Addiction isn’t just about drink and drugs, or the images that come to mind of drunks, beggars, prostitutes and organised crime etc. Addiction is a disease of any recurring misuse of any activity which has harmful consequences to the individual’s health, mental state or social life. This may include drugs, foodsovereating, gambling, sex/pornography, exercise/thrill-seeking sports, shopping, video games/internet addiction, self-harm/cutting, vandalism, or even religion. In this bulletin we will focus on drug and alcohol addiction.

Types of addiction
The DSM-IV TR psychiatric manual defines drug addiction as “substance dependence”:

“Substance dependence may be diagnosed, when an individual persists in use of alcohol or other drugs despite problems related to use of the substance. Compulsive and repetitive use may result in tolerance to the effect of the drug and withdrawal symptoms when use is reduced or stopped. This, along with substance abuse are considered substance use disorders…”

There are two types of substance dependence which are of interest to drug researchers and drug workers:

Physical dependence is characterised by a clear-cut abstinence syndrome, which occurs when the removal of drug is accompanied by physical withdrawal symptoms, such as cramps, sweating, increased heart rate, dilated pupils, pain, excitement, hallucinations, seizures, etc. Physically addicting drugs like opiates, benzodiazepines, barbiturates, alcohol and nicotine may initially induce pleasure, but that changes over time because of tolerance (having to increase the dose to get the same effect). Avoiding the unpleasant withdrawal symptoms, tolerance causes compulsive use.

Nicotine’s initial pharmacological effects, for example, quickly give way to a rapid tolerance, and so any subsequent “pleasure” derived from the drug thereafter consists mainly in alleviating its own withdrawal symp-toms. In the case of opiates like heroin, this is extremely uncomfortable, but rarely life-threatening, whilst in the case of depressant drugs like alcohol, barbiturates or benzodiazepines, can lead to seizures and even death. Some non-addicting medicines like cortisone, beta blockers and most antidepressants (which are not designed to be euphoriants) can still cause a tolerance, physical dependence, and withdrawal symptoms if abruptly discontinued.

Psychological dependence is a more complex phenomenon than physical dependence, and has much more importance in the genesis of compulsive drug-taking. It occurs when the drug produces psychological reinforcement, and its removal is accompanied by impaired psychological function such as cravings, irritability, anger, insomnia, anxiety, difficulty concentrating, depression, anorexia, etc. Most psychologically-addicting drugs, including heroin, morphine, cocaine, amphetamines, alcohol and nicotine produce a reward-reinforcement effect in the dopaminergic system of the brain (or its accessory structures), which, when suspended, cuts off the “anti-stress” neurotransmitter dopamine (DA), and produces unpleasant withdrawal symptoms. For this reason experimentally addicted rats will press a lever up to 2000 times per hour for stimulation of certain brain areas that release dopamine. But because the brain, like the body, exists in a natural state of homeostasis (acting to minimise the effect of external changes), chronic artificial elevation of DA by alcohol, or recreational drugs (or even brain electrodes), eventually results in a reflex decrease in the number, size and sensitivity of brain DA receptors available in a process known as down-regulation. This makes dopamine neurones less excitable in response to the drug, and leads to tolerance. By this time the brain may have become so dependent on the drug that it has stopped producing its own natural neurotransmitters, and started producing opposing, or antagonistic substances to minimise the effects of the drug. When the addictive substance is then withdrawn, the brain cannot restart its functions, leading to the emergence of unpleasant symptoms known as psychological withdrawal symptoms, inverse to the pleasure response.

1. Drug choice and addiction
Drugs known to cause addiction include illegal drugs like amphetamine, methamphetamine, cocaine, crack cocaine, heroin as well as some prescription (POM) or over-the-counter (OTC) drugs such as the sedative/hypnotic barbiturates, tranquilizers like benzodiazepines, methaqualone, analgesics like morphine and codeine, oxycodone, hydromorphone, fentanyl, pethidine, methadone and recreational substances like alcohol, caffeine and nicotine which, incidentally, are the most common addictions in society.

Some drugs, however, are more addictive than others. Alcohol and codeine, for instance, typically require repeated exposures to addict their users, while drugs like heroin or crack cocaine can create dependence after first use. Generally, the most addictive drugs are those which act the quickest and produce the strongest euphoria. The most addictive drugs create the most intensive cravings, and withdrawal symptoms.

