GCE A LEVEL

A290U30-1

S19-A290U30-1

PSYCHOLOGY – A level component 3
Psychology: Implications In The Real World

FRIDAY, 14 JUNE 2019 – MORNING

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 pencil or gel pen. Do not use 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

e.g. | **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 the characteristics of addictive behaviours. [10]

b) Nigel works with people with a variety of addictive behaviours. One of his clients, Richard, has read about individual differences explanations and believes they fully explain his addiction. Nigel disagrees.

Evaluate individual differences explanations of addictive behaviours with reference to Nigel and Richard. [15]

0 2 **Autistic spectrum behaviours**

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

b) Patrick has autistic spectrum behaviour and wants to understand the potential causes of it. Patrick understands that social psychology can explain his behaviour, especially his interactions with others.

Evaluate social psychological explanations of autistic spectrum behaviours with reference to Patrick. [15]

0 3 **Bullying behaviours**

a) Mike, unlike his sister Caroline, is a persistent bully. He is often verbally and physically aggressive. One psychologist he visited suggested that the cause of his behaviours could be biological.

Describe **one** biological explanation of bullying behaviours with reference to the scenario above. [15]

b) Evaluate **one** social psychological explanation of bullying behaviours. [10]

0 4 **Criminal behaviours**

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

b) 'When modifying criminal behaviours, social implications are more important than ethical implications.'

Evaluate methods of modifying criminal behaviours with reference to this statement. [15]

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0 5 **Schizophrenia**

a) Evaluate **two** individual differences explanations of schizophrenia. [10]

b) Edna is trying to understand what might have caused her to have schizophrenia. Her symptoms include hearing voices, being paranoid, disordered thinking and being less sociable. In a session with her psychologist, biological explanations were discussed.

Describe **one** biological explanation for schizophrenia, making reference to Edna's symptoms in your answer. [15]

0 6 **Stress**

a) Describe **two** individual differences explanations of stress. [10]

b) 'Biological explanations of stress are limited.'

Evaluate biological explanations of stress with reference to this statement. [15]

SECTION B: Controversies

Answer **one** *of the questions.*

Either,

0 7 **Sexism**

'Psychology continues to suffer from sexism.'

Using your knowledge of psychology, discuss the extent to which this statement is true. [25]

Or,

0 8 **Scientific status**

'Psychology can never have scientific status because of the methodologies used by various approaches.'

Discuss to what extent 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.

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03 (a)

One biological explanation of bullying behaviour is the role of hormones, especially testosterone. Bullying involves repeated, deliberate aggression towards a less powerful victim, and Mike's verbal and physical aggression fits this, so a hormonal explanation focuses on the biochemical basis of his aggression and dominance.

Testosterone is an androgen produced mainly by the testes in males, and in much smaller amounts by the ovaries and adrenal glands in females. Adult males have far higher levels than females. It acts on areas of the brain involved in aggression, such as the amygdala and hypothalamus, and is thought to reduce the influence of the orbitofrontal cortex, which normally controls impulses. This could explain why Mike is a persistent bully but his sister Caroline is not: although they share a family environment, Mike, as a male, will have much higher circulating testosterone, making aggressive and dominant behaviour more likely. Testosterone also acts before birth, as prenatal exposure masculinises the developing brain, so Mike's brain may have been organised to respond more aggressively from the start.

Research into hormones supports this link. Nelson (1995) reviewed research into how hormones influence aggression and found a positive correlation between the level of circulating androgens and aggressive behaviour, for example in prisoners, where those convicted of violent crimes tended to have higher testosterone levels than those convicted of non-violent crimes. Animal research shows a causal role: Wagner et al. (1979) found that castrating male mice, which removes the main source of testosterone, reduced their aggression, and that injecting them with testosterone restored it. Pillay (2006) found a link between testosterone and aggression in athletes, with higher levels in those taking part

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in more aggressive sports. Mike's physical aggression in particular may therefore reflect high testosterone.

Two models explain how testosterone affects behaviour. The basal model proposes that a person's stable baseline level of testosterone influences their tendency towards dominance and aggression, so Mike may simply have a naturally high baseline, which would make his bullying persistent across situations. The reciprocal model proposes that testosterone and behaviour influence each other: winning a dominance encounter raises testosterone, which encourages further dominant behaviour, whereas losing lowers it. This could explain why Mike's bullying continues: each time he successfully intimidates a weaker child, his testosterone may rise, reinforcing his tendency to bully again.

