GCE A LEVEL

S23-A290U30-1

A290U30-1

MONDAY, 5 JUNE 2023 – AFTERNOON

PSYCHOLOGY – A level component 3

Psychology: Implications In The Real World

2 hours 15 minutes

ADDITIONAL MATERIALS

A WJEC pink 16-page answer booklet.

INSTRUCTIONS TO CANDIDATES

Use black ink or black ball-point pen. Do not use gel pen or correction fluid.

Answer **three** questions in Section A and **one** question in Section B.

Write your answers in the separate answer booklet provided following the instructions on the front of the answer booklet.

Use both sides of the paper. Write only within the white areas of the booklet.

Write the question number in the two boxes in the left-hand margin at the start of each answer, for example | **0** | **1** | .

Leave at least two line spaces between each answer.

INFORMATION FOR CANDIDATES

The number of marks is given in brackets at the end of each question or part-question.

You are reminded of the need for good English and orderly, clear presentation in your answers.

Assessment will take into account the quality of written communication used in your answers.

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SECTION A: Applications

Answer **three** questions in full.

0 1 **Addictive behaviours**

a) Describe biological explanations of addictive behaviours. [10]

b) Briefly explain how biological explanations could be applied to modifying addictive behaviours. [5]

c) Evaluate the effectiveness and ethical implications of **one** method of modifying addictive behaviours. [10]

0 2 **Autistic spectrum behaviours**

a) Describe **one** individual differences explanation of autistic spectrum behaviours. [10]

b) Molly has recently been diagnosed with autistic spectrum behaviours. She has social and communication difficulties. Molly's psychologist suggested to her parents that either Picture Exchange Communication System or Relationship Development Intervention could help to modify Molly's behaviours.

With reference to the above scenario, evaluate the effectiveness of Picture Exchange Communication System (PECS) and Relationship Development Intervention as methods of modifying Molly's autistic spectrum behaviours. [15]

0 3 **Bullying behaviours**

a) **i)** Briefly evaluate the ethical and social implications of Creating A Peaceful School Learning Environment (CAPSLE). [5]

ii) Briefly evaluate the ethical and social implications of the Olweus Bullying Prevention Programme. [5]

b) Ethan seems to be popular at work and appears to have good social skills. However, Ethan's boss Saira has received complaints from some of Ethan's colleagues that he has been displaying bullying behaviours towards them, such as making nasty comments and excluding them from conversations.

With reference to the above scenario, describe **one** individual differences explanation of bullying behaviours. [15]

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0 4 **Criminal behaviours**

a) Describe individual differences explanations of criminal behaviours. [10]

b) Briefly evaluate the effectiveness of restorative justice and anger management as methods of modifying criminal behaviours. [5 + 5]

c) Briefly explain how biological explanations could be applied to modifying criminal behaviours. [5]

0 5 **Schizophrenia**

a) Evaluate **one** explanation of schizophrenia. [10]

b) Alia displays unusual behaviours; she also has disordered thinking, delusions and hallucinations (she hears voices). These symptoms led to a diagnosis of schizophrenia. She was initially given antipsychotic medication, but still has some distressing symptoms.

Describe how cognitive behavioral therapy could be used to modify Alia's behaviours. [15]

0 6 **Stress**

a) Describe and evaluate **one** social psychological explanation of stress. [20]

b) Briefly explain how individual differences explanations could be applied to modifying stress. [5]

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SECTION B: Controversies

Answer **one** of the questions.

Either,

0 7 **Scientific status**

'The changing nature of 'science' has made it difficult to establish the scientific status of psychology.'

To what extent do you agree with this statement? Justify your answer using psychological knowledge. [25]

Or,

0 8 **Sexism**

'When psychological research is conducted today, we can be certain that any gender differences found are real and are not the result of gender bias.'

Using your knowledge of psychology, to what extent do you agree with this statement? [25]

END OF PAPER

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Surname	StemUp Model Solution
Answer booklet	Write the question number in the left-hand margin at the start of each answer.

Question number

01 (a)

Dopamine: most addictive drugs and behaviours act on the mesolimbic dopamine pathway, the brain's reward system, which runs from the ventral tegmental area (VTA) to the nucleus accumbens. Natural rewards such as food release dopamine here, but drugs produce a much larger and faster release: cocaine blocks dopamine reuptake, amphetamine increases its release and nicotine stimulates nicotinic receptors on dopamine neurons. This produces a 'high' that is strongly reinforcing. Boileau et al. (2003) used PET scanning and found that drinking alcohol increased dopamine release in the ventral striatum, and Joutsa et al. (2012) found dopamine release in the ventral striatum during gambling, with greater release linked to more severe gambling symptoms, showing behavioural addictions use the same system.