“What causes addiction?”
Addiction is a multifactorial disease, caused by a complex interplay of genetic, neurochemical, medical, psychosocial, cultural and spiritual factors. How quickly addiction becomes established depends on the substance used, how often it is taken, how it is taken, the intensity of the pleasure it produces, and probably most importantly the individual’s genetic, medical and psychological susceptibility to use again. Some individuals may show signs of addiction from the first moment of exposure to a particular substance, whereas others may use them socially without ever becoming addicted to them. Addiction can rarely be cured, it can only be controlled, but the desire and risk of relapse remain. These controls are nowadays called “harm reduction measures”.

Nicotine is an exception to the “pleasure” rule: its reputation as one of the most addictive drugs is not so much owed to its pleasure-inducing action (often described as a mild “calming euphoric effect”), as it is the rapid tolerance and intense cravings that develop to its milder effects. Tolerance almost immediately increases the dose required to achieve the desired effect, which in turn increases the likelihood of addiction. Once hooked, smokers find themselves cheated of any appreciable pleasure, and stuck in a vicious craving cycle, smoking only to allay the onset of withdrawal symptoms. No wonder some heroin addicts rate the urge to smoke as equal to or stronger than the urge to take heroin!

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“Addiction isn’t just about drugs and alcohol.”
Methamphetamine ("crystal meth", "ice" "crank" etc), for instance, is probably the most potent, long-acting and hence habit-forming stimulant on the black market, and according to recent BBC news reports, has superseeded heroin and crack in popularity in the “Golden Triangle” drug-producing areas of Thailand, Burma and Laos. Although it is not physically addictive it can be smoked and produces a rapid exhilaration and intense euphoria which gives it huge psychological addiction potential.

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The addiction potential of a particular drug will also depend on how quickly the body physically reacts to remove it. The biological half life of a drug is the time taken for the amount of the drug in the body to decrease by 50%. Generally the shorter the half life, the quicker the development of tolerance, and the sooner the withdrawal symp-toms are felt. For example, drugs like heroin and morphine with half lives of 4-6 hours require administration several times a day, and have a greater addiction potential than their longer lasting counterpart metha-done whose half life is 30 hours. They also produce more severe with-drawal symptoms, which unlike methadone’s long withdrawal, their resolution is much quicker. Nicotine’s very short half life of less than 2 hours requires even more frequent administration to stop withdrawal.

Neurobiology of addiction
Addiction may arise because of low DA activity (hypodopamism) in the...
final “pleasure-reward” circuit of the brain. Hypodopaminism can be chemically induced, by recreational drugs and other substances or be an inherited genetic problem (which paradoxically often leads into drug-seeking behaviour to correct the problem). The former case involves a number of discrete neurobiological and biochemical adaptations in the mesolimbic system.

Initial exposure to drugs will cause DA-responsive cells in the “pleasure-reward” pathway of the mesolimbic system to raise levels of a signalling molecule cyclic AMP (cAMP), which in turn activates the genetic transcription factor cyclic AMP response element binding protein (CREB). Importantly, high CREB levels actually slow down DA release, which temporarily inhibits the pleasure-reward circuit. This neurobiological safeguard basically sets a “ceiling effect” to how much pleasure-reward the drug can give between “hits”. Chronic repeated drug use however, causes a sustained activation of CREB which, accompanied by the upregulation of “neuroadaptive” N-methyl d-aspartate (NMDA)-glutamate receptors, dampens the pleasure-reward circuitry, inducing tolerance to the effects and all other natural goal-motivated rewards, rendering the addict depressed, unmotivated and disinterested in their surroundings. If the addict abstains, his CREB levels will return to normal, and the DA release. The financial impact of this process is theoretically, be the end of the problem. But as any addict will tell you, stopping is easier than abstaining.

Even though CREB is switched off after a few days of abstinence, it doesn’t explain the chronic grip that some drugs have over the brain. Certain brain alterations can cause addicts to return to a substance after years or even decades of abstinence. For instance, if the drug supply is disconnected from an animal dispenser, addicted monkeys continue to press its lever at a high rate, and it takes months for the response to be extinguished, implying that in monkeys, as in man, the reinforcing effect of the drug greatly outlasts the duration of the physical abstinence syndrome. This is owed to the phenomenon of sensitisation, which is a process that makes the addict’s brain more sensitive to a particular drug long term. It involves the strengthening of learned drug-associated behaviours at the expense of adaptive responses to natural rewards like food, sex or a job well done.

