GCE A LEVEL

A290U30-1

S18-A290U30-1

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

THURSDAY, 14 JUNE 2018 – 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.
Answer **three** questions in Section A and **one** question in Section B.
Write your answers in the separate answer booklet provided.

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.*

1. Addictive behaviours

(a) Natalie likes going to the casino. At first she would go only once a month. Now she goes at least four times a week. She realises she has an addiction to gambling, and is seeking help. Her psychologist has suggested a number of different methods of modifying this behaviour.

Describe **one** method of modifying addictive behaviour with reference to Natalie. [15]

(b) Evaluate **two** individual differences explanations of addictive behaviour. [10]

2. Autistic spectrum behaviours

(a) Outline the characteristics of autistic spectrum behaviour. [10]

(b) 'Biological explanations are the best way of explaining autistic spectrum behaviours.'

From your study of autistic spectrum behaviours, discuss to what extent this statement is true. [15]

3. Bullying behaviours

(a) Outline the characteristics of bullying behaviours. [10]

(b) 'Individual differences explanations of bullying are an effective means of explaining bullying behaviour.'

With reference to this statement, evaluate individual differences explanations of bullying behaviours. [15]

4. Criminal behaviours

(a) Describe **one** social psychological explanation of criminal behaviour. [10]

(b) 'Criminality is the clear outcome of biological processes.'

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

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5. Schizophrenia

(a) Ronald has not been feeling the same lately. He told his friend Rory what he has been experiencing. Rory suggested that the behaviours Ronald is showing could be characteristics of schizophrenia.

Describe the characteristics of schizophrenia that Ronald may have told Rory he was experiencing. [15]

(b) Evaluate social psychological explanations of schizophrenia. [10]

6. Stress

(a) Describe **two** biological explanations for stress. [10]

(b) Mehdi is highly stressed about his examinations, driving test and other personal issues. Various methods of modifying this behaviour have been suggested to him.

Evaluate **one** method of modifying Mehdi's stress. [15]

SECTION B: Controversies

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

Either,

7. Cultural bias

'Psychological research is culturally biased.'

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

Or,

8. Non-human animals

'Using non-human animals in psychology is useful but also problematic.'

To what extent do you agree with this statement? Justify your answer using your psychological knowledge. [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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1 (a) One method of modifying addictive behaviour is aversion therapy, which comes from the behaviourist approach. It assumes that addictions are learned, so they can be unlearned. It uses classical conditioning to create a new association between the addictive behaviour and something unpleasant, so that the person comes to feel discomfort rather than pleasure and their cravings are reduced.

Before therapy, Natalie has learned to associate the casino with pleasure: the lights, the sounds of the machines and the excitement of a possible win give her a 'buzz'. Aversion therapy aims to replace this pleasant association with an unpleasant one.

First, an unconditioned stimulus (UCS) that naturally produces an unpleasant unconditioned response (UCR) is chosen. For gambling this is usually a mild but painful electric shock, which naturally produces pain and fear (UCS → UCR). At this stage, gambling cues are a neutral stimulus (NS) in that they produce no unpleasant response.

During conditioning, the neutral stimulus is repeatedly paired with the UCS (NS + UCS → UCR). In Natalie's sessions the psychologist could show her gambling-related stimuli, such as photographs of the casino, roulette wheels and slot machines, or words such as 'bet' and 'jackpot', or ask her to read out cards describing her gambling, and each time give her a brief shock to her arm or fingers. Over many sessions, the gambling cues become associated with the pain and fear of the shock.

After conditioning, the gambling cues have become a conditioned stimulus (CS) that produces a conditioned response (CR) of anxiety and discomfort on their own, without any shock (CS → CR). When Natalie thinks about going to the casino, or walks past one, she should feel anxious and uncomfortable rather

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than excited. In order to avoid this unpleasant feeling she would be motivated to stay away, so her visits should fall from four times a week back towards none. Because the behaviour is no longer rewarded with pleasure, her cravings should also weaken.

Natalie would need to give fully informed consent, as the therapy is deliberately unpleasant, and the shocks must be strong enough to be aversive but never harmful. Because a conditioned response can extinguish if the CS is repeatedly presented without the UCS, she may need booster sessions to maintain the association. As the casino is not present in the clinic, Natalie could also be given a self-administered version, such as snapping an elastic band on her wrist whenever she has an urge to gamble, so the conditioning continues in her everyday life.