Repeated use leads to tolerance and withdrawal. The brain adapts by reducing dopamine release and down-regulating D_2 receptors, so more of the drug is needed for the same effect and, without it, the person feels low (withdrawal). Volkow et al. (1997) found that cocaine abusers showed a much smaller dopamine response and less of a 'high' to a stimulant than non-addicts. The cortex helps maintain addiction: Volkow et al. (1992) found reduced activity in the frontal cortex of cocaine abusers, and Bolla et al. (2003) found abnormal orbitofrontal activity during decision-making tasks, which may explain impulsive choices. Wang et al. (1999) found that recalling drug experiences increased orbitofrontal activity during craving. Robinson and Berridge's (2003) incentive sensitisation theory argues that the dopamine system becomes sensitised so drug cues trigger intense 'wanting', even though 'liking' declines.

Addiction genes: twin and adoption studies show addiction is partly inherited. Goldman et al. (2005) reported heritability estimates from about 0.39 for

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hallucinogens to 0.72 for cocaine. The A1 allele of the DRD2 gene is linked to fewer D_2 receptors, so carriers may use drugs to compensate for an under-active reward system (reward deficiency). Genes for alcohol-metabolising enzymes also matter: Higuchi et al. (2006) found the inactive ALDH2 variant, common in Japanese people, causes unpleasant flushing after drinking and protects against alcoholism, and Edenberg et al. (2006) found ADH4 variants affected alcoholism risk in European populations. In line with the diathesis-stress model, Kaufman et al. (2007) found that children with the short SHTT allele who had been maltreated were at greater risk of early alcohol use, so genes create vulnerability that the environment triggers.

Disease of the brain: the National Institute on Drug Abuse describes addiction as a chronic, relapsing brain disease. Drugs change the basal ganglia (reward), the extended amygdala (stress and withdrawal) and the prefrontal cortex (impulse control and decision-making). Buddy T (2021) explains that these changes to the reward system and impulse control mean addicts lose control over use, so addiction is an illness rather than a moral failing.

01 (b)

If addiction is caused by drugs acting on the dopamine reward system, treatment can target the same receptors. Agonist substitution such as methadone binds to the same opioid receptors as heroin, but acts more slowly and lasts longer, so it stabilises the reward system, prevents withdrawal and cravings and allows the dose to be reduced gradually. Antagonists such as naltrexone block the receptors, so taking the drug no longer releases dopamine or produces a high; without reinforcement, drug-taking should be extinguished.

The genetic explanation is applied in aversion therapy: Antabuse (disulfiram) blocks the ALDH enzyme, copying the protective ALDH2 variant, so drinking causes acetaldehyde build-up and sickness. Genetic screening could also identify vulnerable people for early prevention. The disease model justifies treating addiction medically with long-term maintenance and relapse prevention.

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01 (c)

I will evaluate agonist and antagonist substitution. There is good evidence that agonist substitution is effective. NICE recommends methadone and buprenorphine as maintenance treatments for opioid dependence, and Van den Brink and Haasen's (2006) review found that methadone maintenance reduces illicit heroin use, injecting, crime and death. Methadone is still the preferred treatment in the UK, and Whelan and Remski (2021) found that addicts were more likely to stay in treatment because they enjoyed the feeling it gave them, so retention is high.

However, alternatives may be better. Buprenorphine is a partial agonist with a 'ceiling effect', so higher doses do not increase respiratory depression, and Marteau et al. (2015) used longitudinal data and found it about six times safer than methadone in terms of overdose. The antagonist naltrexone has mixed support: NICE found it reduced relapse only when patients were motivated and well monitored, as many stop taking it. Lahti et al. (2010) found naltrexone reduced gambling urges, but this research is limited. There are also problems with the research: results vary between countries with different health systems, and many studies do not follow participants up months or years later to check for relapse. The method also rests on the dopamine explanation, which ignores social and cognitive causes, so drugs treat the physical dependence but not the reasons for use; CBT may address these better and is often combined with it.

Ethically, methadone has side effects and can cause fatal respiratory depression if it interacts with alcohol or antidepressants, and there is a risk of overdose if it is taken alongside other drugs. Critics argue it simply replaces one addiction with another, as some users stay on it for years. Naltrexone requires liver function to be monitored, can cause withdrawal if the person is still opioid dependent, and if a person relapses they may take more heroin to override the block, risking overdose because tolerance has fallen. Valid consent may also be questioned if treatment is a condition of bail or benefits.

