAL, the oral dosage range is listed as 12 to 24 mg.[15] The common oral recreational dose per Erowid is around 15 to 25 mg.[16] Shulgin and others describe 2C-B as having a steep dose–response curve, such that a small increase in dose can result in an unexpectedly large increase in effects.[15][17] 2C-B dosage[citation needed] Oral Insufflated ED50 10 mg 4–6 mg Moderate 15–25 mg 5–9 mg Strong 26–35 mg 10–20 mg Extremely Intense >35 mg >20 mg Duration 4–8 hours 2–4 hours When sold as "Ecstasy", tablets containing 2C-B often contain about 5 mg of the drug, an amount which produces stimulatory effects that mimic the effects of MDMA; in contrast, tablets marketed as 2C-B have larger quantities of the drug (10–20 mg) which cause hallucinogenic effects.[18] When orally consumed, 2C-B has a much longer delay before the onset of effects than when it is insufflated. Oral ingestion generally takes roughly 45–75 minutes for the effects to be felt, plateau lasts 2–4 hours, and coming down lasts 1–2 hours. Rectal administration onset varies from 5 to 20 minutes. Insufflated onset takes 1–10 minutes for effects to be felt. The duration can last from 4 to 12 hours depending on route of administration, dose, and other factors.[19] With insufflation, the effects are more abrupt and intense but have a significantly shorter duration, while oral usage results in a milder, longer experience. When insufflated, the onset happens very rapidly, usually reaching the peak at about 20–40 minutes and plateauing for 2–3 hours. 2C-B is also considered one of the most painful drugs to insufflate, with users reporting intense nasal burning.[20] The sudden intensity of the experience combined with the pain can often start the experience with a negative imprint and nausea is also increased with insufflation, compounding the issue. 2C-B pill with heart logo Little academic research has been conducted on the effects of 2C-B in humans. The information available is largely anecdotal and limited. Effects are often described as being more easily managed than other psychedelics;[20][17] it is often compared to a mixture of a serotonergic psychedelic and MDMA.[21] At 5–10 mg, experiments with young chickens have shown it to produce effects similar to a low dosage of amphetamines.[22] The anecdotal effects of 2C-B that have been reported by users on online discussion forums include:[19][23][24] At low doses, the experience may shift in intensity from engaging to mild/undetectable. Experienced users report the ability to take control of the effects and switch from engaged to sober at will. The hallucinations have a tendency to decrease and then increase in intensity, giving the users a sense of "waves" or even glowing. These are popularly described as "clichéd '70s visuals" or objects taking on "water color"-like textures. While the effects of the drug often render users unable to concentrate deeply on anything in particular, some can become engrossed in an activity such as watching a movie or playing a video game, thus distracting themselves from the visual and auditory effects of the drug. Excessive giggling or smiling is common, as is a tendency for deeper "belly laughs". Some users say that the effects are more intense when listening to music and report that they can see sounds and noises. Some users experience a decrease in visual acuity, although others report sharper vision. Through increased awareness of one's body, attention may be brought to perceived "imperfections" or internal body processes.The following effects are highly dose-dependent. Open eye visuals (OEVs), such as cartoon-like distortions and red or green halos around objects. Closed eye visuals (CEVs) are more common than OEVs. Affects and alters ability to communicate, engage in deep thought, or maintain attention span. Some users report experiencing frightening or fearful effects during the experience. Users describe feeling frigid or cold on reaching a plateau, while others feel wrapped in comfortable blankets/ultimate pleasure. Coordination may be affected; some users lose balance or have perceptual distinction problems. Onset time of 2C-B is highly dose dependent, but usually from 45 to 75 minutes. Taken on a full stomach, the onset time is increased to two hours or more. Before it was scheduled, 2C-B was sold in small doses as an aphrodisiac (see: § History). Some users report aphrodisiac effects at lower doses.[15][23] Clinical studies in humans suggest that 2C-B is a psychedelic with some possible entactogen-like effects.