Discover Plus™

Multi-Drug Rapid Test Cup (Oral Fluid) Package Insert

A rapid test for the simultaneous, qualitative detection of multiple drugs and drug metabolites in human oral fluid. For forensic use only.

【INTENDED USE】

The Multi-Drug Rapid Test Cup for AMP/MET/COC/OPI/MOP/THC/PCP/MTD/MDMA/BZO/OXY/COT/K2/KET/BAR/BUP/6-MAM/TML/FYL/CFYL/MDPV/α-PVP/LSD/PPX/MQL/CAR/EDDP/ABP(K3)/UR-144(K4)/ZOP/GAB/PGB is a lateral flow chromatographic immunoassay for the qualitative detection of multiple drugs and drug metabolites in oral fluid at the following cut-off concentrations:

Test	Calibrator	Cut-off (ng/mL)
Amphetamine (AMP)	d-Amphetamine	50
Methamphetamine (MET)	d-Methamphetamine	50
Marijuana (THC)	11-nor-Δ^9 -THC-9 COOH	50/40/25/20/12
Phencyclidine (PCP)	Phencyclidine	10
Cocaine (COC)	Benzoylecgonine	20
Opiates (OPI/MOP)	Morphine	40/25
Methadone (MTD)	Methadone	30
Methylenedioxymethamphetamine (MDMA)	d,l-Methylenedioxymethamphetamine	50
Oxycodone (OXY)	Oxycodone	50/20
Cotinine(COT)	Cotinine	20
Benzodiazepines (BZO)	Oxazepam	50/30/20/10
Synthetic Marijuana (K2)	JWH -018, JWH- 073	25
Ketamine (KET)	Ketamine	50
Barbiturates (BAR)	Secobarbital	50
Buprenorphine (BUP)	Buprenorphine	10/5
Tramadol (TML)	Tramadol	30
6-Monoacetylmorphine (6-MAM)	6-Monoacetylmorphine	10
Fentanyl (FYL)	Fentanyl	50/20/10
Car fentanyl (CFYL)	Carfentanyl	50
3, 4-methylenedioxypyrovalerone (MDPV)	3, 4-methylenedioxypyrovalerone	300
alpha-Pyrrolidinovalerophenone (α-PVP)	alpha-Pyrrolidinovalerophenone	300
Lysergic Acid Diethylamide (LSD)	Lysergic Acid Diethylamide	10
Propoxyphene (PPX)	d-propoxyphene	50
Methaqualone (MQL)	Methaqualone	300
Carisoprodol (CAR)	Carisoprodol	300
2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine (EDDP)	2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine	50
AB-PINACA (ABP/K3)	AB-PINACA	10
UR-144/K4	UR-144 5-Pentanoic acid	25
Zopiclone (ZOP)	Zopiclone	50
Gabapentin(GAB)	Gabapentin	2000
Pregabalin (PGB)	Pregabalin	500

This assay provides only a preliminary test result. A more specific alternate chemical method must be used in order to obtain a confirmed analytical result. Gas Chromatography/Mass Spectrometry (GC/MS) is the preferred confirmatory methods. Professional judgment should be applied to any drug of abuse test result, particularly when preliminary positive results are indicated.

【SUMMARY】

The Multi-Drug Rapid Test Cup for multiple drugs and their metabolites is a rapid, oral fluid screening test that can be performed without the use of an instrument. The test utilizes monoclonal antibodies to selectively detect elevated levels of specific drugs in human oral fluid.

Amphetamine (AMP)

Amphetamine is a sympathomimetic amine with therapeutic indications. The drug is often self-administered by nasal inhalation or oral ingestion. Depending on the route of administration, amphetamine can be detected in oral fluid as early as 5-10 minutes following use. [1] Amphetamine can be detected in oral fluid for up to 72 hours after use. [1]

Methamphetamine (MET)

Methamphetamine is a potent stimulant chemically related to amphetamine but with greater CNS stimulation properties. The drug is often self-administered by nasal inhalation, smoking or oral ingestion. Depending on the route of administration, methamphetamine can be detected in oral fluid as early as 5-10 minutes following use.[1] Methamphetamine can be detected in oral fluid for up to 72 hours after use. [1]

Cocaine (COC)

Cocaine is a potent central nervous system (CNS) stimulant and a local anesthetic derived from the coca plant (erythroxylum coca). The drug is often self-administered by nasal inhalation, intravenous injection and free-base smoking. Depending on the route of administration, cocaine and metabolites benzoylecgonine and ecgonine methyl ester can be detected in oral fluid as early as 5-10 minutes following use.[1] Cocaine and benzoylecgonine can be detected in oral fluid for up to 24 hours after use.[1]

Opiates (OPI/MOP)

The drug class opiates refers to any drug that is derived from the opium poppy, including naturally occurring compounds such as morphine and codeine and semi-synthetic drugs such as heroin. Opiates act to control pain by depressing the central nervous system. The drugs demonstrate addictive properties when used for sustained periods of time; symptoms of withdrawal may include sweating, shaking, nausea and irritability. Opiates can be taken orally or by injection routes including intravenous, intramuscular and subcutaneous; illegal users may also take the intravenously or by nasal inhalation. Using the OPI test, codeine can be detected in the oral fluid within 1 hour following a single oral dose and can remain detectable for 7-21 hours after the dose.[1] Heroin metabolite 6-monoacetylmorphine (6-MAM) is found more prevalently in excreted unmetabolized, and is also the major metabolic product of codeine and heroin.[2]

Marijuana (THC)

11-nor-Δ^9-tetrahydrocannabinol-9-carboxylic acid (Δ^9-THC-COOH), the metabolite of THC (Δ^9-tetrahydrocannabinol), is detectable in oral fluid shortly after use. The detection of the drug is thought to be primarily due to the direct exposure of the drug to the mouth (oral and smoking administrations) and the subsequent sequestering of the drug in the buccal cavity.[3] Historical studies have shown a window of detection for THC in oral fluid of up to 14 hours after drug use.[3]

Phencyclidine (PCP)

Phencyclidine, the hallucinogen commonly referred to as Angel Dust, can be detected in oral fluid as a result of the exchange of the drug between the circulatory system and the oral cavity. In a paired serum and oral fluid sample collection of 100 patients in an Emergency Department, PCP was detected in the oral fluid of 79 patients at levels as low as 2 ng/mL and as high as 600 ng/mL.[4]

Methadone (MTD)

Methadone is a narcotic analgesic prescribed for the management of moderate to severe pain and for the treatment of opiate dependence (heroin, Vicodin, Percocet, morphine).