Other hormones may interact with testosterone. Low levels of cortisol, the stress hormone, are linked to low fear and anxiety, and according to the dual-hormone hypothesis aggression is most likely when testosterone is high and cortisol is low, because the person feels little fear of the consequences. Mike may therefore not feel anxious about punishment for bullying. Low serotonin is also linked to impulsive aggression, as serotonin normally inhibits responses to emotional stimuli.

Sex differences in hormones can also explain the different forms bullying takes. Males are more likely to use direct physical and verbal bullying, as Mike does, whereas females with lower testosterone more often use indirect, relational bullying such as spreading rumours and social exclusion. So even if Caroline were aggressive, a hormonal explanation would predict her aggression to be less physical than Mike's.

However, as Simpson (2001) noted, aggressive behaviour is complex and multifaceted, so testosterone is best seen as increasing Mike's predisposition to bully, with environmental triggers such as frustration, provocation or a peer group that rewards dominance determining whether and when he acts aggressively.

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03 (b)

Moral disengagement is a social psychological explanation based on Bandura's social cognitive theory. It proposes that most people hold moral standards that stop them harming others, but bullies use cognitive mechanisms to switch these off without feeling guilt. These include moral justification ('he deserved it'), euphemistic labelling ('it was just a joke'), minimising the consequences, displacement or diffusion of responsibility ('everyone was doing it'), dehumanising the victim and blaming the victim.

There is research support. Hymel et al. (2005) surveyed 494 Canadian secondary school students and found that students who bullied more often had higher moral disengagement scores, with moral disengagement accounting for a large proportion (about 38%) of the variance in bullying. Gini et al.'s (2014) meta-analysis of 27 studies found a significant positive association between moral disengagement and aggressive behaviour, including bullying, in children and adolescents, so the link appears reliable across many samples and countries. The explanation also has real-world applications: anti-bullying programmes such as KiVa in Finland target the justifications and diffusion of responsibility that support bullying, encouraging bystanders to empathise with victims and intervene.

However, most of this research is correlational, so it cannot show that moral disengagement causes bullying. It is equally possible that children who bully develop justifications afterwards to reduce the cognitive dissonance caused by their behaviour, or that a third variable, such as low empathy, causes both. The research also relies on self-report questionnaires, which are vulnerable to social desirability bias, as bullies may under-report their behaviour and some victims may be misidentified, reducing validity. There are also problems with defining bullying consistently across studies, which makes comparisons difficult.

The explanation ignores biological factors. Ball et al. (2008) studied over 1,000 families with twins and found that about 61% of the variation in bullying was due to genetic factors, which moral disengagement cannot explain. It also struggles to explain sex differences in bullying, which evolutionary explanations

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account for in terms of different strategies for gaining status and mates (Volk et al., 2012). In addition, it describes how bullies justify aggression but not why some children become aggressive in the first place. Gini et al. (2006) also found that bullies do not necessarily lack understanding of others' minds, suggesting that some bullies are skilled manipulators, so cognition is not the only factor.

In conclusion, moral disengagement is well supported as a factor associated with bullying and has useful applications in school programmes, but correlational evidence means causation is uncertain. A diathesis-stress approach, in which a biological predisposition interacts with social-cognitive factors such as moral disengagement, gives a more complete explanation.

05 (a) The schizophrenogenic mother, a psychodynamic explanation by Fromm-Reichmann (1948), proposes that schizophrenia stems from a cold, rejecting yet overprotective and controlling mother, which creates tension and distrust in the child, leading to paranoia and regression. There is some early support: Kasanin et al. (1934) studied the case histories of 45 patients with schizophrenia and found maternal overprotection in 33 cases. However, the evidence is weak. These were case studies based on retrospective reports and the researchers' interpretations, which may have been biased by their expectations, and later research has failed to find a consistent personality type in the mothers of people with schizophrenia. The concepts are difficult to test, so the theory is unfalsifiable. The explanation has limited usefulness because it has not produced an effective therapy, and it is ethically problematic as it blames mothers, causing distress to families already coping with the illness. Liem (1974) found that parents' communication with their schizophrenic sons was no different from other parents, suggesting any abnormal family interaction is a response to the illness rather than a cause.

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The cognitive explanation of thought disorder argues that symptoms result from faulty information processing. Frith (1992) proposed a deficit in metarepresentation, so the person cannot recognise their thoughts as self-generated and experiences them as voices, and a deficit in central control, causing disorganised speech. This has much better scientific support. Barch et al. (1999) found that patients with schizophrenia had difficulty using context information to guide their responses, and Stirling et al. (2006) found patients were much slower than controls on the Stroop task, suggesting a deficit in suppressing automatic responses. It has also led to an effective treatment, CBT, which helps patients test the reality of their beliefs. However, the explanation describes the symptoms rather than their cause: it does not explain why the processing faults occur, and cognitive deficits may be a result of the illness, or of medication, rather than its origin. Biological explanations fill this gap, for example Gottesman (1991) found concordance of about 48% for identical twins compared with 17% for non-identical twins, and Heston (1966) found that 5 of 47 adopted children of mothers with schizophrenia developed the disorder, compared with none in a control group.

