GCE A LEVEL

A290U30-1

O20-A290U30-1

THURSDAY, 15 OCTOBER 2020 – MORNING

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 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) 'Methods of modifying addictive behaviours are often controversial yet always effective.'

With reference to this statement, evaluate aversion therapy as a method of modifying addictive behaviours. [15]

b) Describe **one** individual differences explanation of addictive behaviours. [10]

0 2 **Autistic spectrum behaviours**

a) Describe social psychological explanations of autistic spectrum behaviours. [10]

b) 'Picture Exchange Communication System (PECS) is widely used but has many limitations.'

With reference to this statement, evaluate Picture Exchange Communication System (PECS) as a method of modifying autistic spectrum behaviours. [15]

0 3 **Bullying behaviours**

a) Patricia is the leader of a popular gang of girls in her school. Her head of year has received numerous concerns from parents of other children in the school about her negative behaviour.

Describe the characteristics of Patricia's bullying behaviours. [15]

b) Briefly evaluate the effectiveness of the Olweus Bullying Prevention Programme as a method of modifying bullying behaviours. [5]

c) Briefly evaluate **one** individual differences explanation of bullying behaviours. [5]

0 4 **Criminal behaviours**

a) 'Individual differences explanations are perhaps the least useful in explaining criminal behaviours.'

With reference to the statement, evaluate individual differences explanations of criminal behaviours. [15]

b) Briefly describe **one** biological explanation of criminal behaviours. [5]

c) Briefly describe **one** method of modification for criminal behaviours. [5]

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0 5 **Schizophrenia**

a) Describe **one** biological explanation and **one** individual differences explanation of schizophrenia. [5 + 5]

b) 'Social psychological explanations provide the most convincing account of schizophrenia.'

Evaluate social psychological explanations of schizophrenia with reference to the statement. [15]

0 6 **Stress**

a) Describe **one** individual differences explanation and **one** social psychological explanation of stress. [5 + 5]

b) 'No one method of modifying stress provides a clear solution to resolving the negative impact stress has on individuals.'

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

SECTION B: Controversies

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

Either,

0 7 **Sexism**

'Women in psychology have gone from a position of invisibility to one of influence.'

Discuss the extent to which you agree with this statement. [25]

Or,

0 8 **Non-human animals**

'Using non-human animals in psychology is unavoidable.'

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

04(a)

Individual differences explanations claim that offending results from characteristics within the person, such as personality or cognition. The statement suggests these are the least useful explanations, but the evidence shows they have real strengths, especially when combined with biological and social factors.

Eysenck's (1964) criminal personality theory proposes that offenders score highly on extraversion, neuroticism and psychoticism. Extraverts have an under-aroused nervous system, so they seek stimulation and condition poorly, failing to learn from punishment; neurotics are unstable and overreact to threat; psychoticism is linked to coldness and aggression. In support, Eysenck and Eysenck (1977) found that prisoners scored higher on all three dimensions than controls, and Dunlop et al. (2012) also found links between these personality dimensions and offending. This suggests the explanation is useful, as it identifies who may be most at risk and why some people fail to be socialised. However, Farrington et al.'s (1982) review found offenders were consistently high only on psychoticism, and results for extraversion were mixed. Mischel (1968) argued that behaviour is not consistent across situations, so a fixed personality cannot fully explain why a person offends in some situations but not others. Personality questionnaires such as the EPQ also have limited reliability and are affected by social desirability bias and mood, and prisoners may answer differently because of the prison environment itself, which supports the statement.

Kohlberg's theory of moral reasoning, an intelligence (cognitive) factor, suggests that offenders are stuck at the pre-conventional level, where decisions are based on avoiding punishment and gaining rewards, so they offend when they think they will not be caught. Palmer and Hollin (1998) found that young offenders showed less mature moral reasoning than non-offenders on the Sociomoral

Question number

Reflection Measure, and Chen and Howitt (2007) found similar results with young offenders in Taiwan, suggesting cross-cultural usefulness. Research has also linked lower IQ with offending (Gudjonsson et al., 2007). However, Gilligan (1982) argued that Kohlberg's theory is androcentric because it was based on interviews with males, so it may not explain female offending. Krebs and Denton (2005) found that moral reasoning about hypothetical dilemmas does not match real-life moral decisions, which reduces validity. Also, many people with immature moral reasoning never commit crime.