Methadone is a long acting pain reliever producing effects that last from twelve to forty-eight hours. Ideally, methadone frees the client from the pressures of obtaining illegal heroin, from the dangers of injection, and from the emotional roller coaster that most opiates produce. Methadone, if taken for long periods and at large doses, can lead to a very long withdrawal period. The withdrawals from methadone are more prolonged and troublesome than those provoked by heroin cessation, yet the substitution and phased removal of methadone is an acceptable method of detoxification for patients and therapists.[1]

Methylenedioxymethamphetamine (MDMA)

Methylenedioxymethamphetamine (ecstasy) is a designer drug first synthesized in 1914 by a German drug company for the treatment of obesity. Those who take the drug frequently report adverse effects, such as increased muscle tension and sweating. MDMA is not clearly a stimulant, although it has, in common with amphetamine drugs, a capacity to increase blood pressure and heart rate. MDMA does produce some perceptual changes in the form of increased sensitivity to light, difficulty in focusing, and blurred vision in some users. Its mechanism of action is thought to be via release of the neurotransmitter serotonin. MDMA may also release dopamine, although the general opinion is that this is a secondary effect of the drug (Nichols and Oberlender, 1990). The most pervasive effect of MDMA, occurring in virtually all people who took a reasonable dose of the drug, was to produce a clenching of the jaws.[1]

Oxycodone (OXY)

Oxycodone is a semi-synthetic opioid with a structural similarity to codeine. The drug is manufactured by modifying the baine, an alkaloid found in the opium poppy. Oxycodone, like all opiate agonists, provides pain relief by acting on opioid receptors in the spinal cord, brain, and possibly directly in the affected tissues. Oxycodone is prescribed for the relief of moderate to high pain under the well-known pharmaceutical trade names of OxyContin®, Tylox®, Percodan® and Percocet®. While Tylox®, Percodan® and Percocet® contain only small doses of oxycodone hydrochloride combined with other analgesics such as acetaminophen or aspirin, OxyContin consists solely of oxycodone hydrochloride in a time-release form. Oxycodone is known to metabolize by demethylation into oxymorphone and noroxycodone.

Cotinine (COT)

Cotinine is the first-stage metabolite of nicotine, a toxic alkaloid that produces stimulation of the autonomic ganglia and central nervous system when in humans. Nicotine is a drug to which virtually every member of a tobacco-smoking society is exposed whether through direct contact or second-hand inhalation. In addition to tobacco, nicotine is also commercially available as the active ingredient in smoking replacement therapies such as nicotine gum, transdermal patches and nasal sprays.

Although nicotine is excreted in oral fluid, the relatively short half-life of the drug makes it an unreliable maker for tobacco use. Cotinine, however, demonstrates a substantially longer half-life than nicotine bears a high correlation with plasma cotinine levels and has been found to be the best maker for smoking status compared with oral fluid nicotine measurement, breath carbon monoxide testing and plasma thiocyanate testing. The window of detection for cotinine in oral fluid test is expected to be up to 1-2 days after nicotine use.

Benzodiazepines (BZO)

Benzodiazepines are medications that are frequently prescribed for the symptomatic treatment of anxiety and sleep disorders. They produce their effects via specific receptors involving a neurochemical called gamma aminobutyric acid (GABA). Because they are safer and more effective, Benzodiazepines have replaced Barbiturates in the treatment of both anxiety and insomnia. Benzodiazepines are also used as sedatives before some surgical and medical procedures, and for the treatment of seizure disorders and alcohol withdrawal. Risk of physical dependence increases if Benzodiazepines are taken regularly (e.g., daily) for more than a few months, especially at higher than normal doses. Stopping abruptly can bring on such symptoms as trouble sleeping, gastrointestinal upset, feeling unwell, loss of appetite, sweating, trembling, weakness, anxiety and changes in perception.[1]

Synthetic Marijuana (K2)

Synthetic Marijuana or K2 is a psychoactive herbal and chemical product that, when consumed, mimics the effects of Marijuana. It is best known by the brand names K2 and Spice, both of which have largely become genericized trademarks used to refer to any synthetic Marijuana product. The studies suggest that synthetic marijuana intoxication is associated with acute psychosis, worsening of previously stable psychotic disorders, and also may have the ability to trigger a chronic (long-term) psychotic disorder among vulnerable individuals such as those with a family history of mental illness.[6]

Elevated levels of oral fluid metabolites are found within hours of exposure and remain detectable window up to 24-48 hours after smoking (depending on usage/dosage).

Ketamine (KET)

Ketamine is a dissociative anesthetic developed in 1963 to replace PCP (Phencyclidine). While Ketamine is still used in human anesthesia and veterinary medicine, it is becoming increasingly abused as a street drug. Ketamine is molecularly similar to PCP and thus creates similar effects including numbness, loss of coordination, sense of invulnerability, muscle rigidity, aggressive / violent behavior, slurred or blocked speech, exaggerated sense of strength, and a blank stare. There is depression of respiratory function but not of the central nervous system, and cardiovascular function is maintained.[7] The effects of Ketamine generally last 4-6 hours following use.

Barbiturates (BAR)

Barbiturates are CNS depressants. They are used therapeutically as sedatives, hypnotics, and anticonvulsants barbiturates are almost always taken orally as capsules or tablets. The effects resemble those of intoxication with alcohol. Chronic use of barbiturates leads to tolerance and physical dependence.[8]

Short-acting barbiturates taken at 400 mg/day for 2-3 months can produce a clinically significant degree of physical dependence. Withdrawal symptoms experienced during periods of drug abstinence can be severe enough to cause death.

The approximate detection time limits for barbiturates are:

Short acting (e.g. Secobarbital)	100 mg PO (oral)	4.5 days
Long acting (e.g. Phenobarbital)	400 mg PO (oral)	7 days[2]

Buprenorphine (BUP)

Buprenorphine is a potent analgesic often used in the treatment of opioid addiction. The drug is sold under the trade names Subutex™, Buprenex™, Temgesic™ and Suboxone™, which contain Buprenorphine HCl alone or in combination with Naloxone HCl. Therapeutically, Buprenorphine is used as a substitution treatment for opioid addicts. Substitution treatment is a form of medical care offered to opiate addicts (primarily heroin addicts) based on a similar or identical substance to the drug normally used. In substitution therapy, Buprenorphine is as effective as Methadone but demonstrates a lower level of physical dependence. The elimination half-life of buprenorphine is 20–73 hours (mean 37). Substantial abuse of Buprenorphine has also been reported in many countries where various forms of the drug are available. The drug has been diverted from legitimate channels through theft, doctor shopping and fraudulent prescriptions, and been abused via intravenous, sublingual, intranasal and inhalation routes

Tramadol (TML)

Tramadol (TML) is a quasi-narcotic analgesic used in the treatment of moderate to severe pain. It is a synthetic analog of codeine, but has a low binding affinity to the mu-opioid receptors. Large doses of tramadol can develop tolerance and physiological dependency and lead to its abuse. Tramadol is extensively metabolized after oral administration. Approximately 30% of the dose is excreted in oral fluid as unchanged drug, whereas 60% is excreted as metabolites. The major pathways appear to be N- and O- demethylation, glucoronidation or sulfation in the liver.