[7][3][25] Specific effects have included slight hallucinogenic states, perceptual changes, ego dissolution, time dilation, euphoria, feelings of well-being, reduced anger, increased reactivity to negative emotional stimuli, decreased ability to recognize expressions of happiness, augmented emotionality in speech, and mild sympathomimetic effects such as pressor effects, among others.[7][3][25] Some users report mild "jitters" (body tremors), shuddering breath, and/or mild muscle spasms after insufflating 2C-B. Whether or not these effects are enjoyable depends on the user; Mild to intense diarrhea, gas, nausea, and general gastrointestinal discomfort; Severe headaches after coming down from large doses have been reported. However, many users report a lack of "comedown" or "crash", instead noting a gradual return to sobriety; At doses over 30–40 mg the user may experience frightening hallucinations, as well as tachycardia, hypertension, and hyperthermia;[26] 2C-B HCl is very painful to insufflate. Anecdotal evidence suggests that 2C-B HBr, the hydrobromide salt with greater water solubility, is less irritating to the mucous membranes lining the nose but slightly less potent when compared dose-for-dose with the HCl salt;[27] Rectal administration of a water-based solution of 2C-B is known to be less painful than insufflation and much more potent than oral administration. Severe adverse reactions are rare, but use of 2C-B was linked to significant brain injury in one case report; the alleged "2C-B" was never actually discovered by testing so the only evidence suggesting 2C-B was the cause was the victim's own words, without taking into consideration that adulteration and impurities are very common in illicit drugs.[28] There is a case report of acquired synesthesia following a single very high dose of 2C-B.[29] There is also a case report of persistent psychosis following a single dose of 2C-B.[30] The lethal dosage is unknown. It was reported in PiHKAL, by Alexander Shulgin, that a psychologist had accidentally taken a 100 mg dose orally without apparent harm.[15] See also: Psychedelic drug § Interactions, and Trip killer § Serotonergic psychedelic antidotes 2C-B is metabolized by the monoamine oxidase (MAO) enzymes MAO-A and MAO-B.[31][32] Monoamine oxidase inhibitors (MAOIs) such as phenelzine, tranylcypromine, moclobemide, and selegiline may potentiate the effects of 2C-B.[31][32][33] This may result in overdose and serious toxicity.[33][31] 2C-B may also have interactions with other medications and drugs.[5] 2C-B activities Target Affinity (Ki, nM) 5-HT1A 130–311 5-HT1B 104 5-HT1D 26 5-HT1E 120 5-HT1F ND 5-HT2A 0.66–32 (Ki)1.20–689 (EC50Tooltip half-maximal effective concentration)4–101% (EmaxTooltip maximal efficacy) 5-HT2B 13.5–97 (Ki)12.6–130 (EC50)52–97% (Emax) 5-HT2C 32–90 (Ki)0.03–493 (EC50)50–116% (Emax) 5-HT3 >10,000 5-HT4 ND 5-HT5A >10,000 5-HT6 320 5-HT7 210 α1A >10,000 α1B >10,000 α1D ND α2A 309–320 α2B >10,000 α2C 103 β1 >10,000 β2 >10,000 β3 ND D1 12,000 D2 2,200–25,200 D3 7,116–10,000 D4 >10,000 D5 >10,000 H1–H4 >10,000 M1–M2 >10,000 M3 822 M4–M5 >10,000 I1 2,155 σ1 >10,000 σ2 >10,000 TAAR1Tooltip Trace amine-associated receptor 1 90–3,000 (Ki) (rodent)3,300–7,190 (EC50) (human) SERTTooltip Serotonin transporter 9,700–13,300 (Ki)18,000–312,900 (IC50Tooltip half-maximal inhibitory concentration) NETTooltip Norepinephrine transporter 27,400–31,000 (Ki)44,000–67,100 (IC50) DATTooltip Dopamine transporter 6,500–>30,000 (Ki)231,000 (IC50) MAO-ATooltip Monoamine oxidase A 125,000 (IC50) MAO-BTooltip Monoamine oxidase B 58,000 (IC50) Notes: The smaller the value, the more avidly the drug binds to the site. All proteins are human unless otherwise specified. Refs: [34][35][36][37][1][38][39][40][41][42][43][44][45][46] Unlike most psychedelics, 2C-B has been shown to be a low efficacy human serotonin 5-HT2A and 5-HT2C receptor partial agonist.[47] This suggests that activation of the 5-HT2A-coupled phospholipase D pathway[47] or functional antagonism of 5-HT2A may also play a role. The rank order of 5-HT2A receptor antagonist potency for this family of drugs in Xenopus is 2C-I > 2C-B > 2C-D > 2C-H.[48] Although 2C-B itself was not evaluated, other closely related members of the 2C series, including 2C-C, 2C-D, 2C-E, 2C-I, and 2C-T-2, all showed no activity as monoamine releasing agents of serotonin, norepinephrine, or dopamine (EC50Tooltip half-maximal effective concentration = >100,000 nM or "inactive").[49][50] Likewise, these other 2C derivatives showed little activity as serotonin 5-HT1A receptor agonists (EC50 = >3,000 nM).