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In conclusion, agonist substitution, especially buprenorphine, is effective at reducing harm, crime and death, but naltrexone works only for motivated patients. Because of the ethical risks, both should be closely supervised and combined with psychological support.

04 (a) Eysenck's criminal personality: Eysenck (1964) proposed that personality has a biological basis and varies on three dimensions: extraversion (E), neuroticism (N) and psychoticism (P), measured by the Eysenck Personality Questionnaire. Extraverts have an under-aroused nervous system (reticular activating system), so they seek stimulation and excitement, take risks and act impulsively. Neurotic people have a reactive sympathetic nervous system, so they are anxious, emotionally unstable and overreact to threat. Psychoticism is linked to higher testosterone and is shown as being cold, aggressive and lacking empathy.

Eysenck argued that people high in E and N are hard to condition, so they do not learn to avoid antisocial behaviour through punishment during socialisation. The typical criminal personality is therefore high in E, N and P, seeking arousal through crime and failing to learn from consequences.

Intelligence factors (cognitive explanations): offenders may show lower IQ (Hirschi and Hindelang (1977) found delinquents averaged about 8 IQ points lower than non-delinquents) and differences in thinking. Kohlberg (1969) proposed that moral reasoning develops through three levels: pre-conventional, conventional and post-conventional. Offenders are more likely to remain at the pre-conventional level, where right and wrong are judged by avoiding punishment and gaining reward, so they offend if they think they can get away with it. Colby et al.'s (1983) 20-year follow-up of Kohlberg's original sample supported this sequence of stages, and Palmer and Hollin (1998) found that young offenders showed less mature moral reasoning than non-offenders. Offenders also show cognitive distortions: hostile attribution bias, where ambiguous behaviour is interpreted as aggressive, leading to violence, and minimalisation, where the

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seriousness of the crime is played down (Barbaree, 1991, found that 54% of rapists denied their offence and 40% minimised it).

Psychopathic personality: psychopaths feel no guilt, remorse or empathy. Hare's Psychopathy Checklist (PCL-R) assesses traits such as superficial charm, manipulation, pathological lying, impulsivity and irresponsibility. These traits allow them to harm others without distress. Psychopaths are estimated to make up around 1% of the population but a much larger proportion of prisoners. However, Theodorakis (2013) argued that psychopathy needs more investigation before we can say with certainty that there is a direct correlation between psychopathy scores and criminal behaviour.

04 (b)

Restorative justice: from the victim's perspective it is effective. The Restorative Justice Council (2015) reported 85% victim satisfaction when victims met their offenders face to face, as they can ask questions, explain the harm and gain closure. Sherman and Strang's (2007) review found that RJ reduced reoffending in many trials, especially for violent crimes, and reduced victims' post-traumatic stress symptoms. However, it only works for some offenders and victims: the offender must admit guilt and be willing, so the samples are self-selected and may be more likely to succeed anyway, and some victims may be re-traumatised. Zehr (2002) argued RJ can take place without the offender present, for example with surrogate offenders, so more victims can benefit. Overall, RJ is effective for victims and for some offenders, but not as a universal method.

Anger management: research shows some success. Landenberger and Lipsey's (2005) meta-analysis of CBT programmes for offenders found an average 25% reduction in reoffending, with anger control components linked to greater effects, and Taylor and Novaco (2006) found anger treatment reduced anger in offenders with intellectual disabilities. However, Howells et al. (2005) found only small, non-significant overall effects in an Australian study. CBT is not for

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everyone: some offenders will not reflect on their thinking and drop out, whereas drama-based courses are more engaging (Blacker et al., 2008), and success depends on 'readiness to change' (Howells and Day, 2003). Loza and Loza-Fanous (1999) found no difference in anger between violent and non-violent offenders in a study of over 300 male prisoners, so anger management may be irrelevant for many crimes and may give offenders an excuse to avoid responsibility.

04 (c)

The amygdala explanation suggests that abnormal amygdala functioning (Raine et al., 1997) means some offenders feel little fear or empathy. Restorative justice could be applied to build empathy by making them face the emotional harm caused, although it may be less effective for those with severe amygdala dysfunction. Anger management targets the physiological arousal produced by the amygdala, teaching offenders to recognise bodily cues and use relaxation before they react.

The disinhibition hypothesis states that alcohol and drugs reduce prefrontal control over impulses, so treatment could include reducing substance use, with anger management teaching self-control strategies to replace inhibition. Inherited criminality (e.g. the MAOA gene) does not mean crime is inevitable, so at-risk people could be offered early intervention.