The neurobiological process underlying sensitisation is thought to be involved expression of the “neuroadaptive” NMDA-glutamate receptor which governs the brain process of “learning” to become more (or less) sensitive to a drug over time, and two genetic transcription factors delta FosB and G-protein signalling 9-2 (RGS 9-2) proteins, responsible for long term molecular changes in the pleasure-reward circuit. Whilst altered glutamate sensitivity strengthens the neuronal pathways that link memories of drug-taking experiences (particularly the amygdala which is the brain’s seat of emotional memories) with high reward, they feed the desire to seek the drug. FosB and RGS 9-2 go to work sensitising the mesolimbic system’s VTA and NA to their inputs. As long as CREB activity is high, tolerance to the drug dominates, but as soon as the addict abstains, CREB levels decline and the hardwired effects of sensitisation take over leading to cravings and drug seeking behaviour. At this stage even the sight of drug paraphernalia or familiar sights, sounds and smells associated with past drug use can trigger pleasurable emotional memories in the amygdala and cause relapse. Interestingly, experimental animals lacking the RGS 9-2 transcription factor show a lack of responsiveness to cocaine and amphetamine and don’t seem to get addicted. Their brains literally don’t “learn” to become addicted.

2. Neurochemical factors and addiction

Natural or genetic hypodopaminism consists of neurochemical factors predisposing a first time user to addiction. They are thought to involve a breakdown in the complex cascade of events leading to the activation of the final common “pleasure-reward pathway” in the brain’s mesolimbic system which starts in the ventral tegmental area (VTA), and ends in the nucleus accumbens (NA) involving several neurotransmitters and neuroanatomical structures. The firing of the “pleasure reward” pathway begins outside in the “starter motor area” of the brain’s hypothalamus with the activation of serotonin (5-HT) neurones.

These neurones project to the neurons of the reward pathway at the VTA where liberation of 5-HT triggers the release of “amplifier” neurones containing one of the brain’s natural pain-killers, histamine-enkephalin (ME). When this reaches a critical point it “lifts the brake” of the inhibitory gamma-aminobutyric acid neurones, originating in the Substantia Nigra’s A9 region, which ordinarily prevent the release of dopamine (DA) from the VTA. The liberation of DA that follows and its final interaction with D2 receptors on the cell bodies of neurones at the NA, produces a pleasure-reward-reinforcement effect, which results in feelings of well-being, satisfaction and stress reduction. This cascade can be genetically faulty at any, or all of its four levels such as the 5-HT neurones of the “starter motor” area of the hypothalamus, the “amplifier system” of the ME neurones, the “braking system” of the GABA neurones, or the DA neurones of the final “pleasure-reward” circuit of the mesolimbic system. The “braking system” of the VTA where liberation of 5-HT triggers the release of dopamine (DA) from the VTA. The liberation of DA that follows and its final interaction with D2 receptors on the cell bodies of neurones at the NA, produces a pleasure-reward-reinforcement effect, which results in feelings of well-being, satisfaction and stress reduction. This cascade can be genetically faulty at any, or all of its four levels such as the 5-HT neurones of the “starter motor” area of the hypothalamus, the “amplifier system” of the ME neurones, the “braking system” of the GABA neurones, or the DA neurones of the final “pleasure-reward” circuit of the mesolimbic system. 3. Genetic factors and addiction

The most common defect to the cascade system is a genetic loss of sensitivity and numbers of dopamine D-2 receptors (hypodopaminergic trait) in the final segment of the “pleasure-reward” pathway, known as the Tac A1 allele. Other genetic polymorphisms like the VNTR intron 1 mutation which normally slow down the dopamine manufacturing enzymes Tyrosine hydroxylase (TH), leading to lowered transmitter levels, whereas the SLC6A3-9 mutant affects the dopamine transporter, impairing dopamine release. The A48G, S9G and VNTR (tandem repeat in exon 3) mutants affect other important classes of DA receptors.

The “starter motor” area of the 5-HT neurones can also be adversely affected by the same genetic polymorphisms, such as the L279 mutant which damages the 5-HT manufacturing enzyme Tryptophan Hydroxylase, and at least two 5-HT transporter mutants which impair transmitter release. 5-HT receptor defects are as varied as the receptor subclass themselves and include the G861C, T102C, G1438A, C238 and P158 mutants.