6-Monoacetylmorphine (6-MAM)

6-Monoacetylmorphine (6-MAM) or 6-acetylmorphine (6-AM) is one of three active metabolites of heroin (diacetylmorphine), the others being morphine and the much less active 3-monoacetylmorphine (3-MAM). 6-MAM is rapidly created from heroin in the body, and then is either metabolized into morphine or excreted in the oral fluid. 6-MAM remains in the oral fluid for no more than 24 hours. So a oral fluid specimen must be collected soon after the last heroin use, but the presence of 6-MAM guarantees that heroin was in fact used as recently as within the last day. 6-MAM is naturally found in the brain, [5] but in such small quantities that detection of this compound in oral fluid virtually guarantees that heroin has recently been consumed.

Fentanyl (FYL)

Fentanyl, belongs to powerful narcotics analgesics, and is a special opiates receptor stimulant. Fentanyl is one of the varieties that been listed in management of United Nations “Single Convention of narcotic drug in 1961”. Among the opiates agents that under international control, fentanyl is one of the most commonly used to cure moderate to severe pain.[5] After continuous injection of fentanyl, the sufferer will have the performance of protracted opioid abstinence syndrome, such as ataxia and irritability etc,[6,7] which presents the addiction after taking fentanyl in a long time. Compared with drug addicts of amphetamine, drug addicts who take fentanyl mainly have got the possibility of higher infection rate of HIV, more dangerous injection behavior and more lifelong medication overdose.[8]

Carfentanyl (CFYL)

Carfentanyl is an analog of the synthetic opioid analgesic fentanyl. It is 10,000 times more potent than morphine, making it among the most potent commercially used opioids. Carfentanyl was first synthesized in 1974.[9] It is marketed under the trade name Wildnil as a general anaesthetic agent for large animals.[10] Side effects of carfentanyl are similar to those of fentanyl, which include itching, nausea and respiratory depression, which can be life-threatening.[11] Carfentanyl is classified as Schedule II under the Controlled Substances Act in the United States with a DEA ACSCN of 9743.

3, 4-methylenedioxypyrovalerone (MDPV)

3, 4-methylenedioxypyrovalerone (MDPV) is a psychoactive recreational drug with stimulant properties which acts as a norepinephrine-dopamine reuptake inhibitor (NDRI). It was first developed in the 1960s by a team at Boehringer Ingelheim[1]. MDPV remained an obscure stimulant until around 2004 when it was reportedly sold as a designer drug. Products labeled as bath salts containing MDPV were previously sold as recreational drugs in gas stations and convenience stores in the United States, similar to the marketing for Spice and K2 as incense.

MDPV is the 3,4-methylenedioxy ring-substituted analog of the compound pyrovalerone, developed in the 1960s, which has been used for the treatment of chronic fatigue and as an anorectic, but caused problems of abuse and dependence. However, despite its structural similarity, the effects of MDPV bear little resemblance to other methylenedioxy phenylalkylamine derivatives such as 3,4-methylenedioxy-N-methylamphetamine (MDMA), instead producing primarily stimulant effects with only mild entactogenic qualities.[12]

MDPV undergoes CYP450 2D6, 2C19, 1A2, and COMT phase 1 metabolism (liver) into

methylcatechol and pyrrolidine, which in turn are glucuronated (uridine 5'-diphospho-glucuronosyl-transferase) allowing it to be excreted by the kidneys, with only a small fraction of the metabolites being excreted into the stools.[13] No free pyrrolidine will be detected in the oral fluid.

alpha-Pyrrolidinovalerophenone (α-PVP)

alpha-Pyrrolidinovalerophenone (also known as α-PVP, A-PVP, alpha-PVP, and Flakka) is a synthetic stimulant substance of the cathinone and pyrrolidine chemical classes. α-PVP may be quantified in blood, plasma or urine to confirm a diagnosis of poisoning in hospitalized patients or to provide evidence in a medicolegal death investigation.[14] It generally comes in the form of either a crystalline powder or crystallized shards which users can ingest to produce powerful but short-lived euphoric stimulant effects which are comparable to those of methamphetamine and cocaine when insufflated or vaporized. α-PVP has been reported to be the cause, or a significant contributory cause of death in suicides and overdoses caused by combinations of drugs.[15] It has also been linked to at least one death where it was combined with pentedrone and caused heart failure.

Lysergic Acid Diethylamide (LSD)

Lysergic acid diethylamide (LSD) is a white powder or a clear, colorless liquid. LSD is manufactured from lysergic acid which occurs naturally in the ergot fungus that grows on wheat and rye. It is a Schedule I controlled substance, available in liquid, powder, tablet (microdots), and capsule form. LSD is recreationally used as a hallucinogen for its ability to alter human perception and mood. LSD is primarily used by oral administration, but can be inhaled, injected, and transdermally applied. LSD is a non-selective 5-HT agonist, may exert its hallucinogenic effect by interacting with 5-HT 2Areceptors as a partial agonist and modulating the NMDA receptor-mediated sensory, perceptual, affective and cognitive processes. LSD mimics 5-HT at 5-HT 1A receptors, producing a marked slowing of the firing rate of serotonergic neurons. LSD has a plasma half-life of 2.5-4 hours. Metabolites of LSD include N-desmethyl-LSD, hydroxy-LSD, 2-oxo-LSD, and 2-oxo-3-hydroxy-LSD. These metabolites are all inactive.

Propoxyphene (PPX)

Propoxyphene (PPX) is a narcotic analgesic compound bearing structural similarity to methadone. As an analgesic, Propoxyphene can be from 50-75% as potent as oral codeine. DarvocetTM, one of the most common brand names for the drug, contains 50-100 mg of Propoxyphene napsylate and 325-650 mg of acetaminophen. Peak plasma concentrations of Propoxyphene are achieved from 1 to 2 hours post dose. In the case of overdose, Propoxyphene blood concentrations can reach significantly higher levels. In humans, Propoxyphene is metabolized by N-demethylation to yield Norpropoxyphene. Norpropoxyphene has a longer half-life (30 to 36 hours) than parent Propoxyphene (6 to 12 hours). The accumulation of Norpropoxyphene seen with repeated doses may be largely responsible for resultant toxicity.

Methaqualone (MQL)

Methaqualone (Quaalude, Sopor) is a quinazoline derivative that was first synthesized in 1951 and found clinically effective as a sedative and hypnotic in 1956.[2] It soon gained popularity as a drug of abuse and in 1984 was removed from the US market due to extensive misuse. It is occasionally encountered in illicit form, and is also available in Europeon countries in combination with diphenhydramine (Mandrax). Methaqualone is extensively metabolized in vivo principally by hydroxylation at every possible position on the molecule. At least 12 metabolites have been identified in the Oral Fluid.

Carisoprodol (CAR)

Carisoprodol, marketed under the brand name Soma among others, is a medication used for musculoskeletal pain. Use is only approved for up to three weeks. Effects generally begin within half an hour and last for up to six hours. It is taken by mouth.