A less distressing alternative is covert sensitisation, where no real aversive stimulus is used. Natalie would be taught to relax and then to imagine herself entering the casino and placing a bet, immediately followed by imagining highly unpleasant scenes, such as feeling sick, losing all her savings or her family's disappointment. The imagined consequences act as the UCS, so the urge to gamble becomes associated with disgust and anxiety.

McConaghy et al. (1983) used aversion therapy with compulsive gamblers, which shows this method has been applied to gambling addiction. For Natalie, aversion therapy is most likely to succeed if it is combined with other support, such as counselling or CBT, that helps her deal with the reasons she began gambling more often.

1 (b)

Personality: Eysenck suggested that people high in neuroticism, extraversion and psychoticism are more vulnerable to addiction, for example because extraverts seek stimulation and neurotic individuals use substances or gambling to manage anxiety. This is supported by Dong et al. (2013), who found personality differences between people with internet addiction and those without, suggesting that particular traits are linked to addictive behaviour. A strength is that this

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could explain why only some people who try a drug or gamble become addicted. However, the evidence is largely correlational. It is unclear whether personality traits cause addiction or whether the addiction, and its effects on mood and relationships, changes personality. Rozin and Stoess (1993) found only weak correlations between addictions to different substances in the same people, which contradicts the idea of a general 'addictive personality' that makes a person vulnerable to any addiction. Personality questionnaires are also self-report measures, so social desirability bias may reduce validity. The explanation also ignores biological factors, such as dopamine activity in the reward pathway or genetic predisposition (addiction genes), which may underlie both the personality traits and the addiction.

Cognitive biases: this explanation suggests that addicts hold irrational beliefs that maintain their behaviour, such as the illusion of control (believing they can influence chance outcomes), the gambler's fallacy and recalling wins but forgetting losses. Griffiths (1994) supports this. He compared 30 regular and 30 non-regular fruit machine gamblers using a 'thinking aloud' method and found that regular gamblers made more irrational verbalisations (about 14% compared with 2.5%) and believed they were more skilful than they were, although there was no difference in actual skill. This shows that cognitive biases help maintain gambling.

However, Griffiths' study has methodological issues that affect the validity of the explanation. Thinking aloud may be unnatural and the coding of what counts as 'irrational' is subjective, while non-regular gamblers may have been unfamiliar with the machines, so differences may reflect experience rather than biased thinking. The explanation also cannot easily account for chemical addictions such as heroin, where physical dependence and withdrawal are central, and it does not explain whether biases cause the addiction or develop as a result of it. A strength is its practical application: CBT can challenge irrational beliefs and has been used successfully to treat gambling.

Both explanations also ignore social psychological factors, such as peer influence

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and the media, which may equally influence addictive behaviour. In conclusion, cognitive biases seem better supported, especially for gambling, while the addictive personality is weakened by Rozin and Stoess. Neither explains addiction fully, so a biopsychosocial approach is more valid.

5 (a) Schizophrenia is a severe mental disorder in which a person loses contact with reality. According to DSM-5, diagnosis requires two or more characteristic symptoms present for a significant part of at least one month, with continuous signs of disturbance for at least six months, and at least one must be delusions, hallucinations or disorganised speech. The characteristics are divided into positive symptoms, which are additions to normal experience, and negative symptoms, which are a loss of normal functioning. Ronald said he has 'not been feeling the same lately', which suggests a change from his usual functioning.

Positive symptoms: Ronald may have told Rory about hallucinations, which are perceptions without any external stimulus. They can occur in any sense: auditory hallucinations are the most common, so Ronald may hear voices commenting on what he is doing, criticising him or telling him what to do. He may also have visual hallucinations, such as seeing people or shapes that are not there, or tactile ones, such as feeling insects crawling under his skin.

Ronald may also describe delusions, which are fixed false beliefs held despite clear evidence that they are untrue. Delusions of persecution would involve Ronald believing that people are plotting against him, following him or trying to poison him. Delusions of grandeur would mean he believes he is someone very important or has special powers, such as being a famous person or able to save the world. Delusions of reference would mean he believes that ordinary events, such as a song on the radio or a news report, contain special messages meant for him. Delusions of control would involve Ronald believing his thoughts or actions are being controlled by an outside force, or that thoughts are being inserted into or withdrawn from his mind.