The ME “amplifier system” of the VTA can be damaged by defects to the u-opioid receptor and the GABA “braking mechanism” by multiple genetic polymorphisms of its receptor subunits too numerous to list here. (For a good review see Tyndale RF, Genetics of alcohol and tobacco use in humans Ann Med 2003, 35: 94-121). One or more interruptions to this complex cascade lead to a dulling of the final reward pathway and this results in an inability to feel pleasure known as anhedonia, often observed in established addicts. It manifests in an form of sensory deprivation of the brain’s pleasure mechanism which produces a biochemical inability to derive reward from ordinary, everyday, activities. Drug researchers call it Reward Deficiency Syndrome (RDS). Some individuals can suffer with it all their lives never knowing why they don’t feel “normal”. When the levels of dopamine in the VTA or the number of receptors in the NA are genetically below par in this way, symptoms of anxiety, anger, fear, impulsiveness, compulsiveness, substance craving and the behavioural rituals which alleviate these negative emo-
violent behaviour and criminal tendencies. Many prisoners and long genetic propensity for antisocial behaviour, conduct disorders, This category is made up of people who may possess a general life-

These are the big drinkers who can consume alcohol all day, and who stay on a high with little signs of inebriation or negative effects. They have a high tolerance for alcohol and after many years of drinking are more prone to develop liver problems than psychiatric symptoms.

Oftentimes, from Northern European or American Indian ancestors, this category is made up of the typical “bad-starters” who learn how to drink. They are often moody, changeable and unpredictable alcoholics who experience very bad hangovers and who may become socially disruptive, engaging in fights and arguments, dangerous driving, irrational or bizarre behaviour and even criminal acts after drinking.

Biotype 3: The PGE1 deficient biotype suffers with lifelong depression resulting from a genetic shortage of the neurotransmitter prostaglandin E1 (PGE1). Alcohol temporarily banishes depression by liberating PGE1 but this is followed by rebound and often suicidal depression when building up a tolerance to the drug.

Biotype 4: The hypoglycaemic biotype is addicted to the sugars in alcohol because the body produces too much insulin which starves his brain of the glucose it needs. Alcohol temporarily gives him a lift before his body overreacts with confusion, weakness, sleepiness and lack of co-ordination, resembling acute alcohol intoxication (even at low intakes).

This category is made up of individuals who cannot handle too much alcohol and who despite feeling a temporary increase in well-being after a drink or two, quickly show signs of neuroglycopenia (brain glucose starvation) resembling intoxication.

Biotype 5: The dopamine deficient biotype brings us back to carriers of the A1 allele with RDS who are not sufficiently rewarded by stimuli that normal people find satisfying or calming. It tends to be made up of risk takers, gamblers, sexually promiscuous, compulsive overeaters, drug takers, and of course alcoholics who possess a lifelong propensity for antisocial behaviour, conduct disorders, violent behaviour, and criminal tendencies.

This category is made up of people who may possess a general life-long genetic propensity for antisocial behaviour, conduct disorders, violent behaviour and criminal tendencies. Many prisoners and re-offenders will probably fall into this category.

4. Brain damage and addiction

Some researchers believe that addiction as a disease may also involve brain damage leading to a failure of the higher rational inhibitory centres in the brain, like the orbitofrontal cortex, to send “stop” signals to the instinctive emotional lower brain centres involved in the generation of pleasure-reward responses; such as the mesolimbic system and the seat of emotional memories the amygdala. Experimentally depressed animals, for instance, will forget food, sleep and sex for continued access to a lever that administers psychoactive drugs which tends to indicate a failure in the higher brain centres to delay gratification for more important goal driven behaviour. Brain imaging has confirmed this in human cocaine addicts who have decreased activity, as compared with non-addicts, in the same area of the pre-frontal cortex and a presentation of stimuli associated with natural rewards. This explains the chaotic lives led by some addicts.

5. Medical disorders and addiction

Psychiatric disorders: Statistically a greater number of addicts present with psychiatric disorders that non-addicts. Anxious individuals and atypical depressives may become psychologically dependent on alcohol or, worse still, prescription tranquilizers for their confidence-enhancing properties before being locked. They may also resort to sugar and carbohydrate binging for a mood boost (many addicts binge on pizza), and by stimulating the release of insulin from the pancreas to withdraw the larger amino acids from the blood stream. This allows the selective build up of tryptophan, and hence the body’s natural antidepressant, Serotonin (5-HT) in the brain. Crude “self-medication” in this way can often mean the beginning of addiction. Stressed-out individuals also tend to convert their DA reserves into the stress transmitter Norepinephrine (NE) too rapidly, which has the two-fold effect of releasing the master stress hormone corticotropin-releasing factor (CRF) and lowering DA levels (hypodopaminism) associated with drug-seeking behaviour. In experimentally stressed rats, CRF blockers and CRF receptor antagonists decrease the self-administration of addictive substances. Both hyperactive biotypes may also run the risk of stimulant abuse in later life to tackle the Tac 1A allele-associated hypodopaminism in the former case and as a misbegotten attempt to lose weight in the latter.