Psychopathy is perhaps the most useful individual differences explanation. Psychopaths are superficially charming, impulsive, manipulative and lack empathy, guilt and remorse, and are assessed with Hare's Psychopathy Checklist Revised (PCL-R). Hemphill et al.'s (1998) meta-analysis found that psychopaths were about three times more likely to reoffend, and four times more likely to reoffend violently, than non-psychopaths, so the PCL-R is used in risk assessment and parole decisions, a clear practical application which goes against the statement. However, the reliability of personality testing has been questioned (Borelli, 2017) and most offenders are not psychopaths.

Compared with other explanations, individual differences theories ignore social factors. Sutherland's (1939) differential association theory argues that crime is learned from attitudes and techniques in the groups a person associates with, and Pollak (1950) argued gender socialisation explains differences in female offending. Biological research also offers strong evidence: Mednick et al.'s (1984) study of over 14,000 adoptees found that 20% of sons with a convicted biological parent had convictions, compared with 13.5% when neither parent had, and Raine et al. (1997) found reduced prefrontal activity and abnormal amygdala activity in murderers. However, Eysenck himself argued that personality is largely inherited, so individual and biological explanations are linked rather than competing.

In conclusion, the statement is only partly true. Individual differences explanations are limited by their methodology and by ignoring social influences,

Question number

but they are not the least useful: psychopathy in particular has strong predictive value. The most complete account is a diathesis-stress approach in which personality and cognition interact with biology and social learning.

04(b)

The role of the amygdala explanation proposes that criminal, especially violent, behaviour is linked to dysfunction of the amygdala, a small almond-shaped structure in the limbic system, one in each hemisphere, made up of about 13 nuclei. It has strong neural connections with the hypothalamus, hippocampus and prefrontal cortex, and is involved in processing emotions such as fear, recognising distress in other people's faces and emotional learning, such as learning to avoid actions that are punished.

If the amygdala is underactive or reduced in size, the person experiences less fear and less empathy for victims' distress, so punishment does not deter them and they do not learn from it. An overactive amygdala that is poorly regulated by the prefrontal cortex may instead produce impulsive, reactive aggression.

Glenn (2009) argued that amygdala dysfunction is a key aspect in understanding psychopathy, and Raine et al. (1997) found abnormal amygdala activity in PET scans of murderers. This dysfunction may be inherited or caused by brain injury.

04(c)

Anger management was developed by Novaco (1975), who argued that anger has cognitive, physiological (biological) and behavioural elements, so treatment targets all three. It is a form of cognitive behavioural therapy based on stress inoculation, and in the UK it is used in prisons through programmes such as CALM (Controlling Anger and Learning to Manage it). It has three stages.

First, cognitive preparation: the offender reflects on past episodes of anger, for

Question number

example in an anger diary, to identify triggers and the irrational thoughts that make them angry, such as seeing a look as a deliberate insult. Second, skills acquisition: they learn techniques to deal with each element, including relaxation and breathing to reduce physiological arousal, cognitive restructuring and positive self-talk to change thought patterns, and assertiveness training so they can express their needs without aggression. Third, application practice: they practise these skills in role plays of increasingly provoking situations with the therapist, before using them in real life.

05(a) Biological: the dopamine hypothesis states that schizophrenia is caused by excess dopamine activity. In its original form it proposed that too much dopamine, or too many or oversensitive D_2 receptors, causes neurons to fire too often, producing positive symptoms such as hallucinations and delusions. Evidence came from amphetamines, which increase dopamine and can produce psychotic symptoms, and from antipsychotic drugs, which block dopamine receptors and reduce symptoms. However, this early view could not explain negative symptoms, so the revised hypothesis looks at different dopamine pathways: excess dopamine in the mesolimbic pathway (hyperdopaminergia) causes positive symptoms, while too little dopamine in the mesocortical pathway to the prefrontal cortex (hypodopaminergia) causes negative symptoms such as avolition and poverty of speech, as well as cognitive problems.