[50] The September 1998 issue of Journal of Analytical Toxicology reported that very little data exists about the pharmacological properties, metabolism, and toxicity of 2C-B.[citation needed] With 30 mg 2C-B by oral administration, its peak concentrations (mean ± SD) were 5.4 ± 1.7 ng/mL and its time to peak concentrations were 2.3 ± 1.0 hours.[4] 2C-B has been shown to be metabolized by liver hepatocytes, resulting in deamination and demethylation that produces several products. Oxidative deamination results in the 2-(4-bromo-2,5-dimethoxyphenyl)ethanol (BDMPE) and 4-bromo-2,5-dimethoxyphenylacetic acid (BDMPAA) metabolites. Additionally, 4-bromo-2,5-dimethoxybenzoic acid (BDMBA) can be produced by oxidative deamination. Further metabolism of BDMPE and BDMPAA may occur by demethylation. Alternatively, the later metabolites can be generated by demethylation of 2C-B followed by oxidative deamination.[26] Deamination of 2C-B is mediated by the monoamine oxidase (MAO) enzymes MAO-A and MAO-B.[31][32][4] There is species differentiation in the metabolism of 2C-B. Mice hepatocytes produce 4-bromo-2,5-dimethoxyphenol (BDMP), a previously unknown metabolite. Meanwhile, human, monkey, and rabbit hepatocytes produce 2-(4-bromo-2-hydroxy-5-methoxyphenyl)-ethanol (B-2-HMPE), but dog, rat, and mouse hepatocytes do not.[26] 2C-B's metabolites BDMPAA and 4-bromo-2-hydroxy-5-methoxyphenylacetic acid (B-2-HMPAA) in humans occur at peak concentrations 280-fold and 17-fold higher than those of 2C-B with oral administration of 2C-B, respectively.[4] The elimination half-life of 2C-B in humans is 1.2 to 2.5 hours.[3][4] Analogues and derivatives of 2C-B: 25-N: 25B-N1POMe 25B-NAcPip 25-NB: 25B-NB 25B-NB23DM 25B-NB25DM 25B-NB3OMe 25B-NB4OMe 25B-NBF 25B-NBMD 25B-NBOH 25B-NBOMe (NBOMe-2CB) DMBMPP 25-NM: 25B-NMe7BF 25B-NMe7BT 25B-NMe7Bim 25B-NMe7Box 25B-NMe7DHBF 25B-NMe7Ind 25B-NMe7Indz 25B-NMePyr Substituted benzofurans: 2C-B-FLY 2C-B-BUTTERFLY 2C-B-DRAGONFLY 2CBFly-NBOMe (NBOMe-2CB-Fly) DOB-FLY DOB-2-DRAGONFLY-5-BUTTERFLY N-(2C)-fentanyl: N-(2C-B) fentanyl[51] N-(2C-B-FLY) fentanyl[52] Other: BOB BOH-2C-B, β-Hydroxy-2C-B, βOH-2CB[53][54] BMB 2C-B-5-hemifly 2C-B-aminorex (2C-B-AR) 2C-B-AN 2C-B-BZP 2C-B-FLY-NB2EtO5Cl 2C-B-PP 2CB-Ind βk-2C-B (beta-keto 2C-B) N-Ethyl-2C-B TCB-2 (2C-BCB) A variety of N-substituted derivatives of 2C-B have been tested, including N-methyl-2CB, N,N-dimethyl-2CB, N-ethyl-2CB and N-benzyl-2CB. Most simple alkyl derivatives were considerably less potent than 2C-B, with N-ethyl-2CB for instance having a 40 times lower affinity for the 5-HT2A receptor. The N-benzyl derivative however was found to have higher binding affinity than 2C-B itself, with N-(4-bromobenzyl)-2CB binding even more tightly.[55] This initial research did not include functional assays of activity, but later led to the development of potent substituted N-benzyl derivatives such as 25B-NBOMe,[56] and 25B-NBOH. βk-2C-B shows dramatically reduced potency and efficacy as a serotonin 5-HT2A receptor agonist compared to 2C-B.[42] ASR-2001 (2CB-5PrO) is another notable analogue of 2C-B which is under development for treatment of psychiatric disorders.[57][58][59] Exposing compounds to the reagents gives a colour change which is indicative of the compound under test. Marquis Mecke Mandelin Liebermann Froehde Robadope Yellow to green Yellow to olive brownish green Yellow to black Yellow to green Slow pink Ehrlich Hofmann Simon's Scott Folin No reaction No reaction No reaction No reaction (Light) purple 2C-B was synthesized from 2,5-dimethoxybenzaldehyde by American chemist Alexander Shulgin in 1974. It first saw use among the psychiatric community as an aid during therapy, but was abandoned due to gastrointestinal effects and the lack of empathogenic effects.[1] 2C-B was first sold commercially as a purported aphrodisiac[60] under the trade name "Erox", which was manufactured by the German pharmaceutical company Drittewelle.[61] For several years, it was available as tablets in Dutch smart shops under the name "Nexus" and "B-Dub".[citation needed] Street purity of 2C-B, when tested, has been found to be relatively high.[62] Researchers in Spain found that 2C-B samples in the country doubled between 2006 and 2009, switched from primarily powder form to tablets, and exhibited "low falsification rates".[21] An analysis of street samples in the Netherlands found impurities "in small percentages"; only one of the impurities, the N-acetyl derivative of 2C-B, could be identified, and comprised 1.3% of the sample. The authors suggested that this compound was a by-product of 2C-B synthesis.