In conclusion, the thought disorder explanation is more scientific and useful than the schizophrenogenic mother, but both ignore genetic and biochemical factors, so they are best combined with biological explanations in a diathesis-stress model.

05 (b)

The dopamine hypothesis states that schizophrenia is caused by abnormal activity of the neurotransmitter dopamine. In its original form it proposed that people with schizophrenia have too much dopamine, or too many or oversensitive dopamine receptors, especially D_2 receptors (one of five receptor types, D_1 to D_5), so dopamine neurons fire too often and transmit too many messages. This excess activity is associated with psychotic behaviours.

The hypothesis developed from drug evidence. Amphetamines increase dopamine

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activity, and large doses given to people without psychosis can quickly produce symptoms similar to schizophrenia, such as hallucinations and paranoid delusions, while L-dopa, a drug that raises dopamine in Parkinson's disease, can also cause psychotic symptoms. Conversely, antipsychotic drugs such as chlorpromazine block D_2 receptors and reduce these symptoms, and the clinical strength of these drugs is closely related to how strongly they bind to D_2 receptors.

The revised dopamine hypothesis (Davis et al., 1991) proposes that different symptoms come from different dopamine pathways. Hyperdopaminergia, excess dopamine in the mesolimbic pathway, which links the ventral tegmental area to the nucleus accumbens and limbic system, produces positive symptoms.

Hypodopaminergia, too little dopamine in the mesocortical pathway to the prefrontal cortex, produces negative symptoms and cognitive problems.

This can explain Edna's symptoms. Her hearing voices is an auditory hallucination, a positive symptom. Excess dopamine in subcortical areas, which also affects areas involved in speech such as Broca's area, may mean that Edna's own inner speech is experienced as coming from an external voice. Her paranoia is a persecutory delusion, also a positive symptom. Kapur (2003) proposed that excess dopamine in the mesolimbic pathway causes aberrant salience: ordinary events, such as people talking near her, are given exaggerated importance, and Edna then develops the belief that she is being watched or plotted against to make sense of these strange experiences.

Edna's disordered thinking can be linked to both pathways. Excess mesolimbic dopamine produces a flood of salient thoughts and associations, so her thinking may jump between unconnected ideas (derailment), and low dopamine in the prefrontal cortex impairs working memory and executive functions, making it difficult for her to organise her thoughts logically. Her being less sociable is a negative symptom, such as social withdrawal, avolition and lack of emotional expression. According to the revised hypothesis, this is caused by hypodopaminergia in the mesocortical pathway, as the prefrontal cortex is involved in motivation and social behaviour, so Edna may lack the drive to seek

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out others.

The dopamine hypothesis would therefore suggest that Edna has a dopamine imbalance, with excess dopamine activity in the mesolimbic pathway producing her voices and paranoia, and too little in the prefrontal cortex producing her reduced sociability and contributing to her disordered thinking. This abnormality could be inherited, as genes linked to dopamine regulation have been associated with schizophrenia, or triggered by stress or drug use, such as cannabis, whose active ingredient THC increases dopamine release in vulnerable people. On this view, her psychologist would expect antipsychotic medication that alters dopamine activity to reduce her symptoms, with atypical drugs such as clozapine being more likely to help her negative symptoms as well as her voices and paranoia.

06 (a)

Hardiness: Kobasa (1979) proposed that some people have a hardy personality that protects them from the negative effects of stress. Hardiness has three components, the '3 Cs'. Control is the belief that you can influence events in your life rather than being a victim of circumstances. Commitment is a sense of involvement and purpose in work, relationships and activities, so the person engages fully rather than withdrawing. Challenge means seeing change and difficulties as opportunities for growth rather than threats. Hardy people therefore appraise stressors as less threatening and use active, problem-focused coping, so their physiological stress response is reduced and they are less likely to develop stress-related illness. Kobasa studied around 800 male business executives in the USA using questionnaires on life events and illness. Those who had experienced high levels of stress but little illness scored higher on all three components of hardiness than those with high stress and high illness.