06 (a)

Holmes and Rahe's life events explanation proposes that major life changes, such as divorce, bereavement, moving house or starting a new job, are a source of stress because each change requires psychological adjustment. The more change a person experiences, the more energy and resources they must use to readjust, and this cumulative demand makes them vulnerable to stress-related illness. Both positive events, such as marriage, and negative events, such as being fired, are stressful because it is the amount of change that matters.

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To measure this, Holmes and Rahe (1967) examined the medical records of about 5,000 patients and identified 43 life events that often occurred in the months before illness. They asked 394 participants to rate how much readjustment each event would require compared with marriage, which was given an arbitrary value of 500. The average ratings were divided by ten to produce life change units (LCUs), forming the Social Readjustment Rating Scale (SRRS). For example, death of a spouse scores 100, divorce 73, marriage 50, retirement 45 and Christmas 12. A person ticks the events they have experienced in the past year and their LCUs are totalled. Holmes and Rahe suggested that a score of 300 or more in a year greatly increases the risk of stress-related illness, and 150 to 299 gives a moderate risk.

The link to illness is explained physiologically: the stress of readjustment activates the sympathomedullary pathway and the HPA axis, releasing cortisol, and prolonged cortisol suppresses the immune system, making illness more likely. Rahe et al. (1970) tested this with about 2,500 male US Navy sailors. Before a six-month tour of duty, the sailors completed a version of the SRRS for the previous six months, and their illness records were kept during the tour. There was a positive correlation of +0.118 between LCU scores and illness, supporting the idea that life events are linked to illness.

One strength is that the SRRS is quick, standardised and objective, giving a numerical score that can be compared and used in large-scale research. There is supporting evidence: in addition to Rahe et al., Kendler et al. (1999) found that stressful life events were strongly associated with the onset of major depression in a large twin sample. The explanation is also useful because it can identify people at risk, who could be offered support or stress management such as SIT.

However, the evidence is correlational, so we cannot conclude that life events cause illness. Rahe's correlation of +0.118 is significant but very weak, meaning life events explain only a small amount of the variation in illness, and a third variable, such as anxiety, could make people both report more events and more illness. Rahe's sample was all male sailors in a particular military setting, so the

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findings may not generalise to women or civilians, which suggests gender bias and limited population validity. The SRRS was also developed in 1960s America, so some events and weightings, such as 'mortgage over $10,000', are culturally and historically specific.

A major weakness is that the SRRS ignores individual differences in appraisal. Lazarus argued that stress depends on how a person perceives an event and their ability to cope, yet the scale assumes an event means the same to everyone. Divorce may be devastating for one person but a relief for another. It also does not separate positive and negative events, although research suggests negative, uncontrollable events are more harmful; the Life Experiences Survey (Sarason et al., 1978) improved on this by letting people rate events as positive or negative. In addition, the measure relies on retrospective self-report, so people may forget events or, if ill, search for explanations and over-report them, reducing validity.

An alternative is the daily hassles explanation. Kanner et al. (1981) developed the Hassles and Uplifts Scale and found that hassles were a better predictor of psychological symptoms than life events, and DeLongis et al. (1982) found hassles were more strongly related to health. Major life events are rare, whereas hassles are frequent and may accumulate; indeed, life events may cause stress partly through the hassles they create, such as the extra jobs after a bereavement. The explanation also ignores social support and individual differences such as hardiness and locus of control, which affect how well people cope.

In conclusion, the life events explanation was important in establishing that psychological factors can contribute to illness, and the SRRS remains useful for research. However, the weak correlations, biased samples and failure to consider appraisal, hassles and individual differences mean life events are only a small part of the explanation of stress.

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06 (b)	Type A people are competitive, impatient and hostile, which increases physiological reactivity and the risk of heart disease. Stress inoculation training (SIT) could be applied: in the conceptualisation phase they learn how their time urgency and hostility cause stress, then acquire skills such as relaxation and challenging irrational thoughts, and practise these in real situations. Friedman et al.'s (1986) Recurrent Coronary Prevention Project showed that counselling to reduce Type A behaviour reduced repeat heart attacks. Beta blockers could also reduce their high heart rate and blood pressure, although they do not change the personality. Hardiness (Kobasa) involves control, commitment and challenge, so SIT could build hardiness by encouraging people to see stressors as challenges. Self-efficacy (Bandura) could be raised by the mastery experiences in SIT's application phase, increasing confidence to cope.
07	The statement suggests that because the definition of science has changed over time, it is hard to say whether psychology is a science. I agree to some extent: views of science have shifted from simple observation to falsification and paradigms, and psychology fits some definitions better than others. However, I will argue that the main difficulty comes from the variety of approaches and the nature of human behaviour rather than only from changes in science. Science is traditionally defined by key features, which can be remembered as FORCE: falsifiability, objectivity, replicability, control and empiricism, together with the hypothetico-deductive method, in which theories generate testable hypotheses. Psychology began with clear scientific aims. Wundt opened the first psychology laboratory in Leipzig in 1879, using controlled conditions and standardised instructions, although his method of introspection was subjective. Watson (1913) rejected introspection and argued psychology should study only observable behaviour, so behaviourism tried to make psychology a natural science.