Individual differences: the cognitive explanation of thought disorder was developed to correct the psychodynamic approach's lack of scientific rigour. It argues that the symptoms of schizophrenia result from malfunctions in information processing. Frith (1992) proposed a deficit in metarepresentation, the ability to reflect on our own thoughts and others' intentions (theory of mind). If a person cannot recognise their thoughts as self-generated, they experience them as voices or thought insertion, and failing to understand others' intentions leads to paranoid delusions. A deficit in central control, the ability to suppress automatic

Question number

responses, causes derailment of thought and speech. Hemsley (1993) suggested a breakdown in the pre-conscious filter that links stored knowledge with current sensory input, so the person is overwhelmed by irrelevant stimuli and attaches special significance to them.

05(b) Social psychological explanations argue that schizophrenia is caused, or its course worsened, by the person's social environment: expressed emotion, dysfunctional families and cultural norms. The statement claims these are the most convincing account, but evidence suggests they are better seen as triggers acting on a biological vulnerability.

Expressed emotion (EE) refers to a family climate of criticism, hostility and emotional over-involvement. Brown et al. found that patients returning to high EE families were more likely to relapse, and Vaughn and Leff (1976) confirmed this, especially when the patient had a lot of face-to-face contact with the family and was not taking medication. Kavanagh's (1992) review of 26 studies found an average relapse rate of 48% in high EE families compared with 21% in low EE families. This supports the statement and has a practical application: family interventions that reduce EE lower relapse rates. However, EE mainly explains relapse rather than the original onset, and findings are not consistent across all studies (McCreadie et al., 1998). The direction of causation is also unclear, as high EE may be a reaction to the stress of caring for someone with symptoms.

Dysfunctional families: Bateson et al.'s (1956) double bind theory proposes that a child repeatedly receives contradictory messages from a parent, such as being told they are loved while being rejected through body language, and cannot respond correctly, leading to confusion and disorganised thinking. Berger (1965) found that schizophrenic patients recalled more double bind statements from their mothers than controls. However, this relies on retrospective recall, which may be distorted by the illness. Liem (1974) found that parents of

Question number

schizophrenic sons communicated no differently from other parents, and that abnormal communication was a response to the son's symptoms, not the cause. This weakens the statement and raises ethical concerns about blaming families.

Cultural norms: diagnosis and experience of schizophrenia differ across cultures. Hearing voices may be accepted as normal in some cultures, and culture-bound symptoms exist (Kaplan et al., 1994), while Lopez et al. (2004) found family warmth was linked with better outcomes in Mexican-American families. In the UK, African-Caribbean people are diagnosed far more often than white people, which may reflect social stress, discrimination or diagnostic bias. This shows social factors shape diagnosis and outcome. However, schizophrenia occurs at a similar rate of about 1% in most cultures, suggesting a universal biological basis.

Biological explanations are arguably more convincing. Gottesman (1991) found concordance of about 48% for MZ twins compared with 17% for DZ twins, enlarged ventricles have been found in CAT and MRI scans, and antipsychotics that block dopamine reduce positive symptoms. Cognitive research also shows processing deficits (Barch et al., 1999) that social theories ignore. However, MZ concordance is not 100%, so environment must play a role. Tienari et al.'s Finnish adoption study found that adoptees with a genetic risk were most likely to develop schizophrenia when raised in dysfunctional adoptive families, supporting the diathesis-stress model.

In conclusion, social psychological explanations are useful for explaining relapse and the influence of culture, and have led to effective family interventions, but they are not the most convincing account on their own, as they cannot explain why only some people exposed to these environments develop schizophrenia. The most convincing account is the diathesis-stress model, where social stressors trigger an inherited biological vulnerability.