Type A and Type B personality: Friedman and Rosenman proposed that people with a Type A personality are more vulnerable to stress-related illness, especially coronary heart disease (CHD). Type A individuals are competitive and

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achievement-striving, impatient with a sense of time urgency, and hostile towards others. Type B individuals are relaxed, patient, calm and less competitive, and more able to express their emotions. Type A people perceive more situations as stressful, so the sympathomedullary pathway is repeatedly activated, releasing adrenaline and noradrenaline, raising heart rate and blood pressure, and cortisol is released through the HPA axis. Over time this damages blood vessels and increases the risk of CHD. In the Western Collaborative Group Study, Friedman and Rosenman followed 3,154 healthy men aged 39 to 59 for eight and a half years. 257 developed CHD, and 70% of these were Type A, so Type A men were about twice as likely to develop CHD as Type B men, even after controlling for factors such as smoking. Later research identified hostility as the most harmful component.

06 (b)

Biological explanations of stress include the role of adrenaline, evolutionary adaptation (the fight or flight response) and stress genes. The statement claims they are limited. I will argue that they explain the physiological stress response and its effects on health well, but on their own they cannot explain why people respond to stress so differently.

The adrenaline explanation states that when a stressor is perceived, the hypothalamus activates the sympathomedullary pathway and the adrenal medulla releases adrenaline, increasing heart rate and blood pressure, while the HPA axis releases cortisol. There is strong evidence that this response damages health. Leor et al. (1996) found that on the day of the 1994 Los Angeles earthquake, sudden cardiac deaths rose to 24, compared with a daily average of about 4.6 in the preceding week, suggesting that acute stress, through a surge of adrenaline, can trigger heart attacks in vulnerable people. Kiecolt-Glaser et al. (1984) found that medical students' immune functioning was lower during exams, showing that the stress response affects health. Heim et al. (2000) found that women who had experienced childhood abuse showed greater

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pituitary-adrenal and autonomic responses to stress, showing how stress hormones are linked to experience. This objective, physiological evidence goes against the statement, as it shows a clear biological mechanism.

However, there are problems with cause and effect. Much of the research is correlational or natural experiments, so it cannot show that adrenaline alone causes illness: the people who died in the earthquake were likely to already have heart disease. Dimsdale (2008) reviewed research on stress and cardiovascular disease and noted that stress is not always a causal factor, as the relationship is complex and involves lifestyle behaviours such as smoking and poor diet, which people often use to cope with stress. This supports the statement.

The evolutionary explanation argues that fight or flight evolved to help our ancestors survive immediate physical threats, such as predators, by preparing the body for action. This explains why the response is so fast and automatic.

However, modern stressors, such as exams or work deadlines, are usually psychological and long-lasting, so the response is no longer adaptive and may harm health, which the explanation does not fully address. Cannon's research was mostly on male animals, and Taylor et al. (2000) argued that females are more likely to 'tend and befriend', protecting offspring and seeking social support, linked to oxytocin. This suggests a gender bias, supporting the statement that biological explanations are limited.

Stress genes may explain why some people are more vulnerable. Caspi et al. (2003) found that people with the short version of the serotonin transporter gene (5-HTT) were more likely to become depressed after stressful life events than those with the long version, and variations in the FKBP5 gene, which regulates the cortisol response, have been found to interact with childhood abuse to predict PTSD symptoms (Binder et al., 2008). This supports a biological basis for individual differences in stress. However, these findings show a gene-environment interaction rather than genes causing stress alone, and no single gene explains a large amount of variance.

The biggest limitation is that biological explanations ignore cognitive appraisal.

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Lazarus argued that stress only occurs when a person appraises a situation as exceeding their ability to cope, so the same event can be stressful for one person but not another. Individual differences research shows this: Kobasa found that hardy executives became ill less often, Friedman and Rosenman linked Type A behaviour to CHD, and Cohen et al. (1993) found that people with higher life stress were more likely to develop a cold after being exposed to a cold virus. Social support also buffers the stress response. These factors cannot be explained by adrenaline or genes alone, so biological explanations are reductionist. On the other hand, biological explanations have led to effective treatments, such as beta blockers, which block the action of adrenaline and reduce physical symptoms, and they can be studied objectively using physiological measures such as heart rate and cortisol levels, which makes them more scientific than self-report measures of appraisal.

In conclusion, the statement is true to an extent. Biological explanations describe how the body responds to stress and how this can lead to illness, with strong evidence such as Leor et al. However, they are limited because they cannot explain individual and gender differences or the role of appraisal, personality and social support. The best explanation is a biopsychosocial one that combines biology with psychological and social factors.