Common side effects include headache, dizziness, and sleepiness. Serious side effect may include addiction, allergic reactions, and seizures. In people with a sulfa allergy certain formulations may result in problems. Safety during pregnancy and breastfeeding is not clear. Meprobamate and other muscle-relaxing drugs often were subjects of misuse in the 1950s and 60s.[16,17] Overdose cases were reported as early as 1957, and have been reported on several occasions since then.[18,19,20,21,22,23]

Carisoprodol is metabolized by the liver and excreted by the kidneys so this drug must be used with caution with patients that have impaired hepatic or renal function. Because of potential for more severe side effects, this drug is on the list to avoid for elderly people.

2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine (EDDP)

Methadone is an unusual drug in that its primary metabolites (EDDP and EMDP) are cyclic in structure, making them very difficult to detect using immunoassays targeted to the native compound.

Exacerbating this problem, there is a subsection of the population classified as “extensive metabolizers” of methadone. In these individuals, a specimen may not contain enough parent methadone to yield a positive drug screen even if the individual is in compliance with their methadone maintenance. EDDP represents a better marker for methadone maintenance than unmetabolized methadone.

AB-PINACA (ABP/K3)

AB-PINACA is a compound that was first identified as a component of synthetic cannabis products in Japan in 2012. It was originally developed by Pfizer in 2009 as an analgesic medication.[24] AB-PINACA acts as a potent agonist for the CB1 receptor (Ki = 2.87 nM, EC50 = 1.2 nM) and CB2 receptor (Ki = 0.88 nM, EC50 = 2.5 nM) and fully substitutes for Δ^9-THC in rat discrimination studies, while being 1.5x more potent.[25,26]

UR-144/K4

UR-144 is a synthetic cannabinoid receptor agonist (SCRA) and has affinity for CB1 and CB2 receptors. It has a high selectivity for the CB2-receptors.

UR-144 is a psychoactive substance and has effects similar to delta-9-tetrahydrocannabinol (THC), though slightly less potent than THC. UR-144 has been detected in herbal products marketed under a variety of names.

In mice, UR-144 is moderately potent in reducing in a time- and dose-dependent manner the locomotor activity (ID50-value 7.8 mg/kg), induces an anti-nociceptive effect, and decreases rectal temperature and ring immobility with potencies several-fold greater than THC. In mice, UR-144 substituted for THC in a THC discrimination study (ED50-value 7.1 to 7.4 µmol/kg intra-peritoneal), an effect antagonized by rimonabant.

Zopiclone (ZOP)

Zopiclone is a nonbenzodiazepine hypnotic agent used in the treatment of insomnia. It is a cyclopyrrolone, which increases the normal transmission of the neurotransmitter gamma-aminobutyric acid in the central nervous system, as benzodiazepines do, but in a different way. Zopiclone is indicated for the short-term treatment of insomnia where sleep initiation or sleep maintenance are prominent symptoms. Long-term use is not recommended, as tolerance, dependence, and addiction can occur with prolonged use. Zopiclone is partly extensively metabolized in the liver to form an active N-demethylated derivative (N-desmethylzopiclone) and an inactive zopiclone-N-oxide.

In urine, the N-demethyl and N-oxide metabolites account for 30% of the initial dose. Between 7 and 10% of zopiclone is recovered from the urine, indicating extensive metabolism of the drug before excretion. The terminal elimination half-life of zopiclone ranges from 3.5 to 6.5 hours (5 hours on average). Time to peak plasma concentration is 1 - 2 h, the absorption rate constant is 1.3 h-1 and maximum plasma concentration after administration of 7.5 mg is 131µg/L.

Zopiclone may be measured in blood, plasma, or urine by chromatographic methods. Plasma concentrations are typically less than 100µg/L during therapeutic use, but frequently exceed 100µg/L in automotive vehicle operators arrested for impaired driving ability and may exceed 1000µg/L in acutely poisoned patients. Post mortem blood concentrations are usually in a range of 0.4-3.9 mg/L in victims of fatal acute overdose.

Gabapentin (GAB)

Gabapentin, sold under the brand name Neurontin among others, is a medication which is used to treat epilepsy (specifically partial seizures), neuropathic pain, hot flashes, and restless legs syndrome.

Common side effects of gabapentin include sleepiness and dizziness. Serious side effects include an increased risk of suicide, aggressive behavior, and drug reaction with eosinophilia and systemic symptoms. In 2009 the U.S. Food and Drug Administration issued a warning of an increased risk of suicidal thoughts and behaviors in patients taking some anticonvulsant drugs, including gabapentin, modifying the packaging inserts to reflect this.

The oral bioavailability of gabapentin enacarbil (as gabapentin) is greater than or equal to 68%, across all doses assessed (up to 2,800 mg), with a mean of approximately 75%.Gabapentin undergoes little or no metabolism.The Tmax of the instant-release (IR) formulation of gabapentin enacarbil (as active gabapentin) is about 2.1 to 2.6 hours across all doses (350–2,800 mg) with single administration and 1.6 to 1.9 hours across all doses (350–2,100 mg) with repeated administration.

Pregabalin (PGB)

Pregabalin, also known as β-isobutyl-γ-amino butyric acid (beta-isobutyl-GABA), is a medication used to treat epilepsy, neuropathic pain, fibromyalgia, and generalized anxiety disorder.Common side effects include: sleepiness, confusion, trouble with memory, poor coordination, dry mouth, problem with vision, and weight gain. Potentially serious side effects include angioedema, drug misuse, and an increased suicide risk.

Pregabalin is eliminated from the systemic circulation primarily by renal excretion as unchanged drug. The Pregabalin is predominantly excreted unchanged in urine (≥ 98%) . Pregabalin mean elimination half-life is 6.3 hours. 50% would be expected to have negative urine specimens within 3 days and a total of 5 days would be needed to achieve negative urine specimens in the subject with the maximum urinary concentration measured.

【PRINCIPLE】

The Multi-Drug Rapid Test Cup is an immunoassay based on the principle of competitive binding. Drugs that may be present in the oral fluid specimen compete against their respective drug conjugate for binding sites on their specific antibody.

During testing, a portion of the oral fluid specimen migrates upward by capillary action. A drug, if present in the oral fluid 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. The presence of drug above the cut-off concentration in the oral fluid specimen will saturate all the binding sites of the antibody. Therefore, the colored line will not form in the test line region.

A drug-positive oral fluid specimen will not generate a colored line in the specific test line region of the strip because of drug competition, while a drug-negative oral fluid specimen will generate a line in the test line region because of the absence of drug competition.

To serve as a procedural control, a colored line will always appear at the control line region, indicating that proper volume of specimen has been added and membrane wicking has occurred.

【REAGENTS】

The test contains membrane strips coated with drug-protein conjugates (purified bovine albumin) on the test line, a goat polyclonal antibody against gold-protein conjugate at the control line, and a dye pad which contains colloidal gold particles coated with mouse monoclonal antibody specific to corresponding drug.

【PRECAUTIONS】

- Do not use after the expiration date.
- The test should remain in the sealed pouch until use.
- Oral fluid is not classified as biological hazard unless derived from a dental procedure.
- The used swab and cup should be discarded according to local regulations.

【STORAGE AND STABILITY】

Store as packaged in the sealed pouch at 2-30°C. The test is stable through the expiration date printed on the sealed pouch. The test cup must remain in the sealed pouch until use. **DO NOT FREEZE**. Do not use beyond the expiration date.