Formal thought disorder (disordered thinking) shows in Ronald's speech. When

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	talking to Rory he may jump between unrelated topics (loose associations or derailment), so his conversation is hard to follow. He may use neologisms, which are made-up words that only have meaning to him, or clanging, choosing words because they rhyme or sound alike, for example 'I was feeling blue, the shoe, the glue'. In extreme cases his speech may become an incoherent 'word salad'. Negative symptoms: Ronald may tell Rory that he has little to say, showing alogia (poverty of speech), giving short, empty replies and long pauses. He may have avolition, a lack of motivation to start or continue goal-directed activity, so he may have stopped going to work or college, not washed or tidied his home and given up hobbies he used to do with Rory. Anhedonia is the loss of pleasure in previously enjoyable activities, such as going out with friends or watching football, so Ronald might say nothing feels enjoyable anymore. He may show flattened affect, a reduced display of emotion, speaking in a monotone with a blank expression, or inappropriate affect, such as laughing when talking about something sad. He may also have withdrawn socially, explaining why he has 'not been feeling the same' and spending time alone. Ronald may also describe catatonic behaviour, which affects movement. This ranges from catatonic stupor, where he remains motionless in one position for long periods and is unresponsive, to catatonic excitement, involving purposeless repetitive or agitated movements, and echopraxia, where he mimics the movements of others, such as copying Rory's gestures, or echolalia, repeating Rory's words. Overall, if Ronald reported a combination of positive symptoms such as voices and persecutory delusions, and negative symptoms such as avolition and flat affect, over several months, Rory would be right that these are characteristics of schizophrenia, although only a clinician could diagnose him.
5 (b)	Social psychological explanations argue that schizophrenia is caused or triggered by the person's family and wider social environment. One explanation is dysfunctional families. Bateson et al.'s (1956) double bind theory suggests that

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a child who repeatedly receives contradictory messages from a parent, for example verbal affection with cold body language, cannot respond correctly, leading to confused thinking. Expressed emotion (EE) is a family communication style involving criticism, hostility and emotional over-involvement, which acts as a stressor. Vaughn and Leff (1976) found that about half of patients returning to high EE families relapsed within nine months, compared with about 12% of those from low EE families, which strongly supports EE as an explanation of relapse. This has a real-world application, as family therapy that reduces EE lowers relapse rates.

However, research on the double bind is less supportive. Liem (1974) studied the communication patterns of parents of schizophrenic and non-schizophrenic sons and found no evidence that the parents' communication was faulty in the way the theory predicts, suggesting that disturbed communication may be a response to the child's disorder rather than its cause. EE research explains relapse rather than onset, and causation is unclear, because a family may become critical as a result of coping with the person's symptoms. Early ideas such as Kasanin et al. (1934), who reported maternal overprotection in many cases, relied on case records and have been criticised for blaming parents.

Cultural and sociocultural factors are also suggested. Van Os (2005) found that the risk of schizophrenia is higher in people who grow up in urban areas (urbanicity), possibly because of the stress of social adversity and crowding. Jones et al. (1994) found in a large British birth cohort that children who preferred solitary play were more likely to develop schizophrenia later, supporting social isolation as a factor. However, these findings are correlational: the social drift hypothesis suggests that people developing schizophrenia move down the social scale and into cities because of their symptoms. Early social isolation may be an early sign of the disorder rather than a cause.

Social explanations also ignore strong biological evidence. Heston (1966) found that 5 of 47 children adopted away from mothers with schizophrenia developed it, compared with none of 50 controls, despite being raised in different families,

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and McCarley (1999) linked schizophrenia to structural brain abnormalities. Cognitive explanations such as Barch et al. (1999) point to deficits in processing context. In conclusion, social psychological factors cannot fully explain schizophrenia alone, but the diathesis-stress model suggests that stressors such as high EE trigger it in genetically vulnerable people.

6 (a) Adrenaline and the sympathomedullary (SAM) pathway: this is the body's immediate, acute response to a stressor. When a person perceives a situation as threatening, the hypothalamus activates the sympathetic branch of the autonomic nervous system (ANS). Sympathetic nerves stimulate the adrenal medulla, at the centre of the adrenal glands above the kidneys, to release the hormones adrenaline and noradrenaline into the bloodstream.

Adrenaline produces the 'fight or flight' response. Heart rate and blood pressure rise, breathing becomes faster and the airways dilate, so more oxygen reaches the muscles. Glucose and fats are released from stores for energy, blood is diverted away from the digestive system and skin to the muscles and brain, pupils dilate and sweating increases. These changes prepare the body to fight or to run away from the threat. When the stressor has passed, the parasympathetic branch of the ANS takes over and has a dampening effect ('rest and digest'), returning heart rate and blood pressure to normal. If the stressor is long-term, the SAM pathway is repeatedly activated, and the HPA axis also releases cortisol, which can lead to high blood pressure, damage to blood vessels and suppression of the immune system.