06(a) Individual differences: Friedman and Rosenman proposed that Type A personality

Question number

makes people more vulnerable to stress and stress-related illness. Type A individuals are competitive and achievement-striving, impatient with a sense of time urgency, and hostile. Because they perceive many situations as stressful, the sympathomedullary pathway is repeatedly activated, releasing adrenaline and noradrenaline, raising heart rate and blood pressure, and cortisol is released via the HPA axis. Over time this damages arteries and increases the risk of coronary heart disease (CHD). Type B individuals are relaxed and patient, with a lower physiological response and fewer health problems. In the Western Collaborative Group Study, Friedman and Rosenman followed 3,154 healthy men for eight and a half years; 257 developed CHD, 70% of whom were Type A, and Type A men were about twice as likely to develop CHD as Type B men.

Social psychological: the life events explanation, from Holmes and Rahe, proposes that major life changes, positive or negative, require psychological adjustment, and that the more change a person experiences, the greater their stress and risk of illness. Holmes and Rahe (1967) developed the Social Readjustment Rating Scale (SRRS) of 43 life events, each given a number of life change units (LCUs): death of a spouse scores 100, divorce 73 and marriage 50, the reference point. A score of over 300 LCUs in a year is linked to a high risk of stress-related illness. Rahe et al. (1970) gave a version of the SRRS to about 2,500 US Navy sailors and found a small but significant positive correlation (+0.118) between LCUs in the previous six months and illness during their tour of duty.

06(b)

Methods of modifying stress include biological methods such as beta blockers and psychological methods such as stress inoculation training (SIT). The statement suggests that none of them provides a clear solution to the negative impact of stress, and evaluating each method shows that this is largely true, although a combination may come close.

Beta blockers, such as propranolol, block beta-adrenergic receptors in the heart

Question number

and blood vessels so adrenaline and noradrenaline cannot bind, slowing heart rate and lowering blood pressure. Neftel et al. (1982) found that musicians with stage fright given a beta blocker reported less stage fright and performed better than those given a placebo, and Schwabe et al. (2011) found that propranolol prevented the stress-induced shift from goal-directed to habit behaviour. They are quick, cheap and need little effort, which suggests they can resolve the immediate impact of stress. However, they only relieve physical symptoms in the short term and are not a cure, as they do not address the cause of stress, especially when it is psychological or emotional, so symptoms return when they are stopped. They also have side effects such as tiredness and dizziness, especially if stopped suddenly, which supports the statement.

Stress inoculation training, developed by Meichenbaum, is a form of CBT with three phases: conceptualisation, where the client learns how their appraisals create stress; skills acquisition and rehearsal, such as relaxation and positive self-talk; and application and follow-through in increasingly stressful situations. It may give individuals a long-term coping strategy. Saunders et al.'s (1996) meta-analysis of 37 studies found SIT reduced performance and state anxiety, and Sheehy and Horan (2004) found it reduced anxiety and stress in first-year law students, showing benefits in many contexts. Foa et al. (1991, 1999) found it effective at reducing PTSD symptoms, similar to prolonged exposure, and Meichenbaum (2007) argued it is especially useful given the wide range of stressors in modern life. This challenges the statement, as SIT tackles the cause. However, Foa et al. found little difference between SIT and prolonged exposure over long periods, so it is not uniquely effective. SIT is also time-consuming and needs high motivation, although Meichenbaum showed it can be effective in short bursts.

Other methods also have limits. Benzodiazepines reduce anxiety quickly but cause dependency, and social support depends on the quality of a person's relationships. Individual differences matter too: a Type A person or someone with an external locus of control may respond differently from others.

Question number

In conclusion, the statement is largely true: beta blockers give fast but temporary relief, while SIT offers long-term coping but needs time and commitment. The best solution is probably a combination, using a biological method to control immediate symptoms while a psychological method addresses the cause.

08

The statement claims that psychology could not progress without using non-human animals. I will argue that animal use is unavoidable for some types of research and has clear benefits, but that much of it can now be avoided, so the statement is only partly true.