【SPECIMEN COLLECTION AND PREPARATION】

The oral fluid specimen should be collected using the swab provided with the kit. Follow the detailed Directions for Use below. No other cup should be used with this assay. Oral fluid collected at any time of the day may be used.

【MATERIALS】

Materials Provided

- Test Cups
- Package Insert
- Sterile Swabs
- Procedure Card

Materials Required but Not Provided

- Timer

【DIRECTIONS FOR USE】

Allow the test cup, specimen, swab and/or controls to reach room temperature (15-30°C) prior to testing. Instruct the donor to not place anything in the mouth including food, drink, gum or tobacco products for at least 10 minutes prior to collection.

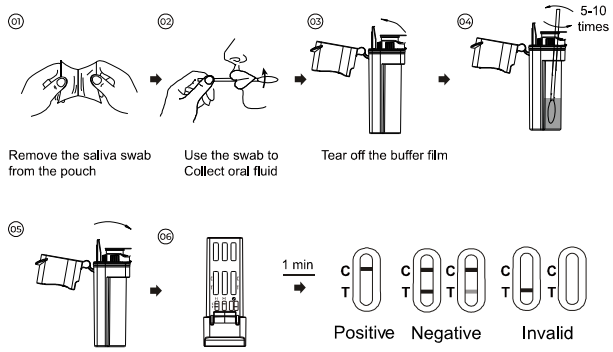
1. Remove the swab and the cup from the sealed pouch and collect oral fluid specimen.

Important: Put the swab into the mouth, and wipe it repeatedly on the inner cheeks **10-15 times** until the swab tip becomes fully saturated.

2. Open the cap of the cup and tear off the buffer film, put the collected swab into the bottom of buffer tube, rotate **5-10 times** and remove.
3. Close the cap, invert the test cup and place it on a clean and level surface. Wait for the flow to appear in test windows and start a timer.

If the specimen does not migrate in the test cup even after 1 minute, please rotate the cup 4-5 times.

4. **The result should be read at 1 minute,** don’t interpret the results after 5 minutes.



【INTERPRETATION OF RESULTS】

(Please refer to the previous illustration)

NEGATIVE:* A colored line appears in the control region (C) and another colored line appears in the test region (T). This negative result means that the concentration in the oral fluid sample are below the designated cut-off levels for a particular drug tested.

***NOTE:** The shade of the colored lines(s) in the Test region (T) may vary. The result should be considered negative whenever there is even a faint line.

POSITIVE:* A colored line appears in the control region (C) and no line appears in the test region (T). The positive result means that the drug concentration in the oral fluid sample is greater than the designated cut-off for a specific drug.

INVALID:* No line appears in the control region (C). Insufficient specimen volume or incorrect procedural techniques are the most likely reasons for Control line failure. Read the directions again and repeat the test with a new test. If the result is still invalid, contact your manufacturer.

【QUALITY CONTROL】

A procedural control is included in the test. A colored line appearing in the control region (C) is considered an internal procedural control. It confirms sufficient specimen volume, adequate membrane wicking and correct procedural technique.

【LIMITATIONS】

1. The Multi-Drug Rapid Test Cup provides only a qualitative, preliminary result. A secondary analytical method must be used to obtain a confirmed result. Gas Chromatography/Mass Sctrometry (GC/MS) is preferred confirmatory methods.[28]
2. A positive test result does not indicate the concentration of drug in the specimen or the route of administration.
3. A negative result may not necessarily indicate a drug-free specimen. Drug may be present in the specimen below the cutoff level of the assay.

【PERFORMANCE CHARACTERISTICS】

Analytical Sensitivity

A Phosphate-buffered saline (PBS) pool was spiked with drugs to target concentrations of ± 50% cut-off, ± 25% cut-off and 300% cut-off and tested with the Multi-Drug Rapid Test Cup. The results are summarized below.

Drug Concentration Cut-off Range	AMP 50		MET 50		THC 12		COT 20		PCP 10		FYL 50		COC 20		THC 25	
	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
0%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-50%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-25%	27	3	28	2	27	3	25	5	25	5	27	3	27	3	27	3
Cut-off	15	15	16	14	12	18	20	10	14	16	15	15	15	15	12	18
+25%	7	23	6	24	8	22	7	23	10	20	8	22	8	22	8	22
+50%	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30

3X	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30

Drug Concentration Cut-off Range	TML 30		BZO 20		FYL 20		CFYL 50		MDPV 300		α-PVP 300		KET 50		6-MAM 10		GAB 2000	
	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
0%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-50%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-25%	27	3	25	5	26	4	25	5	27	3	26	4	26	4	28	2	28	2
Cut-off	13	17	13	17	15	15	15	15	20	10	19	11	18	12	20	10	16	14
+25%	7	23	4	26	3	27	7	23	4	26	6	24	8	22	2	28	3	27
+50%	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30
3X	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30

Drug Concentration Cut-off Range	OPI/MOP 40		K2 25		MTD 30		OXY 20		MDMA 50		BZO 50		BAR 50		PGB 500		OPI/MOP 25	
	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
0%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-50%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-25%	27	3	26	4	25	5	27	3	26	4	25	5	25	5	25	5	27	3
Cut-off	13	17	15	15	15	15	20	10	19	11	13	17	18	12	15	15	13	17
+25%	7	23	3	27	7	23	4	26	6	24	4	26	8	22	6	24	7	23
+50%	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30
3X	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30

Drug Concentration Cut-off Range	THC 20		THC 50		OXY 50		LSD 10		BZO 30		BZO 10		BUP 10		PPX 50	
	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
0%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-50%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-25%	27	3	27	3	27	3	26	4	25	5	26	4	26	4	26	4
Cut-off	12	18	14	16	20	10	16	14	13	17	19	11	14	16	16	14
+25%	8	22	9	21	4	26	7	23	4	26	6	24	10	20	6	24
+50%	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30
3X	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30

Drug Concentration Cut-off Range	MQL 300		THC 40		CAR 300		EDDP 50		ABP/K3 10		UR-144/ K4 25		ZOP 50		BUP 5		FYL 10	
	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+	-	+
0%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-50%	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0
-25%	27	3	27	3	28	2	27	3	25	5	28	2	27	3	26	4	26	4
Cut-off	15	15	14	16	16	14	14	16	15	15	15	15	17	13	14	16	15	15
+25%	4	26	9	21	3	27	4	26	4	26	3	27	4	26	10	20	3	27
+50%	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30
3X	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30	0	30

Analytical Specificity

The following table lists the concentration of compounds (ng/mL) above which the Multi-Drug Rapid Test Cup for identified positive results at a read time of 1 minute.