08

The statement claims that psychology can never be a science because of the methods its approaches use. I partly agree: some approaches use methods that are not scientific, and human behaviour is hard to study scientifically. However, other approaches use rigorous scientific methods, so 'never' is too strong.

Science is usually defined by key features, remembered as FORCE: falsifiability, objectivity, replicability, control and empiricism, together with the hypothetico-deductive method, where theories generate testable hypotheses that are tested and the theory is refined. Psychology had scientific origins: Wundt opened the first psychology laboratory in Leipzig in 1879, using controlled

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conditions and standardised instructions, although his method of introspection was subjective and could not be verified by others.

Some approaches clearly meet these criteria. Watson (1913) rejected introspection and argued that psychology should study only observable behaviour, so behaviourism used tightly controlled laboratory experiments. Skinner's research with rats and pigeons in the Skinner box measured objective responses such as lever pressing and produced universal principles of learning, such as the effects of reinforcement schedules, which have been replicated many times and allow behaviour to be predicted and controlled, for example through token economies. This is strong evidence against the statement.

Biological psychology also uses scientific methods. Brain imaging techniques such as fMRI and PET scans give objective, quantifiable data: Raine et al. (1997) found reduced activity in the prefrontal cortex in PET scans of murderers compared with matched controls. Twin and adoption studies, drug trials using double-blind placebo controls and measures of hormones such as cortisol all emphasise objectivity and control. Cognitive psychology uses laboratory experiments to test falsifiable hypotheses about mental processes; for example, Loftus and Palmer (1974) manipulated the verb in a question and found that 'smashed' produced higher speed estimates than 'contacted', and Baddeley's research on working memory used controlled experiments that have been replicated. These approaches show that psychology can achieve scientific status.

However, other approaches use methods that are not scientific. The psychodynamic approach relies on case studies and interpretation, such as Freud's study of Little Hans, where evidence came largely from the boy's father and was interpreted to fit the theory. Concepts such as the unconscious and the Oedipus complex cannot be directly observed or measured, so the theory is unfalsifiable: Popper (1959) used it as an example of a pseudoscience, as any outcome could be explained. The methods depend on the analyst's subjective interpretation, so findings are neither objective nor replicable. This supports the statement.

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The humanistic approach goes further, rejecting the scientific method deliberately. Rogers argued that each person's subjective experience is unique, so an idiographic approach using methods such as unstructured interviews and Q-sorts is more appropriate than a nomothetic search for general laws. Here the focus on the individual is more important than generalisable rules, so this approach would never claim scientific status, again supporting the statement.

The subject matter also limits psychology's methods. Human behaviour is affected by many variables, such as culture, emotion, past experience and possibly free will, so it is very difficult to control all extraneous variables, and psychology has few universal laws compared with the natural sciences.

Participants react to being studied: Orne (1962) showed that demand characteristics change behaviour, and Rosenthal showed that experimenter expectations can influence results, so complete objectivity is difficult. When laboratory experiments do achieve control, they often lack ecological validity, so scientific rigour can come at the cost of understanding real behaviour. Some behaviours, such as the experience of a mental disorder or the effects of trauma, cannot ethically be manipulated in experiments and are better studied with qualitative methods. It could be argued that it is not right to be scientific when the subject matter is not best analysed in that way.

Kuhn (1962) argued that a mature science has a single paradigm, a shared set of assumptions and methods, and that psychology is pre-paradigmatic because its approaches disagree about what to study and how. This suggests the variety of methods prevents psychology being a science. However, it can be argued that psychology has had paradigm shifts, such as the cognitive revolution, and that cognitive neuroscience, which combines cognitive experiments with brain scanning, is becoming a dominant, scientific paradigm.

The replication crisis is also relevant. The Open Science Collaboration (2015) tried to replicate 100 published psychology studies and found that only about 36% produced significant results again, questioning replicability. Yet psychology's response, including preregistration, open data and larger samples, shows it

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correcting itself through scientific processes, and similar problems exist in medicine. Even physics accepts, through Heisenberg's uncertainty principle, that measurement can affect what is observed, so psychology's problems with objectivity are not unique.

In conclusion, I agree with the statement only to a limited extent. The psychodynamic and humanistic approaches use subjective methods, such as case studies and interviews, that lack falsifiability, objectivity and replicability, and the complexity of human behaviour makes complete control impossible. However, biological, behaviourist and cognitive psychology use the hypothetico-deductive method, laboratory experiments and objective measures, and produce falsifiable, replicable findings. Psychology is therefore best seen as a discipline with scientific and non-scientific parts, so it is wrong to say it can never have scientific status.