Hypoglycaemia: Individuals who suffer with poor blood glucose control (hypoglycaemia) have lower levels of brain 5-HT and their hypoglycaemics (which we have previously seen is involved in the final “pleasure-reward” circuit), are also more likely to develop carbohydrate addiction and alcoholism than those who don’t. Because they tend to overproduce insulin (hyperinsulinism) and suffer with low blood glucose symptoms of irritability, tiredness, confusion, orientation, for blindness and depressed mood they may also run the risk of stimulant abuse in later life to tackle the Tac 1A allele-associated hypodopaminism in the former case and as a misbegotten attempt to lose weight in the latter.

Adrenal insufficiency: This can often be the start of caffeineism, smoking or more serious stimulant abuse. Individuals who live fast-paced, high stress, fear-based lifestyles running on “emergency mode” all the time can eventually start to experience adrenal, burnout associated with low secretion of the stress hormones cortisol, adrenaline and a host of symptoms like chronic fatigue, anxiety, low blood sugar and irritability. When the stress hormones fall, the liver’s manufacture of the copper-binding protein ceruloplasmin falls too, and free copper levels start to rise and wreak havoc on brain function by inhibiting DA-forming enzymes like DOPA decarboxylase and stimulating enzymes involved in the breakdown of DA and 5-HT like Dopamine Beta Carboxylase, 5-hydroxytryptophan decarboxylase and Monoamine Oxidase (MAO). A cigarette (containing the copper antagonist cadmium), an espresso or something stronger like cocaine or prescription stimulants like Ritalin will often whip the adrenals back into action and shift the brain fog and fatigue that results from a build up of unbound copper in the blood. Overtime, however, the body becomes tolerant to these substances and addiction creeps in.

Food allergy often starts with a “leaky” gut which admits partially digested food particles to the blood stream, provoking immune and behavioural reactions. Gluten exorbin B5 protein found in wheat, barley, rye and oats and alphaS1 casein protein or alpha S1 caseomorph, as it is also known, in cow’s milk are known as exorphins for their
ability, when improperly digested in this way, to mimic the brain’s own natural painkillers (endorphins) by activating a special class of opioid receptors in the brain. Individuals with a leaky gut often experience a short-lived high and a longer—lived withdrawal phase after consuming foods high in these substances, which keeps them stuck in a recurring cycle of food addiction. Heroin addicts routinely resort to pints of milk or milk chocolate snacks between fixes for precisely the same reason.

8. Spiritual factors
With the advent of liberal democracies, the increase in materialism and weakening of stabilising moral influences in western societies over the last couple of hundred years, there has been a radical shift in human behaviour away from leading moral lives, to enjoyment and indulging pleasure, to excess in all its forms, good and bad. According to scientists, this is a disturbing trend because neurochemical studies are showing that pleasure-seeking behaviour is a common denominator of addiction to alcohol, drugs, and carbohydrates. Certainly this neo-liberal notion, born out of a superabundance of material wealth in the west, has no doubt encouraged the growth of addictive disorders in the genetically predisposed and curious to a degree unknown. Traditional values are viewed by most of today’s youth as out of date, and tend to be fuelled by the mass media’s epithet that religion is “the enemy of freedom and pleasure”. On the contrary, it is precisely this sort of “moderation in all things” (temperance) which produces happier, healthier people, free of the need to be satisfied by stimulants. Christian rehab programmes like Cenacolo for ex-drug addicts, for instance, have a 93% success rate which greatly surpasses that for most medical treatments. If nothing else history has taught us that decadence and moral corruption cannot be sustained.

Conclusions
Addiction is a complex subject, and isn’t just confined to drug and alcohol use. Indeed most stimulants, edibles, routines and activities that trigger a feeling of well-being can become addictive. Such addictions are not usually as dangerous as drugs or alcohol, and unless chronic, don’t cause serious damage to the body or have the same social or economic implications.

Addicted people can be genetically predisposed to addiction, and others can become addicted through the body’s need to stabilise mood after repeated stimulation.

Substances such as alcohol, drugs and food can alter the mood of the user, and therefore when addicted, taking these extinguishes the craving, withdrawal symptoms, and normalises the mood. Many addicts, for example smokers, feel ‘normal’ when under the influence of their addicted substance.

Addicted eaters must really struggle, because the food that they are addicted to, such as carbohydrates, may form part of their daily diet. We also know that associated activities can instigate cravings; so eating alone could start a craving for the very thing they are trying to avoid.

Crawling triggers can be as diverse as:
- Associated routine activity
- Smells
- Sight
- Sound
- Medication

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