Compound	ng/mL	Compound	ng/mL
Amphetamine (AMP 50)			
d-Amphetamine	50	p-Hydroxyamphetamine	100
d/l-Amphetamine	100	(+)3,4-Methylenedioxyamphetamine (MDA)	100
ß-Phenylethylamine	25,000	l-Amphetamine	25,000
Tryptamine	12,500	Methoxyphenamine	12,500
Methamphetamine (MET 50)			
d-Methamphetamine	50	l-Phenylephrine (R)-(-)-Phenylephrine	6,250
Fenfluramine	60,000	Procaine	2,000
p-Hydroxymethamphetamine	400	(1R,2S) - (-) Ephedrine	400
Methoxyphenamine	25,000	Ephedrine	400
Mephentermine	1,500	Benzphetamine	25,000
3,4-Methylenedioxymethamphetamine (MDMA)	50		
Marijuana (THC 12)			
11-nor-Δ^9 -THC-9 COOH	12	Δ^9 -THC	10,000
Cannabinol	12,500	11-nor-Δ^8-THC-9 COOH	12
Δ^8 -THC	6,000		
Marijuana (THC 50)			
11-nor-Δ^9 -THC-9 COOH	50	Δ^9 -THC	40,000
Cannabinol	50,000	11-nor-Δ^8-THC-9 COOH	40
Δ^8 -THC	25,000		
Marijuana (THC 20)			
11-nor-Δ^9 -THC-9 COOH	20	Δ^9 -THC	17,000
Cannabinol	20,000	11-nor-Δ^8-THC-9 COOH	15
Δ^8 -THC	10,000		
Marijuana (THC 40)			
11-nor-Δ^9 -THC-9 COOH	40	Δ^9 -THC	35,000
Cannabinol	40,000	11-nor-Δ^8-THC-9 COOH	35
Δ^8 -THC	20,000		
Marijuana (THC 25)			
11-nor-Δ^9-THC-9 COOH	25	Δ^8-THC	10,000
Cannabinol	20,000	Δ^9-THC	17,000
11-nor-Δ^8-THC-9 COOH	25		
Cocaine (COC 20)			
Benzoylecgonine	20	Ecgonine	1,500
Cocaine	20	Ecgonine methyl ester	12,500
Cocaethylene	30		
Opiates (OPI/MOP 40)			
Morphine	40	Norcodeine	6,250
Codeine	25	Normorphine	25,000
Ethylmorphine	25	Nalorphine	10,000
Hydromorphine	100	Oxymorphone	25,000
Hydrocodone	100	Thebaine	2,000
Levorphanol	400	Diacetylmorphine (Heroin)	50
Oxycodone	25,000	6-Monoacetylmorphine	25
Morphine 3-β-D-Glucuronide	50		
Opiates (OPI/MOP 25)			
Morphine	25	Morphine 3-β-D-Glucuronide	40
Codeine	20	Norcodeine	4,250
Ethylmorphine	20	Normorphine	17,000
Hydromorphine	70	Nalorphine	7,000
Hydrocodone	70	Oxymorphone	17,000
Levorphanol	300	Thebaine	1,500
Oxycodone	17,000	Diacetylmorphine (Heroin)	40
6-Monoacetylmorphine	20		
Phencyclidine (PCP 10)			
Phencyclidine	10	4-Hydroxyphencyclidine	2,500
Oxycodone (OXY 20)			
Oxycodone	20	Hydromorphone	10,000
Oxymorphone	40	Naloxone	5,000
Levorphanol	10,000	Naltrexone	5,000
Hydrocodone	1,500		
Oxycodone (OXY 50)			
Oxycodone	50	Hydromorphone	20,000
Oxymorphone	100	Naloxone	12,5000
Levorphanol	25,000	Naltrexone	12,500
Hydrocodone	3,750		
Cotinine (COT 20)			
(-)-Cotinine	20	(-)-Nicotine	300
Synthetic Marijuana (K2-25)			
JWH-018 5-Pentanoic acid metabolite	25	JWH-018 5-Hydroxypentyl metabolite	250
JWH-073 4-butanoic acid metabolite	25	JWH-073 4-Hydroxybutyl metabolite	250
JWH-018 4-Hydroxypentyl metabolite	200		
Benzodiazepines (BZO 50)			
Alprazolam	25	Flunitrazepam	25
a-hydroxyalprazolam	250	(±) Lorazepam	500
Bromazepam	130	RS-Lorazepamglucuronide	25
Chlordiazepoxide	130	Midazolam	1,000
Clobazam	25	Nitrazepam	25
Clonazepam	65	Norchlordiazepoxide	25
Clorazepatedipotass	65	Nordiazepam	130
Delorazepam	130	Oxazepam	50
Desalkylflurazepam	25	Temazepam	25
Diazepam	250	Triazolam	500
Estazolam	1,000		
Benzodiazepines (BZO 30)			
Alprazolam	15	Flunitrazepam	15
a-hydroxyalprazolam	150	(±) Lorazepam	300
Bromazepam	75	RS-Lorazepamglucuronide	15
Chlordiazepoxide	75	Midazolam	600
Clobazam	15	Nitrazepam	15
Clonazepam	40	Norchlordiazepoxide	15
Clorazepatedipotass	40	Nordiazepam	75
Delorazepam	75	Oxazepam	30
Desalkylflurazepam	15	Temazepam	15
Diazepam	150	Triazolam	300
Estazolam	600		
Benzodiazepines (BZO 20)			
Alprazolam	10	Flunitrazepam	10
a-hydroxyalprazolam	100	(±) Lorazepam	200
Bromazepam	50	RS-Lorazepamglucuronide	10
Chlordiazepoxide	50	Midazolam	400
Clobazam	10	Nitrazepam	10
Clonazepam	25	Norchlordiazepoxide	10
Clorazepatedipotass	25	Nordiazepam	50
Delorazepam	50	Oxazepam	20
Desalkylflurazepam	10	Temazepam	10
Diazepam	100	Triazolam	200
Estazolam	400		
Benzodiazepines (BZO 10)			
Alprazolam	10	Flunitrazepam	10
a-hydroxyalprazolam	80	(±) Lorazepam	150
Bromazepam	40	RS-Lorazepamglucuronide	10
Chlordiazepoxide	40	Midazolam	300
Clobazam	10	Nitrazepam	10
Clonazepam	20	Norchlordiazepoxide	10
Clorazepatedipotass	20	Nordiazepam	40
Delorazepam	40	Oxazepam	10
Desalkylflurazepam	10	Temazepam	10
Diazepam	80	Triazolam	150
Estazolam	300		
Methadone (MTD 30)			
Methadone	30	LAAM	200
Disopyramide	400	Doxylamine	12,500
(+)-Chlorpheniramine	6,250	Nor-LAAM	12,500
Methylenedioxymethamphetamine (MDMA 50)			
(±) 3,4-Methylenedioxymetham-phetamine HCl (MDMA)	50	3,4-Methylenedioxyethyl-amphetamine (MDE)	30
(±) 3,4-Methylenedioxyamphe-tamine HCl (MDA)	300	l-Methamphetamine	25,000
Ketamine (KET 50)			
Ketamine	50	Mephentermine	1,250
Tetrahydrozoline	20	Phencyclidine	625
Benzphetamine	1,250	(1R, 2S) - (-)-Ephedrine	5,000
d-Methamphetamine	1,250	Promazine	1,250
(+)Chlorpheniramine	1,250	4-Hydroxyphencyclidine	2,500
l-Methamphetamine	2,500	Promethazine	1,250
Clonidine	5,000	Levorphanol	2,500
Methoxyphenamine	625	Thioridazine	2,500
Disopyramide	625	MDE	2,500
d-Norpropoxyphene	625	Meperidine	1,250
EDDP	2,500	Dextromethorphan	75
Pentazocine	1,250	(+)3,4-Methylendioxymetham-phetamine (MDMA)	5,000
Barbiturates (BAR 50)			
Amobarbital	800	Alphenol	100
5,5-Diphenylhydantoin	1,500	Aprobarbital	80
Allobarbital	100	Butabarbital	40
Barbital	1,500	Butalbital	1,500
Talbutal	40	Butethal	90
Cyclopentobarbital	5,000	Phenobarbital	50
Pentobarbital	1,500	Secobarbital	50
Buprenorphine (BUP 10)			
Buprenorphine	10	Norbuprenorphine	50
Buprenorphine 3-D-Glucuronide	50	Norbuprenorphine 3-D-Glucuronide	100
Buprenorphine (BUP 5)			
Buprenorphine	5	Norbuprenorphine	25
Buprenorphine 3-D-Glucuronide	25	Norbuprenorphine 3-D-Glucuronide	50
Tramadol (TML 30)			
n-Desmethyl-cis-tramadol	60	o-Desmethyl-cis-tramadol	3,000
Cis-tramadol	30	Phencyclidine	30,000
Procyclidine	30	d,l-O-Desmethylvenlafaxine	15,000
6-Monoacetylmorphine (6-MAM 10)			
6-Monoacethylmorphine	10	Morphine	100,000
Fentanyl (FYL 50)			
Alfentanyl	1,500,000	Buspirone	37,500
Fenfluramine	125,000	Fentanyl	50
Norfentanyl	10	Sufentanyl	125,000
Fentanyl (FYL 20)			
Alfentanyl	600,000	Buspirone	37,500
Fenfluramine	50,000	Fentanyl	20
Norfentanyl	8	Sufentanyl	50,000