[18] In 2011, street prices in the United States ranged between $10 and $30 per tablet when purchased in small quantities.[8] Larger retail purchases cost between $200 and $500 per gram. Wholesale purchases of 2C-B would lower the price ($100 to $300 per gram in 2001, $30 to $100 on the darknet in 2020).[60] The UN Commission on Narcotic Drugs added 2C-B to Schedule II of the Convention on Psychotropic Substances in March 2001.[63] 2C-B is a scheduled drug in most jurisdictions.[64] The following is a partial list of territories where the substance has been scheduled. 2C-B is controlled under the List 1, as well as similar substances like 2C-I or 2C-T-2.[65] 2C-B is controlled in Australia and on the list of substances subject to import and export controls (Appendix B). It was placed on Schedule One of the Drugs Misuse and Trafficking Act when it first came to notice in 1994, when in a showcase legal battle chemist R. Simpson was charged with manufacturing the substance in Sydney. Alexander Shulgin came to Australia to testify on behalf of the defense, to no avail. 2C-B is not specifically listed in the Australia Poisons Standard (October 2015), however similar drugs such as 2C-T-2 and 2C-I are making 2C-B fall under the Australian analogue act.[66] In Belgium, 2C-B is a controlled substance making production, distribution, and possession illegal. In Brazil, 2C-B is a controlled substance making production, distribution, and possession illegal. In Canada, 2C-B is classified under Controlled Drugs and Substances Act as Schedule III as "4-bromo-2,5-dimethoxybenzeneethanamine and any salt, isomer or salt of isomer thereof".[67] 2C-B has been rescheduled (Schedule III), in a new amendment, taking effect on October 31, 2016. This is to include the other 2C-x analogues.[68] In August 2007, 2C-B, along with many other psychologically active substances,[69] was added to Ley 20.000, known as the Ley de Drogas [es]. Possession of more than 200 mg of 2C-B is punishable with a two years jail sentence.[70] Smaller amount is punishable by a fine. The 200 mg threshold is merely a guideline which the court can reconsider depending on circumstances. In Denmark, 2C-B is listed as a category B drug.[71] In Estonia, 2C-B is classified as Schedule I. In Germany, 2C-B is controlled in the Betäubungsmittelgesetz (BtMG) Anlage I as "Bromdimethoxyphenethylamin" (BDMPEA). 2C-B is schedule I (tabella I).[72] In Japan, 2C-B was scheduled in 1998. It was previously marketed as "Performax". In Luxembourg, 2C-B is a prohibited substance since 2001.[73] In the Netherlands, 2C-B was scheduled on July 9, 1997. In the Netherlands, 2C-B became a list I substance of the Opium Law despite no health incidents occurring. Following the ban, other phenethylamines were sold in place of 2C-B until the Netherlands became the first country in the world to ban 2C-I, 2C-T-2 and 2C-T-7 alongside 2C-B. In Norway, 2C-B was classified as Schedule II on March 22, 2004, listed as 4-bromo-2,5-dimethoxyphenethylamine.[74] 2C-B is schedule I (I-P group) in Poland. Banned as a narcotic drug with a criminal penalty for possession of at least 10 mg.[75] In Spain, 2C-B was added to Category 2 prohibited substances in 2002. 2C-B is currently classified as Schedule I in Sweden. 2C-B was first classified as "health hazard" under the act Lagen om förbud mot vissa hälsofarliga varor (Act on the Prohibition of Certain Goods Dangerous to Health) as of April 1, 1999, under SFS 1999:58[76] that made it illegal to sell or possess. Then it became schedule I as of June 1, 2002, published in LVFS 2002:4[77] but mislabeled "2-CB" in the document. However, this was corrected in a new document, LVFS 2009:22[78] effective December 9, 2009. In Switzerland, 2C-B is listed in Anhang D of the DetMV and is illegal to possess.[79] All drugs in the 2C family are Class A under the Misuse of Drugs Act which means they are illegal to produce, supply or possess. Possession carries a maximum sentence of seven years imprisonment while supply is punishable by life imprisonment and an unlimited fine.[80] In the United States, 2C-B is classified as a Schedule I controlled substance. This became permanent law on June 2, 1995,[81] following a proposal by the Drug Enforcement Administration in December 1994.[82] ^ a b c d e f g Nugteren-van Lonkhuyzen JJ, van Riel AJ, Brunt TM, Hondebrink L (December 2015). "Pharmacokinetics, pharmacodynamics and toxicology of new psychoactive substances (NPS): 2C-B, 4-fluoroamphetamine and benzofurans". Drug Alcohol Depend. 