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A-Level Psychology

Model Answers · 16-Mark Exemplars

Edexcel Psychology (9PS0) | 16-Mark Exemplars | 18 entries

Full worked 16-mark model essays - see exactly what a top-band answer looks like, paragraph by paragraph.

Evaluate the multi-store model of memory. You must refer to research evidence in your answer.

Introduction	The multi-store model, proposed by Atkinson and Shiffrin in 1968, is a structural account that describes memory as a flow of information through three separate, fixed stores: the sensory register, short-term memory and long-term memory. Information is held in the sensory register by modality, passes into short-term memory only if it is attended to, and is then transferred to long-term memory through maintenance rehearsal. The stores are said to differ in capacity, duration and coding. This essay will argue that, while the model is supported by clear experimental evidence for separate stores, its insistence on a single, unitary short-term store and on rehearsal as the only route to long-term memory is its central weakness, and that on balance it is now best regarded as an oversimplified but historically valuable framework.
AO1	According to the model, the sensory register receives raw input from every sense, codes it in a modality-specific way, such as iconically for vision or echoically for sound, and holds it for a fraction of a second with a very large capacity. If attention is paid, the trace enters short-term memory, which the model claims codes acoustically, holds roughly 7 plus or minus 2 items, and lasts around 18−30 seconds unless rehearsed. Maintenance rehearsal both keeps material in short-term memory and, crucially, transfers it to long-term memory, which the model treats as a single store coding semantically, with potentially unlimited capacity and lifelong duration. Retrieval involves bringing material from long-term memory back into short-term memory. The defining feature is therefore that the three stores are unitary and linear.
Evaluation 1	A major strength is that controlled research supports the existence of two functionally separate stores. Glanzer and Cunitz in 1966 presented participants with word lists and found a clear serial position curve: the first words were recalled well, the primacy effect, because they had been rehearsed into long-term memory, and the last words were recalled well, the recency effect, because they were still in short-term memory. When recall was delayed by a 30-second counting task, the recency effect disappeared while primacy survived. This matters because two stores being knocked out by different manipulations is exactly what the model predicts, so the data provide strong converging evidence that short-term and long-term memory are dissociable systems rather than one continuous store, which is the core claim the model is built on.
Evaluation 2	However, a serious limitation is that the model treats short-term memory as a single passive store, which the case of patient KF directly contradicts. Shallice and Warrington in 1970 reported that KF, after brain damage, had a badly impaired digit span for verbal material presented out loud yet a near-normal short-term memory for visual and meaningful information. If short-term memory were the one unitary store the model describes, damage should impair it across the board. The fact that one type of short-term information could be selectively destroyed while another was spared shows that short-term memory must have separable components, as later captured by the working memory model. This undermines the model because its central architecture is too simple to explain the neuropsychological evidence, suggesting it describes structure but misrepresents how short-term memory is actually organised.

Evaluation 3	A further weakness is that the model overstates the role of maintenance rehearsal as the route into long-term memory. Craik and Lockhart in 1972 argued instead that it is the depth of processing, not the amount of repetition, that determines durable storage, and supporting studies show that semantically processed words, judged for meaning, are remembered far better than words rehearsed by rote or processed only for their physical appearance. This is damaging because everyday experience agrees: a single emotionally meaningful event, such as a flashbulb memory of bad news, can enter long-term memory permanently with no rehearsal at all. So what this implies is that the model has identified a route into long-term memory but mistaken it for the only route, leaving it unable to account for how meaning and elaboration create lasting memories, which is a substantial gap in its explanatory power.
Conclusion	Weighing these points, the model deserves credit for being testable and for correctly establishing that short-term and long-term memory are genuinely separate stores, a finding that has survived decades of scrutiny and that more recent models still build upon. Yet the evidence from KF and from depth-of-processing research shows that its two key assumptions, a unitary short-term store and rehearsal as the sole transfer mechanism, are both wrong. Because these flaws strike at the heart of the model rather than its edges, the most defensible judgement is that the multi-store model is a valuable but outdated foundation: it should be retained as a starting point for teaching the architecture of memory, but replaced by the working memory model and levels-of-processing accounts when explaining how short-term memory operates and how long-term memories are actually formed.