Evolutionary adaptation: this explanation suggests the stress response evolved because it helped our ancestors survive in the environment of evolutionary adaptedness (EEA). Ancestors who responded quickly to acute physical threats, such as a predator or an attack from a rival group, with fight or flight were more likely to survive and reproduce, so the genes for a strong stress response were passed on by natural selection. These threats were usually short-lived and resolved by physical action, which used up the energy mobilised by the response.

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In modern society, however, most stressors are psychological and long-term, such as work pressure, exams, money worries or relationship problems. They cannot be dealt with by fighting or fleeing, yet the same ancient response is triggered, often repeatedly. Because our biology has not changed as quickly as our environment (genome lag), this response is now maladaptive: the body's resources are depleted and the chronic arousal has deleterious effects on health, such as heart disease and lowered immunity. Our stress response is therefore adapted to a past environment rather than to the present one.

6 (b) Stress inoculation training (SIT), developed by Meichenbaum (1985), is a form of CBT that aims to 'inoculate' a person against future stress by giving them coping skills before the stressor occurs. It has three phases: conceptualisation, where Mehdi would learn how his appraisal of his exams and driving test as threatening increases his stress; skills acquisition and rehearsal, where he would learn skills such as relaxation, controlled breathing, positive self-talk and time management; and application and follow-through, where he would practise these in increasingly stressful situations, such as timed mock exams or practice drives with his instructor, with booster sessions.

A major strength is the supporting evidence. Saunders et al. (1996) carried out a meta-analysis of 37 studies and concluded that SIT reduced performance anxiety and state anxiety and improved performance under stress. This is very relevant to Mehdi, as his examinations and driving test are both performance situations where anxiety could impair how well he does. Sheehy and Horan (2004) found that SIT reduced anxiety and stress in first-year law students, and that those who completed it achieved better results, suggesting it could help Mehdi with his examinations in particular.

SIT is also effective for more serious stress. Foa et al. (1991, 1999) found that SIT reduced the symptoms of post-traumatic stress disorder in victims of assault, with results similar to prolonged exposure therapy. This suggests SIT is a robust method that could cope with the range of stressors Mehdi faces.

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Another strength is that SIT tackles the cause of stress rather than just the symptoms. Mehdi is stressed by several sources, his exams, driving test and other personal issues, and SIT gives him transferable skills that he can apply to all of them and to future stressors such as job interviews. This gives it long-term effectiveness, unlike beta blockers, which only reduce physical symptoms such as a racing heart and sweating by blocking adrenaline. Neftel et al. (1982) found beta blockers reduced stage fright in musicians, but once Mehdi stopped taking them his stress would probably return, and side effects such as tiredness and dizziness could be dangerous during his driving test.

Ethically, SIT is also a good choice for Mehdi. It is non-invasive and has no physical side effects, and because he is an active participant he keeps control of his treatment, which may increase his self-efficacy and confidence.

However, SIT has weaknesses. It is time-consuming and requires a lot of motivation and commitment. Mehdi is already under pressure with revision and driving lessons, so he may find it hard to attend sessions and practise, and he could drop out. It may also be expensive if he has to pay a trained therapist. If his exams are very soon, he may not complete all three phases in time, so a quick-acting method such as beta blockers could be more practical in the short term.

It is also unclear which parts of SIT are effective. Moses and Hollandsworth (1985) found with dental phobics that education alone (the conceptualisation phase) was not enough to reduce anxiety, and that the application of skills was most important. So Mehdi would need to complete the whole programme, and simply understanding why he is stressed may not be enough. Foa et al. also found that, in the long term, SIT was no more effective than prolonged exposure. Moreover, SIT may be less suitable for stressors Mehdi cannot control: if his 'other personal issues' involve family problems or bereavement, coping skills may help but cannot remove the stressor.

In conclusion, SIT is a well-supported, ethical and long-lasting method that suits Mehdi well because his stress comes from predictable performance situations

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such as exams and a driving test. Its main drawbacks are the time and commitment required, so it would work best if Mehdi starts well before his exams, perhaps alongside short-term support.

8

Non-human animals have been used in psychology both as research subjects, in laboratory experiments and naturalistic observation, and as therapeutic devices. I agree with the statement: animal research has been useful, but it is also problematic, both scientifically, because findings may not generalise to humans, and ethically, because of the harm caused. The key question is whether the usefulness outweighs the problems.