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Skinner's operant conditioning research with rats and pigeons used controlled experiments and produced universal principles of learning, such as reinforcement schedules, that can predict behaviour and have been replicated many times.

Early views of science were inductivist: scientists collected observations and built general laws from them. Popper (1959) changed this by arguing that science advances through falsification: a theory is scientific only if it could be proved false, and no number of confirming observations can prove it true. By this definition, much of psychology is scientific. The cognitive approach uses lab experiments to test falsifiable hypotheses, such as Loftus and Palmer (1974), who found that the verb 'smashed' produced higher speed estimates than 'contacted'. Biological psychology uses objective methods such as fMRI and PET scans; for example, Raine et al. (1997) found reduced prefrontal activity in murderers. However, Freud's psychodynamic theory fails Popper's test, as concepts such as the unconscious and the Oedipus complex cannot be tested, and any outcome can be explained; Popper used it as an example of pseudoscience. Freud's evidence came from case studies such as Little Hans, interpreted subjectively.

Kuhn (1962) changed the definition again, arguing that a mature science has a single paradigm, a shared set of assumptions and methods, and progresses through scientific revolutions, or paradigm shifts. He argued that psychology is pre-paradigmatic, as it has no agreed paradigm: the biological, behaviourist, cognitive, psychodynamic and humanistic approaches disagree about what should be studied and how. Others argue that psychology has had paradigm shifts, such as the cognitive revolution of the 1950s and 1960s that replaced behaviourism, and today cognitive neuroscience is becoming a dominant paradigm. This supports the statement: whether psychology is a science depends on which definition is used, Popper's or Kuhn's. Lakatos added that science consists of research programmes with a protected core, and Feyerabend argued there is no single scientific method at all, which makes the boundary between science and non-science even less clear.

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Developments in the natural sciences have also changed the meaning of objectivity. Heisenberg's uncertainty principle showed that the act of measurement can alter what is observed, even in physics. Similarly, in psychology, participants react to being studied: Orne (1962) showed that demand characteristics change behaviour, and experimenter bias (Rosenthal) shows that researchers' expectations influence results. If even physics accepts that observation is not completely objective, psychology may be no less scientific than other subjects, which suggests the changing nature of science has helped rather than hindered psychology's claim.

However, the difficulty may come from psychology itself. Human behaviour is affected by many variables, such as culture, emotion and free will, so few universal laws exist. Lab experiments achieve control but often lack ecological validity, so scientific rigour can come at the cost of explaining real behaviour. Some approaches deliberately reject the scientific method: humanistic psychologists such as Rogers argued that each person's subjective experience is unique, so an idiographic approach, using methods such as interviews and Q-sorts, is more appropriate than a nomothetic one searching for general laws. Qualitative methods such as thematic analysis are valued for depth, not replicability. For these approaches, being scientific is not the aim, so the question of scientific status depends on the approach rather than the definition of science.

The replication crisis has also raised doubts. The Open Science Collaboration (2015) attempted to replicate 100 published psychology studies and found that only about 36% produced significant results, compared with 97% of the originals, which questions replicability, a key feature of science. However, the response to the crisis, with preregistration of hypotheses, open data and larger samples, shows psychology correcting itself, which is itself a feature of science. Similar replication problems have been found in medicine and biology, so this is not unique to psychology.

In conclusion, I partly agree with the statement. The changing definitions of

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science, from inductivism to Popper's falsification, Kuhn's paradigms and Feyerabend's rejection of a single method, do make it harder to give a fixed answer, as psychology meets some criteria but not others. However, the main reason for the difficulty is that psychology is a diverse discipline: biological, behaviourist and cognitive psychology clearly use scientific methods and produce falsifiable, replicable findings, whereas psychodynamic and humanistic approaches do not aim to. Psychology is therefore best seen as a subject with scientific and non-scientific parts, using the scientific method where it is appropriate to the behaviour being studied.