Discuss social psychological explanations of obedience. You must refer to research in your answer.

Introduction	Obedience is following the direct orders of a figure perceived to hold legitimate authority. Social psychological explanations locate the cause of obedience not in the personality of the individual but in the situation: features of the social setting that shift the person into an obedient frame of mind. The two most influential ideas, both arising from Milgram's work, are agency theory, which proposes that people enter an agentic state in which they no longer feel personally responsible, and the role of legitimate authority and proximity. This essay will discuss these explanations, argue that they are strongly supported by experimental variations, but conclude that they are incomplete because they neglect the role of personal agreement with the cause, and so explain how obedience happens better than they explain when it will not.
AO1	Agency theory states that people operate in one of two states. In the autonomous state they act on their own values and feel responsible for their behaviour. When confronted by a legitimate authority, they undergo an agentic shift into the agentic state, in which they see themselves merely as an instrument carrying out another person's wishes, so responsibility is displaced upwards to the authority figure. This produces moral strain, the discomfort of acting against one's conscience, which is reduced by denial and by focusing on the procedure rather than the victim. Alongside this, the explanation stresses legitimacy of authority, the perceived right of a person to give orders, often signalled by uniforms or institutional settings, and proximity, the physical and psychological closeness of the authority figure and of the victim, both of which raise or lower the likelihood of obedience.
Evaluation 1	Strong support for the situational account comes from Milgram's own variations in 1963 and after. In the baseline study 65 percent of participants delivered the full 450 volts to a learner, but when the experimenter gave orders by telephone rather than in person, obedience fell to around 21 percent, and when the study was moved from prestigious Yale University to a run-down office, it dropped to roughly 48 percent. This matters because the participants were the same kind of people in each condition, so the dramatic swings in obedience cannot be explained by personality and must be caused by the situation, exactly as the social explanation claims. The fact that reducing the proximity or legitimacy of the authority reliably reduced obedience gives the theory real predictive power, which is a hallmark of a strong explanation.

Evaluation 2	The explanation also gains credibility because it generalises beyond the laboratory to real atrocities. Hofling and colleagues in 1966 found that 21 of 22 nurses were prepared to administer a clearly excessive and unauthorised dose of a drug simply because a doctor instructed them to over the phone, despite breaking hospital rules. Likewise, agency theory has been used to explain how ordinary soldiers in events such as My Lai followed orders to harm civilians. This is important because a key criticism of laboratory obedience research is that it lacks ecological validity, yet here the same situational mechanism, displacement of responsibility onto a legitimate authority, appears in genuine high-stakes settings. So what this shows is that the explanation is not an artefact of an artificial task but captures a real social process, strengthening its claim to be a valid account of destructive obedience.
Evaluation 3	However, a significant weakness is that the explanation underplays how much obedience depends on the participant identifying with the aims of the authority. Reicher and Haslam's social identity approach argues that Milgram's participants did not blindly enter an agentic state but actively chose to follow the experimenter when they identified with the scientific goal of the study, and resisted when they identified instead with the suffering learner. Supporting this, Milgram's own prods worked in a revealing order: the engaged appeal that the experiment requires you to continue produced obedience, whereas the bare command that you have no other choice almost always produced defiance. This is damaging because if people were truly in a passive agentic state, a direct order should be the most effective prod, not the least. So this implies the situational explanation describes the conditions for obedience but misses the active, identity-based decision the person is still making.
Conclusion	On balance, social psychological explanations of obedience are among the best supported in psychology: the systematic variations in Milgram's research, backed by field evidence from Hofling and by real historical cases, show convincingly that obedience is driven by situational factors such as legitimacy and proximity rather than by a faulty personality. However, the social identity critique exposes a real limitation, namely that the agentic state is too passive a concept and ignores the participant's continued engagement with the cause. The most defensible judgement is therefore that the situational explanation is necessary but not sufficient: it correctly identifies the social conditions that make obedience likely, but a complete account must combine it with social identity theory to explain why, under identical conditions, some people obey and others resist.