Animals as a therapeutic device are clearly useful. Animal-assisted therapy uses animals such as dogs and horses to improve human wellbeing. Allen (2003) reviewed evidence that pet ownership and the presence of pets lower blood pressure and heart rate responses to stress, suggesting animals reduce stress and the risk of cardiovascular illness. Therapy dogs are used with children with autism, older people with dementia and hospital patients, where they may increase social interaction and reduce anxiety and loneliness. This involves little harm, as the animals are usually companions treated with care. However, it is also problematic: much of the evidence is correlational, so pet owners may already be healthier or less stressed, and therapy animals may become stressed by repeated handling in unfamiliar environments.

Animal research has also been useful in understanding behaviour. Ethological research such as Lorenz (1935), who found that goslings imprinted on the first moving object they saw, was largely naturalistic and had limited impact on the animals, yet it informed our understanding of critical periods and influenced Bowlby's theory of attachment. Skinner's research on operant conditioning in rats and pigeons led to behaviour modification techniques such as token economies used in prisons and psychiatric hospitals, and Pavlov's work with dogs on classical conditioning led to therapies such as systematic desensitisation and aversion therapy.

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Animal research has been particularly useful for psychopathological disorders. Randrup and Munkvad (1966) found that giving rats amphetamines, which increase dopamine, produced stereotyped behaviours similar to some symptoms of schizophrenia, and that antipsychotic drugs reduced these, supporting the dopamine hypothesis and the development of drug treatments. Seligman's research on learned helplessness in dogs provided a model of depression. Research can therefore identify possible causes of disorders and establish whether treatments reduce the severity of symptoms before they are tested on humans, reducing the impact of the disorder on people's lives.

There are also practical reasons why animals are useful. Their shorter lifespans and breeding cycles allow many generations to be studied, for example in genetic research, and greater control over their environment increases internal validity. Procedures that would be unethical with humans, such as lesioning parts of the brain or long-term deprivation, become possible. Gray (1991) argued that we have a special duty to our own species, so if research reduces human suffering it is morally justified.

Supporters also argue that the problems are reduced because such research is strictly controlled. In the UK the Animals (Scientific Procedures) Act 1986 requires that research is licensed by the Home Office and that the likely benefits outweigh the costs to the animals. The BPS guidelines for psychologists working with animals set out a clear structure, requiring researchers to follow the 3Rs (Russell and Burch, 1959): replacement with alternatives where possible, reduction of the number of animals used and refinement of procedures to minimise suffering, as well as choosing suitable species, housing and caging, and minimising pain and deprivation. This means needless experiments should be avoided.

However, the main scientific problem is generalisation. Animals are not humans: they differ in physiology, brain structure, cognition, language and culture, so findings may apply only to the species tested. Schizophrenia involves hallucinations, delusions and disordered speech, which cannot be observed in

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rats, so Randrup and Munkvad's model only captures some behaviours. Similarly, human attachment involves cognitive and cultural factors absent in geese. Interpreting animal behaviour in human terms also risks anthropomorphism. If findings do not generalise, their importance is reduced and the suffering the animal has endured is needless.

Some research has also been ethically questionable and detrimental to the animals. Harlow (1959) separated infant rhesus monkeys from their mothers and raised them with wire and cloth surrogates. Although this showed that contact comfort, not food, is the basis of attachment, the monkeys became socially and emotionally damaged and later abused or neglected their own offspring, a long-lasting impact. Brady's (1958) executive monkey study, in which monkeys received electric shocks and developed ulcers, was used to suggest that responsibility causes stress, but the monkeys were not randomly allocated to conditions, so the findings were flawed and the suffering produced little benefit.

There is also the pure issue of animal rights. Singer (1975) argued that discriminating against animals because of their species is speciesism, comparable to racism, since what matters is the capacity to suffer. Regan (1984) argued that animals have inherent value and that there is no circumstance in which an animal should be used in research, even if it improves human life. From this view, animal research is always problematic, however useful. In wider society, public attitudes have changed, with many people opposed to animal testing, and psychology has moved towards alternatives such as brain scanning, computer modelling and observing animals in their natural habitats.

A useful way to resolve this is Bateson's (1986) decision cube, which weighs the quality of the research, the degree of animal suffering and the certainty of benefit. Research should proceed only when quality is high, benefit is likely and suffering is minimal. Under this model, Lorenz's research would be acceptable, but Harlow's and Brady's would not, showing that usefulness and problems vary between studies.

In conclusion, I largely agree with the statement. Using animals has been useful,

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giving insights into learning, attachment and mental disorders and providing therapeutic benefits. However, it is problematic because findings may not generalise and some studies caused severe suffering for little benefit. Its use is justified only when the research is high quality, the benefits clearly outweigh the costs, no alternative exists and the 3Rs and BPS guidelines are followed.