Fentanyl (FYL 10)			
Alfentanyl	300,000	Buspirone	20,000
Fenfluramine	25,000	Fentanyl	10
Norfentanyl	8	Sufentanyl	25,000
Carfentanyl (CFYL 50)			
Carfentanyl	50	Fentanyl	25
Sufentanil	300	(±)cis-3-Methylfentanyl	50,000
Ramifentanil	500	Butylfentanyl	200
3, 4-methylenedioxypyrovalerone (MDPV 300)			
3, 4-methylenedioxypyrovalerone	300		
alpha-Pyrrolidinovalerophenone (α-PVP 300)			
alpha-Pyrrolidinovalerophenone	300		
Lysergic Acid Diethylamide (LSD 10)			
Lysergic Acid Diethylamide	10		
Propoxyphene (PPX 50)			
d-Norpropoxyphene	50	d-Propoxyphene	50
Methaqualone (MQL 300)			
Methaqualone	300		
Carisoprodol (CAR 300)			
Carisoprodol	300		
2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine (EDDP 50)			
2-ethylidene-1,5-dimethyl-3,3-diphenylpyrrolidine (EDDP)	50		
AB-PINACA (ABP/K3 10)			
AB-PINACA	10	UR-144 4-hydroxypentyl	10,000
AB-PINACA 5-Pentanoic	10	APINACA 5-hydroxypentyl	10,000
AB-PINACA 5-hydroxypentyl	10	AB-FUBINACA	10
ADB-PINACA N-(5-hydroxypentyl)	30	ADB-PINACA Pentanoic Acid	10
AB-PINACA 4-hydroxypentyl	10,000	5-fluoro AB-PINACAN-(4-hydroxypentyl)	30
UR-144 5-hydroxypentyl	10,000	5-fluoro AB-PINACA	25
UR-144 5-Pentanoic		AB-CHMINACA	100
UR-144/K4 (25)			
UR-144 5-Pentanoic acid	25	UR-144 4-hydroxypentyl	10,000
5-fluoro AB-Pinaca N-(4- hydroxypentyl)	10,000	ADB-PINAC N-(4- hydroxypentyl)	＞10,000
UR-144 5-hydroxypentyl	5,000	AB-PINACA 4-hydroxypentyl	＞10,000
XLR-11 4-hydroxypentyl	2,000		
Zopiclone (ZOP 50)			
Zopiclone-N-oxide	50	Zopiclone	50
Gabapentin (GAB 2,000)			
Gabapentin	2,000		
Pregabalin(PGB 500)			
Pregabalin	500		

Cross-Reactivity

A study was conducted to determine the cross-reactivity of the test with compounds spiked into drug-free PBS stock. The following compounds demonstrated no false positive results on the Multi-Drug Rapid Test Cup when tested with at concentrations up to 100 μg/mL.

Acetaminophen	d/l-Chloropheniramine	Sulfamethazine
N-Acetylprocainamide	Chloroquine	Tetracycline
Aminopyrine	Clonidine	Tetrahydrocortisone 3 (β-D-glucuronide)
Ampicillin	l-Cotinine	Thioridazine
Apomorphine	Deoxycorticosterone	Tolbutamide
Atropine	Diclofenac	Trifluoperazine
Benzoic acid	Digoxin	d/l-Tryptophan
d/l-Brompheniramine	l -Ψ-Ephedrine	Uric acid
Chloral-hydrate	Estrone-3-sulfate	Ketoprofen
Chlorothiazide	l(–)-Epinephrine	Loperamide
Chlorpromazine	Fenoprofen	Meprobamate
Cholesterol	Gentisic acid	Nalidixic acid
Cortisone	Hydralazine	Niacinamide
Creatinine	Hydrocortisone	Norethindrone
Dextromethorphan	p-Hydroxytyramine	Noscapine
Diflunisal	Iproniazid	Oxalic acid
Diphenhydramine	Isoxsuprine	Oxymetazoline
β-Estradiol	Labetalol	Penicillin-G
Ethyl-p-aminobenzoate	Meperidine	Perphenazine
Erythromycin	Methylphenidate	Trans-2-phenylcyclopropylaminehydrochloride
Furosemide	Naproxen	Prednisolone
Hemoglobin	Nifedipine	d/l-Propranolol
Hydrochlorothiazide	d/l-Octopamine	d-Pseudoephedrine
o-Hydroxyhippuric acid	Oxolinic acid	Quinine
Ibuprofen	Papaverine	Ranitidine
d/l-Isoproterenol	Pentazocine	Serotonin
	hydrochloride	
Acetophenetidin	Phenelzine	Sulindac
Acetylsalicylic acid	Phenylpropanolamine	Tetrahydrocortisone 3-acetate
Amoxicillin	Prednisone	Thiamine
l-Ascorbic acid	Quinacrine	d/l-Tyrosine
Aspartame	Quindine	Triamterene
Benzilic acid	Salicylic acid	Trimethoprim
Benzphetamine	Zomepirac	Tyramine
Caffeine	Chloramphenicol	Verapamil