157: 18–27. doi:10.1016/j.drugalcdep.2015.10.011. 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Retrieved from " 3 Pharmaceutical compound 2C-iBuClinical dataOther names2,5-Dimethoxy-4-isobutylphenethylamine; 4-Isobutyl-2,5-dimethoxyphenethylamine; 2C-iBu; 2C-IB; ELE-02; ELE02; ELEU02Routes ofadministrationOral;[1] Ophthalmic[2][3]Drug classSerotonin 5-HT2A receptor agonist; Serotonergic psychedelic; Hallucinogen; Anti-inflammatory drugPharmacokinetic dataProtein binding74%[4]Duration of action20 hours[1]Identifiers IUPAC name 2-[2,5-dimethoxy-4-(2-methylpropyl)phenyl]ethanamine PubChem CID57486931Chemical and physical dataFormulaC14H23NO2Molar mass237.343 g·mol−13D model (JSmol)Interactive image SMILES CC(C)CC1=C(C=C(C(=C1)OC)CCN)OC InChI InChI=1S/C14H23NO2/c1-10(2)7-12-9-13(16-3)11(5-6-15)8-14(12)17-4/h8-10H,5-7,15H2,1-4H3Key:FLBABUVVTQBINW-UHFFFAOYSA-N 2,5-Dimethoxy-4-isobutylphenethylamine (2C-iBu or 2C-IB), also known by its developmental code name ELE-02, is a serotonin 5-HT2A receptor agonist, serotonergic psychedelic, and anti-inflammatory drug which is under development for the treatment of inflammation.[2][3][4][5][6][7] It is a member of the phenethylamine and 2C families of compounds.[4][5][7] The drug is being developed as a topical eye drop for treatment of inflammatory eye conditions.[2][3] There is also interest in 2C-iBu and related drugs for treatment of systemic inflammation and neuroinflammation.[8][9][10][11][12][5] 2C-iBu was not assessed or discovered by Alexander Shulgin and was not described in PiHKAL (Phenethylamines I Have Known and Loved) (1991).[7][13] However, he did include 2C-iBu (as "2C-IB") as a DOM analogue in a table in The Shulgin Index, Volume One: Psychedelic Phenethylamines and Related Compounds (2011).[1] In addition, he stated in a footnote that a 5 mg oral dose of 2C-iBu produces threshold activity and has a long duration of about 20 hours.[1] The cited source for these observations, however, was only a 2006 personal communication with "M. Mueller." [1] 2C-iBu was subsequently more thoroughly characterized by Charles D. Nichols and colleagues at Louisiana State University School of Medicine as a novel anti-inflammatory drug in the late 2010s.[4][7] Eleusis has licensed 2C-iBu intellectual property from LSU and the drug has reached the preclinical research stage of development, but no recent development has been reported as of October 2023.[2][3] 2C-iBu molecular targets[4] Target Affinity (pKi) 2C-iBu (R)-DOI 5-HT1A 7.1 5.9 5-HT1B 7.3 5.6 5-HT1D 7.2 ND 5-HT2A 8.9 10.4 5-HT2B 7.8 8.6 5-HT2C 9.6 9.2 5-HT6 5.9 ND 5-HT7 6.5 ND 2C-iBu is a highly potent and robustly efficacious serotonin 5-HT2A receptor agonist.[4] Its EC50Tooltip half-maximal effective concentration values are 1.3 nM for calcium mobilization and 57.5 nM for β-arrestin-2 recruitment, whereas its EmaxTooltip maximal efficacy values are 103% for calcium mobilization and 77% for β-arrestin-2 recruitment relative to serotonin.[4] The drug showed higher potency and efficacy as a serotonin 5-HT2A receptor agonist than several other 2C drugs, including 2C-NP, 2C-B, 2C-I, 2C-H, and 2C-iP, whereas its activities were more comparable to or less than those of the DOx drugs DOIB, (R)-DOB, (R)-DOI, and DOiP.[4] 2C-iBu has also been assessed and found to bind to other serotonin receptors, including the serotonin 5-HT2C, 5-HT2B, 5-HT1B, 5-HT1D, 5-HT1A, 5-HT7, and 5-HT6 receptors, in that order of affinity and with varying avidities.[4] 2C-iBu dose-dependently produces the head-twitch response (HTR), a behavioral proxy of psychedelic effects, in rodents.[4] In terms of ED50Tooltip median effective dose, 2C-iBu is about 3-fold less potent than (R)-DOI in producing the HTR.[4] According to Eleusis, it is expected to have "greatly reduced" psychoactivity or hallucinogenic effects compared to related drugs like other members of the 2C family.[6][14] The drug is effective in an allergic asthma model in rodents and showed similar potency as (R)-DOI.[4] Due to its reduced potency in producing the HTR but retained anti-inflammatory potency, 2C-iBu is expected to show greater separation between the desired anti-inflammatory and the undesired psychedelic effects in humans compared to (R)-DOI.[4] In contrast to certain other anti-inflammatory drugs like corticosteroids, serotonin 5-HT2A receptor agonists like 2C-iBu are not immunosuppressants.