Some areas of psychology study animals for their own sake. Comparative psychology and ethology aim to understand animal behaviour, which is impossible without animals. Lorenz (1935) found that goslings imprinted on the first moving object they saw during a critical period. This naturalistic research had limited impact on the animals, yet it influenced Bowlby's theory of attachment, so this type of animal use is unavoidable and causes little harm.

Animal research has also led to benefits for human psychopathology that could not have been achieved ethically with humans. Randrup and Munkvad (1966) found that giving rats amphetamines, which increase dopamine, produced stereotyped behaviours similar to some symptoms of schizophrenia, and that antipsychotics reduced these behaviours, supporting the dopamine hypothesis and the development of drug treatments. Seligman's learned helplessness research with dogs provided a model of depression. Skinner's research on rats and pigeons led to token economies in hospitals and prisons. Research can identify causes and test whether treatments reduce symptoms, so where procedures such as brain lesions or drug trials would be unethical in humans, animals may be unavoidable. Animals also have short lifespans, so several generations can be studied.

Animals are also used to improve human life as therapeutic devices. Allen

Question number

(2003) reviewed evidence that pet ownership and the presence of pets reduced blood pressure and heart rate responses to stress. Animal-assisted therapy with dogs and horses is used with children with autism and older people with dementia. Here the animal itself is the treatment, so its use is unavoidable, although therapy animals' welfare must still be protected.

Supporters argue that strict regulation means only unavoidable research takes place. The Animals (Scientific Procedures) Act 1986 requires Home Office licences and that the benefits outweigh the costs to the animals. The BPS Guidelines for Psychologists Working with Animals require researchers to consider the 3Rs (Russell and Burch, 1959), choice of species, numbers, caging and housing, and to minimise pain and deprivation, so needless experiments are avoided. Gray (1991) argued that we have a special duty to our own species to reduce human suffering.

However, the 3Rs themselves show that animal use is often avoidable, since replacement means using alternatives wherever possible. Brain scanning (fMRI, PET), computer modelling, case studies of brain-damaged patients and observations of animals in their natural habitats now answer many questions once studied through experiments, which is why the number of animals used in psychology has fallen sharply.

There is also a problem of generalisation. Humans differ from animals in physiology, cognition, language and culture, so findings may only apply to the species tested. Schizophrenia involves hallucinations, delusions and disorganised speech, which cannot be observed in rats, so Randrup and Munkvad's model only captures some behaviours. Interpreting animal behaviour in human terms also risks anthropomorphism. If findings do not generalise, the animals' suffering is needless and the research was avoidable.

Some research has been ethically questionable and seriously harmful. Harlow (1959) raised infant rhesus monkeys with wire and cloth surrogate mothers; they became socially and emotionally damaged and later neglected or abused their own offspring. Brady's (1958) executive monkey study, in which monkeys

Question number

received electric shocks and developed ulcers, was flawed because the monkeys were not randomly allocated. It is hard to argue that this suffering was unavoidable, especially as human attachment and stress can be studied in ethical ways.

There are also animal rights arguments. Singer (1975) argued that discriminating against animals because of their species is speciesism, comparable to racism, as what matters is the capacity to suffer. Regan (1984) argued that animals have inherent value and should never be used, even to benefit humans, so from this view all animal research should be avoided. In wider society, public opposition to animal testing has increased, which has pushed psychology towards alternatives. Bateson's (1986) decision cube offers a way to judge when animal research is acceptable, considering the quality of the research, the degree of suffering and the certainty of benefit. Under this model, Lorenz's research would be justified, whereas Harlow's and Brady's would not.

In conclusion, using non-human animals is unavoidable for comparative and ethological research, for animal-assisted therapy and for some biological research into disorders that cannot be done ethically on humans. However, much research is avoidable through scanning and other alternatives, findings often do not generalise and some studies have caused unjustifiable suffering. Therefore the statement is true only to a limited extent.