【BIBLIOGRAPHY】

1. Moolchan, E., et al, “Saliva and Plasma Testing for Drugs of Abuse: Comparison of the Disposition and Pharmacological Effects of Cocaine”, Addiction Research Center, IRP, NIDA, NIH, Baltimore, MD. As presented at the SOFT-TIAFT meeting October 1998.
2. Kim, I, et al, “Plasma and oral fluid pharmacokinetics and pharmacodynamics after oral codeine administration”, *ClinChem,* 2002 Sept.; 48 (9), pp 1486-96.
3. Schramm, W. et al, “Drugs of Abuse in Saliva: A Review,” *J Anal Tox*, 1992 Jan-Feb; 16 (1), pp 1-9
4. McCarron, MM, et al, “Detection of Phencyclidine Usage by Radioimmunoassay of Saliva,” *J Anal Tox*. 1984 Sep-Oct.; 8 (5), pp 197-201.
5. International Narcotics Control Board.Report of the International Narcotics Control Board for 2009[R].New York：UN，2010
6. Lane JC, Tennison MB, Lawless ST, et al.Movement disorder after withdrawal of fentanyl infusion.J Pediatr，1991，119（4）：649-651
7. Dominguez KD, Lomako DM，Katz RW，et al. Opioid withdraw in critically ill neonates.Ann Pharmacotherm，2003，37（4）：473-477
8. European Monitoring Centre for Drugs and Drug Addiction.Annual Report 2009[R].Lisbon：EMCDDA，2010
9. Stanley, Theodore H.; Egan, Talmage D.; Aken, Hugo Van (February 2008). "A Tribute to Dr. Paul A. J. Janssen: Entrepreneur Extraordinaire, Innovative Scientist, and Significant Contributor to Anesthesiology". Anesthesia & Analgesia. 106 (2): 451–462.
10. Vos, V. De (22 July 1978). "Immobilisation of free-ranging wild animals using a new drug". Veterinary Record. 103 (4): 64–68.Dominguez KD, Lomako DM，Katz RW，et al. Opioid withdraw in critically ill neonates.Ann Pharmacotherm，2003，37（4）：473-477
11. Mounteney, Jane; Giraudon, Isabelle; Denissov, Gleb; Griffiths, Paul (July 2015). "Fentanyls: Are we missing the signs? Highly potent and on the rise in Europe". International Journal of Drug Policy. 26 (7): 626–631.
12. Glass, IB. The International Handbook of Addiction Behavior. Routledge Publishing, New York, NY. 1991, 216
13. Baselt RC. Disposition of Toxic Drugs and Chemicals in Man. 6th Ed.
14. Waugh; et al. (2013). "Deaths Involving the Recreational Use of α-PVP (α-pyrrolidinopentiophenone)" (PDF). AAFS Proceedings. Abstract K16.
15. Glass, IB. The International Handbook of Addiction Behavior. Routledge Publishing, New York, NY. 1991, 216
16. Kamin I, Shaskan D (1959). "Death due to massive overdose of meprobamate". Am J Psychiatry. 115 (12): 1123–1124. doi:10.1176/ajp.115.12.1123-a. PMID 13649976.
17. Hollister LE (1983). "The pre-benzodiazepine era". J Psychoactive Drugs. 15 (1–2): 9–13. doi:10.1080/02791072.1983.10472117. PMID 6350551.
18. Gaillard Y, Billault F, Pepin G (1997). "Meprobamate overdosage: a continuing problem. Sensitive GC-MS quantitation after solid phase extraction in 19 fatal cases". Forensic Sci. Int. 86 (3): 173–180. doi:10.1016/S0379-0738(97)02128-2. PMID 9180026.
19. Allen MD, Greenblatt DJ, Noel BJ (1977). "Meprobamate overdosage: a continuing problem". Clin Toxicol. 11 (5): 501–515. doi:10.3109/15563657708988216. PMID 608316.
20. Kintz P, Tracqui A, Mangin P, Lugnier AA (1988). "Fatal meprobamate self-poisoning". Am J Forensic Med Pathol. 9 (2): 139–140. doi:10.1097/00000433-198806000-00009. PMID 3381792.
21. Eeckhout E, Huyghens L, Loef B, Maes V, Sennesael J (1988). "Meprobamate poisoning, hypotension and the Swan-Ganz catheter". Intensive Care Med. 14 (4): 437–438. doi:10.1007/BF00262904. PMID 3403779.
22. Lhoste F, Lemaire F, Rapin M (1977). "Treatment of hypotension in meprobamate poisoning". N Engl J Med. 296 (17): 1004. doi:10.1056/NEJM197704282961717. PMID 846530.
23. Bedson H (1959). "Coma due to meprobamate intoxication. Report of a case confirmed by chemical analysis". Lancet. 273 (1): 288–290. doi:10.1016/S0140-6736(59)90209-0. PMID 13632000.
24. Uchiyama, N.; Matsuda, S.; Wakana, D.; Kikura-Hanajiri, R.; Goda, Y. (2012). "New cannabimimetic indazole derivatives, N-(1-amino-3-methyl-1-oxobutan-2-yl)-1-pentyl-1H-indazole-3-carboxamide (AB-PINACA) and N-(1-amino-3-methyl-1-oxobutan-2-yl)-1-(4-fluorobenzyl)-1H-indazole-3-carboxamide (AB-FUBINACA) identified as designer drugs in illegal products". Forensic Toxicology. 31: 93–100. doi:10.1007/s11419-012-0171-4.
25. AB-PINACA". Cayman Chemical. Retrieved 25 June 2015.
26. Banister, Samuel D.; Moir, Michael; Stuart, Jordyn; Kevin, Richard C.; Wood, Katie E.; Longworth, Mitchell; Wilkinson, Shane M.; Beinat, Corinne; Buchanan, Alexandra S.; Glass, Michelle; Connor, Mark; McGregor, Iain S.; Kassiou, Michael (2015). "Pharmacology of Indole and Indazole Synthetic Cannabinoid.Designer Drugs AB-FUBINACA, ADB-FUBINACA, AB-PINACA, ADB-PINACA, 5F-AB-PINACA, 5F-ADB-PINACA, ADBICA, and 5F-ADBICA". ACS Chemical Neuroscience. 6 (9): 1546–59.
27. Volpicellim, Joseph R., M.D., Ph.D.: Alcohol Dependence: Diagnosis, Clinical Aspects and Biopsychosocial Causes., Substance Abuse Library, University of Pennsylvania, 1997.
28. Baselt RC. Disposition of Toxic Drugs and Chemicals in Man. 2nd Ed. Biomedical Publ., Davis, CA. 1982; 488

Manufactured for:
American Screening Corp
9742 St Vincent Ave Ste 100
Shreveport, LA 71106
1-318-798-3306
sales@americanscreeningcorp.com

Number: 14602045800
Revision date: 2024-01-23