[14] 2C-iBu, also known as 2,5-dimethoxy-4-isobutylphenethylamine, is a phenethylamine and 2C derivative.[4][5][7] Related drugs to 2C-iBu include 2C-Bu (the butyl analogue), 2C-tBu (the tert-butyl analogue), 2C-sBu (the sec-butyl analogue), and 2C-CPM (the cyclopropylmethyl analogue).[4] In addition, 2C-iBu is related to DOx drugs such as DOIB (DOiBu).[4][15] According to Charles D. Nichols, 2,5-dimethoxyamphetamine (2,5-DMA) has potent anti-inflammatory activity with weak or no hallucinogenic effects.[7][15] Moreover, DOTFM has potent psychedelic effects with no anti-inflammatory activity.[7][16][17] Hence, it appears that the anti-inflammatory effects and psychedelic effects of serotonin 5-HT2A receptor agonists can be fully dissociated.[7] The chemical synthesis of 2C-iBu has been described.[4][1] 2C-iBu was developed as a novel anti-inflammatory drug by Charles D. Nichols and colleagues at Eleusis in the late 2010s.[7][4] They are developing it for treatment of inflammatory conditions.[2][3] Eleusis was acquired by and merged into Beckley Psytech in October 2022.[2][18][19] The drug has reached the preclinical research stage of development, but no recent development has been reported as of October 2023.[2][3] Eleusis has licensed intellectual property surrounding 2C-iBu and has patent protection for 2C-iBu.[6][4] 2C-iBu is not a controlled substance in the United States as of 2020.[6] 5-HT2A receptor § Anti-inflammatory effects ^ a b c d e f Shulgin A, Manning T, Daley PF (2011). "#60. DOM". The Shulgin Index, Volume One: Psychedelic Phenethylamines and Related Compounds. Vol. 1. Berkeley, CA: Transform Press. pp. 118–129. ISBN 978-0-9630096-3-0. OCLC 709667010. 2c-IB: [...] (15) Synthesis (from 2,5-dimethoxyisobutylbenzene) (Mueller, 2006); although this compound was reported here, a structure-search in Chemical Abstracts does not produce a CAS registry number (16) Threshold activity in humans at 5 mg orally; long duration (about 20 hours; Mueller, 2006). [...] Mueller, M. (2006) Personal communication with A.T. Shulgin. ^ a b c d e f g "ELE 02". AdisInsight. 28 October 2023. Retrieved 16 February 2025. ^ a b c d e f "Delving into the Latest Updates on ELE-02 with Synapse". Synapse. 23 January 2025. Retrieved 16 February 2025. ^ a b c d e f g h i j k l m n o p q r s WO published 2020210823, Nichols CD, Billac G, Nichols DE, "Compounds and methods for treating inflammatory disorders", published 15 October 2020 ^ a b c d Raz S (February 2020). Eleusis Drug Development Overview. LSX World Congress 2020. ^ a b c d Newvine C (8 July 2020). "Eleusis Draws on Research Into Psychedelics To Develop New Medicines for Inflammation". 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ACS Pharmacol Transl Sci. 4 (2): 488–502. doi:10.1021/acsptsci.0c00063. PMC 8033619. PMID 33860179. The nature of the 4-position substituent of phenethylamine psychedelics has been previously linked to 5-HT2 receptor selectivity as well as agonist properties at 5-HT2 receptors.40 Analysis of the 4-position demonstrated that the identity of the moiety at this position was rather flexible. Fully efficacious substitutions at the 4-position included the halogens iodine and bromine (R)-DOI (Figure 3), 2C-B (Figure 7A), methoxy (TMA-2) (Figure 7G), short-chain hydrocarbons (R)-DOM (Figure 7H), (R)-DOET) (Figure 7I), and a branched hydrocarbon (DOiBu) (Figure 7J). [...] In a comparison of PenH-AUC values determined for each drug as a proxy measure of anti-inflammatory efficacy (Figure 8A) to either EC50 or EMax for calcium mobilization downstream of 5- HT2A receptor activation (Table 1), [...] ^ Flanagan TW, Billac G, Nichols CD (2022). "Differential Regulation of Inflammatory Responses Following 5-HT 2 Receptor Activation in Pulmonary Tissues". The FASEB Journal. 36 (S1). doi:10.1096/fasebj.2022.36.S1.R2617. ISSN 0892-6638. ^ Flanagan TW, Foster TP, Galbato TE, Lum PY, Louie B, Song G, et al. (February 2024). "Serotonin-2 Receptor Agonists Produce Anti-inflammatory Effects through Functionally Selective Mechanisms That Involve the Suppression of Disease-Induced Arginase 1 Expression". ACS Pharmacology & Translational Science. 7 (2): 478–492. doi:10.1021/acsptsci.3c00297. PMC 10863441. PMID 38357283. The effects of (R)-DOTFM were examined in the head-twitch response (HTR) assay. (R)-DOTFM produced a strong HTR with a potent ED 50 of 0.60 μmol/kg. These values are equivalent to (R)-DOI, as previously determined. ^ Gunther M (26 October 2022). "What's the Future of Eleusis Therapeutics After Acquisition by Beckley Psytech?". Lucid News - Psychedelics, Consciousness Technology, and the Future of Wellness. Retrieved 17 February 2025. ^ "Beckley Psytech Strengthens Pipeline and Development Team With Acquisition of Eleusis Therapeutics Limited". Psychedelic Alpha. 