Evaluate biological explanations of aggression. You must refer to research evidence in your answer.

Introduction	Biological explanations regard aggression as rooted in the body rather than in upbringing or learning. Three strands dominate: the influence of hormones, especially testosterone; the role of neurotransmitters, especially low serotonin; and the contribution of genes, including the so-called warrior gene that affects the enzyme MAOA. Together these propose that individual differences in aggression reflect differences in physiology. This essay will evaluate these explanations, arguing that although there is consistent evidence linking each biological factor to aggression, the relationships are correlational and modest, and that the explanation is most convincing when it is integrated with environmental triggers rather than treated as a complete cause in itself.
AO1	The hormonal explanation argues that testosterone increases aggression by acting on brain regions such as the amygdala that process threat, which is offered as one reason males are typically more physically aggressive than females. The neurotransmitter explanation proposes that serotonin normally inhibits the firing of impulses in the frontal cortex, so low serotonin removes this brake and leaves the person more impulsive and reactively aggressive. The genetic explanation points to heritable factors, in particular a variant of the gene controlling MAOA, an enzyme that breaks down neurotransmitters after use; a low-activity version is said to leave neurotransmitter levels poorly regulated and to predispose individuals towards aggression. Crucially, biological explanations treat these factors as internal causes that operate largely independently of the social environment.

Evaluation 1	A clear strength is the supporting evidence for the role of serotonin and MAOA. Brunner and colleagues in 1993 studied a Dutch family in which many of the men showed a history of impulsive violence, including arson and assault, and found that the affected men shared a defective MAOA gene that disrupted serotonin metabolism. This is valuable because it links a specific, identifiable genetic abnormality to a specific pattern of aggressive behaviour, rather than relying on a vague claim that aggression runs in families. The convergence of a faulty gene, disturbed neurotransmitter function and observable violence in the same individuals provides strong biological support, suggesting that at least some aggression has a genuine physiological basis that cannot be explained by learning alone.
Evaluation 2	However, a major limitation is that the evidence is largely correlational, so cause and effect cannot safely be inferred, particularly for testosterone. While studies report associations between high testosterone and aggressive behaviour, experimental and longitudinal work shows the relationship is two-way: aggressive or competitive encounters can themselves raise testosterone, as seen when testosterone rises in the winners of sporting contests. This matters because it means high testosterone may often be a consequence of aggressive situations rather than their cause. The same problem affects serotonin research, where it is unclear whether low serotonin produces aggression or aggressive lifestyles lower serotonin. So what this implies is that the biological explanation frequently mistakes a correlate for a cause, weakening its claim to explain why aggression occurs.
Evaluation 3	A further weakness is that purely biological accounts are reductionist and cannot explain why the same biology produces aggression in some environments but not others. Caspi and colleagues in 2002 found that the low-activity MAOA gene predicted antisocial and aggressive behaviour only in men who had also suffered maltreatment in childhood; those with the same genotype but a stable upbringing were not unusually aggressive. This is damaging to a strictly biological view because it shows the gene is not deterministic but acts as a diathesis that requires an environmental stressor to be expressed. So this implies that biology sets a predisposition while experience pulls the trigger, and that any explanation treating genes or hormones as a sufficient cause on their own is incomplete and overlooks the demonstrable role of upbringing.
Conclusion	Taking these arguments together, the biological explanation of aggression is well supported in the sense that genes, the MAOA enzyme, serotonin and testosterone are all reliably associated with aggressive behaviour, and the Brunner findings show these links can be highly specific. However, the correlational nature of much of the evidence and, most tellingly, the gene–environment interaction shown by Caspi mean that biology alone cannot determine whether aggression actually appears. The most defensible judgement is therefore a diathesis–stress position: biological factors create a vulnerability to aggression, but environmental triggers such as maltreatment are needed to convert that vulnerability into behaviour. Biological explanations are thus an essential part of the picture but are most accurate when combined with social and developmental factors rather than standing alone.