24 October 2022. Retrieved 17 February 2025. 2C-IB - isomer design PODCAST 33: An interview with Dr. Charles D. Nichols (November 14, 2021) - The Hamilton Morris Podcast (discusses 2C-iBu/ELE-02 from 48:22 to 53:56) Retrieved from " 4 Pharmaceutical compound 2C-BuClinical dataOther names2,5-Dimethoxy-4-butylphenethylamine; 4-Butyl-2,5-dimethoxyphenethylamine; 2C-BUIdentifiers IUPAC name 2-(4-butyl-2,5-dimethoxyphenyl)ethan-1-amine CAS Number2888537-44-6PubChem CID172187929Chemical and physical dataFormulaC14H23NO2Molar mass237.343 g·mol−13D model (JSmol)Interactive image SMILES CCCCC1=CC(=C(C=C1OC)CCN)OC InChI InChI=1S/C14H23NO2/c1-4-5-6-11-9-14(17-3)12(7-8-15)10-13(11)16-2/h9-10H,4-8,15H2,1-3H3Key:WVGDKSDIKCPRKV-UHFFFAOYSA-N 2C-Bu, also known as 2,5-dimethoxy-4-butylphenethylamine, is a chemical compound of the phenethylamine and 2C families.[1] It has been said by Daniel Trachsel to be completely unknown.[1] 2C-Bu is the 2C analogue of the DOx derivative DOBU, which is active but does not appear to have psychedelic-type effects in either animals or humans.[1][2][3] 2C-Bu has several notable skeletal isomers, including 2C-iBu, 2C-tBu, and 2C-sBu.[1][4] 2C-iBu and 2C-tBu are both active and produce hallucinogen-type effects in animals and/or humans.[5][6][7][4] This is in spite of 2C-tBu being predicted to be inactive[4] and DOTB (the DOx analogue of 2C-tBu) being inactive as a hallucinogen in animals and humans.[2][8] 2C-iBu may have reduced hallucinogenic potency than other 2C drugs and is being developed as a potential anti-inflammatory medication.[6][7] 2C-P ^ a b c d Trachsel D, Lehmann D, Enzensperger C (2013). Phenethylamine: von der Struktur zur Funktion [Phenethylamines: From Structure to Function]. Nachtschatten-Science (in German). Solothurn: Nachtschatten-Verlag. pp. 763–764, 766–767, 771, 901. ISBN 978-3-03788-700-4. OCLC 858805226. Retrieved 29 January 2025. ^ a b Shulgin AT, Shulgin A (1991). "#63 DOBU 2,5-DIMETHOXY-4-(n)-BUTYLAMPHETAMINE". PiHKAL: A Chemical Love Story (1st ed.). Berkeley, CA: Transform Press. ISBN 978-0-9630096-0-9. OCLC 25627628. ^ Seggel MR, Yousif MY, Lyon RA, Titeler M, Roth BL, Suba EA, et al. (March 1990). "A structure-affinity study of the binding of 4-substituted analogues of 1-(2,5-dimethoxyphenyl)-2-aminopropane at 5-HT2 serotonin receptors". Journal of Medicinal Chemistry. 33 (3): 1032–1036. doi:10.1021/jm00165a023. PMID 2308135. ^ a b c Varty GB, Canal CE, Mueller TA, Hartsel JA, Tyagi R, Avery K, et al. (April 2024). "Synthesis and Structure-Activity Relationships of 2,5-Dimethoxy-4-Substituted Phenethylamines and the Discovery of CYB210010: A Potent, Orally Bioavailable and Long-Acting Serotonin 5-HT2 Receptor Agonist". J Med Chem. 67 (8): 6144–6188. doi:10.1021/acs.jmedchem.3c01961. PMID 38593423. The 4-tert-butyl group was considered as a spot for potential hydroxylation by cytochrome P450s to discover analogs with short-lasting effects. However, 2C-t-Bu was a potent agonist at the 5-HT2A receptor (Ki = 9.9 nM, EC50 = 4.2 nM) and elicited a robust HTR (Supporting Information, Table S1), providing in vivo evidence that the tert-butyl group is not rapidly metabolized to an inactive compound in mice, despite predictions. ^ Shulgin A, Manning T, Daley PF (2011). "#60. DOM". The Shulgin Index, Volume One: Psychedelic Phenethylamines and Related Compounds. Vol. 1. Berkeley, CA: Transform Press. pp. 118–129. ISBN 978-0-9630096-3-0. OCLC 709667010. 2c-IB: [...] (15) Synthesis (from 2,5-dimethoxyisobutylbenzene) (Mueller, 2006); although this compound was reported here, a structure-search in Chemical Abstracts does not produce a CAS registry number (16) Threshold activity in humans at 5 mg orally; long duration (about 20 hours; Mueller, 2006). [...] Mueller, M. (2006) Personal communication with A.T. Shulgin. ^ a b WO published 2020210823, Nichols CD, Billac G, Nichols DE, "Compounds and methods for treating inflammatory disorders", published 15 October 2020 ^ a b Raz S (February 2020). Eleusis Drug Development Overview. LSX World Congress 2020. ^ Oberlender RA (May 1989). "Stereoselective aspects of hallucinogenic drug action and drug discrimination studies of entactogens". Purdue e-Pubs. Purdue University. Retrieved 17 February 2025. Table 7. Hallucinogenic potency of 4-alkyl-2,5-dimethoxyamphetamines.a [...] DOTB: [...] Hallucinogenic Potencyb: -c. [...] c this compound has not been established as hallucinogenic. [...] Within this homologous series, optimum activity is straight chain alkyl group, two and three carbons in length 1975). The potency increases by an order of magnitude as associated with a (Shulgin and Dyer, the 4-hydrogen of 2,5-DMA is replaced by a short alkyl chain, then decreases if the chain length exceeds four carbons. In addition, the lack of hallucinogenic activity for the tertiary butyl derivative, DOTB, suggested that branching of the 4-alkyl substituent was not tolerated. [...] In a study employing 5-MeO-DMT as the training drug in rats, DOTB and DOAM were distinguishable from this hallucinogen, while 2,5-DMA and DOM were not (Glennon et al., 1981). This was consistent with the studies described above. Surprisingly, however, stimulus generalization was not observed for DOET, DOPR, and DOBU (Glennon et al., 1981a). [...] Aldous et al. (1974) noted steric restrictions on the 4-substituent in the rabbit hyperthermia model since the 4-isopropyl derivative was more potent than the 4-tert-butyl analogue, DOTB. Additional studies with DOTB, which contains a more highly hindered benzylic carbon, indicate that hallucinogen-like activity may actually be abolished in man (Shulgin and Dyer, 1975) and drastically attenuated in animals (Glennon et al., 1982). 2C-BU - Isomer Design Retrieved from " 5 Pharmaceutical compound 2C-tBuClinical dataOther names2,5-Dimethoxy-4-tert-butylphenethylamine; 4-tert-Butyl-2,5-dimethoxyphenethylamine; 2C-TBU; 2C-t-BuDrug classSerotonin receptor agonist; Serotonergic psychedelic; HallucinogenIdentifiers IUPAC name 2-(4-tert-butyl-2,5-dimethoxyphenyl)ethanamine PubChem CID117347542ChemSpider76370331Chemical and physical dataFormulaC14H23NO2Molar mass237.343 g·mol−13D model (JSmol)Interactive image SMILES CC(C)(C)C1=C(C=C(C(=C1)OC)CCN)OC InChI InChI=1S/C14H23NO2/c1-14(2,3)11-9-12(16-4)10(6-7-15)8-13(11)17-5/h8-9H,6-7,15H2,1-5H3Key:MHNFTGKRRUDUST-UHFFFAOYSA-N 2C-tBu, or 2C-t-Bu, also known as 4-tert-butyl-2,5-dimethoxyphenethylamine, is a serotonin receptor agonist and putative serotonergic psychedelic of the phenethylamine and 2C families.[1][2] It is a potent serotonin 5-HT2A receptor agonist (Ki = 9.9–35 nM, EC50Tooltip half-maximal effective concentration = 4.2 nM) and also binds to the serotonin 5-HT2C receptor (Ki = 7–24 nM).[1][2] The drug produces a robust head-twitch response, a behavioral proxy of psychedelic effects, in rodents.[2] It also produces hyperlocomotion in rodents.[2] The active dosage in humans is >5 mg per Daniel Trachsel and its duration is unknown.[1] Initial tests with 7 mg and with 10 mg (as 5 mg plus 5 mg 2 hours apart) produced no psychedelic effects in humans, but instead induced a pronounced and long-lasting tiredness.[1] It was hypothesized that 2C-tBu might be a serotonin 5-HT2A receptor antagonist and might thereby be hypnotic,[1] but it was instead shown to be an agonist in subsequent studies.[2] 2C-Bu 2C-iBu DOTB ^ a b c d e Trachsel D, Lehmann D, Enzensperger C (2013). Phenethylamine: von der Struktur zur Funktion [Phenethylamines: From Structure to Function]. Nachtschatten-Science (in German). Solothurn: Nachtschatten-Verlag. pp. 766–767, 771, 901. ISBN 978-3-03788-700-4. OCLC 858805226. Retrieved 29 January 2025. ^ a b c d e Varty GB, Canal CE, Mueller TA, Hartsel JA, Tyagi R, Avery K, et al. (April 2024). "Synthesis and Structure-Activity Relationships of 2,5-Dimethoxy-4-Substituted Phenethylamines and the Discovery of CYB210010: A Potent, Orally Bioavailable and Long-Acting Serotonin 5-HT2 Receptor Agonist". Journal of Medicinal Chemistry. 67 (8): 6144–6188. doi:10.1021/acs.jmedchem.3c01961. PMID 38593423. The 4-tert-butyl group was considered as a spot for potential hydroxylation by cytochrome P450s to discover analogs with short-lasting effects. However, 2C-t-Bu was a potent agonist at the 5-HT2A receptor (Ki = 9.9 nM, EC50 = 4.2 nM) and elicited a robust HTR (Supporting Information, Table S1), providing in vivo evidence that the tert-butyl group is not rapidly metabolized to an inactive compound in mice, despite predictions. 2C-TBU - Isomer Design This psychoactive drug-related article is a stub. You can help Wikipedia by expanding it.vte Retrieved from " 6 The following pages link to 2C-tBu External tools (link count transclusion count sorted list) · See help page for transcluding these entries Showing 50 items. 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