SimplyPsychology

A-LEVEL PSYCHOLOGY REVISION NOTES

Stress

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.3.7 · A-level Paper 3

Compiled by **Simply Psychology** · simplypsychology.org/stress.html

Contents

2025 spec change: the **named drugs** (benzodiazepines and beta blockers) have been **removed** from the specification. Generic "drug therapy" as a way of managing stress remains, so the named drugs may still be used as examples but a 2026 question will not ask for them specifically.

AQA 2025 SPECIFICATION — STRESS (4.3.7)

- **Physiology of stress**: general adaptation syndrome; the hypothalamic-pituitary-adrenal (HPA) system; the sympathomedullary (SAM) pathway; the role of cortisol.
- **Stress and illness**: immunosuppression and cardiovascular disorders.
- **Sources of stress**: life changes and daily hassles; workplace stress (workload and control).
- **Measuring stress**: self-report scales (SRRS; Hassles and Uplifts Scale) and physiological measures (skin conductance response).
- **Individual differences**: personality types A, B and C; hardiness (commitment, challenge, control).
- **Managing and coping**: drug therapy, stress inoculation therapy, biofeedback; gender differences; social support (instrumental, emotional, esteem).

1 The Physiology of Stress

General Adaptation Syndrome (Selye 1936)

Selye proposed that the body responds to *any* prolonged stressor in the same three-stage pattern:

- **Alarm** — a stressor is perceived; the sympathetic nervous system triggers fight-or-flight arousal.
- **Resistance** — the body adapts and appears to cope, but is using resources to do so.
- **Exhaustion** — after prolonged stress, resources are depleted; stress-related illnesses (e.g. high blood pressure) appear.

The Sympathomedullary Pathway (SAM) — Acute Stress

For sudden, short-term stressors, the **hypothalamus** activates the **sympathetic** branch of the autonomic nervous system, which stimulates the **adrenal medulla** to release **adrenaline** and noradrenaline. This produces **fight-or-flight** arousal (raised heart rate, blood pressure and breathing). When the threat passes, the **parasympathetic** branch restores the body to rest.

The Hypothalamic-Pituitary-Adrenal (HPA) System — Chronic Stress

For ongoing stressors, the **hypothalamus** releases **CRH** → the **pituitary** releases **ACTH** → the **adrenal cortex** releases **cortisol**. Cortisol mobilises energy and helps the body cope, but **prolonged high cortisol suppresses the immune system**. A negative-feedback loop normally returns cortisol to baseline.

Evaluation

A strength of the physiological approach is the wealth of supporting research evidence. The roles of the SAM and HPA systems are supported by reliable, objective measures such as cortisol assays and heart-rate recordings, and the same response is observed across studies and species. Because this evidence is gathered using standardised physiological methods rather than subjective report, it gives the account high scientific credibility and internal validity. This strengthens the claim that stress has an identifiable, measurable biological basis.

However, a key limitation is that the physiological model is largely based on research with non-human animals. Selye developed the GAS by exposing rats to stressors, and much SAM/HPA research is animal-based. Whilst this allows tight experimental control, humans differ in that our stress response is heavily shaped by *cognitive appraisal* — how we interpret a situation. This means a purely physiological account is **reductionist**, ignoring the psychological dimension of stress, and we should be cautious about extrapolating animal findings directly to people.

A further limitation is that the model may be androcentric (a beta bias). Early stress research focused on a "fight-or-flight" response derived largely from male samples. Taylor et al. (2000) argued that females may instead show a **"tend-and-befriend"** response, governed partly by oxytocin, that protects offspring and builds

social networks. This suggests the classic physiological account over-generalises a male pattern to everyone, limiting its validity as a universal model of stress.

2 The Role of Stress in Illness

Immunosuppression

Chronic stress raises **cortisol**, which suppresses the immune system — reducing the number and activity of white blood cells (lymphocytes and natural-killer cells) and leaving the body more vulnerable to infection.

- **Kiecolt-Glaser et al. (1984)** — medical students showed reduced natural-killer-cell activity during exam periods compared with a month before, indicating exam stress weakened immune function.
- **Kiecolt-Glaser et al. (1995)** — carers of relatives with dementia healed from a small wound far more slowly than matched controls, showing chronic stress slows healing.

Cardiovascular Disorders

Chronic stress repeatedly raises heart rate and blood pressure and elevates cortisol, which over time damages blood vessels and contributes to **hypertension** and **coronary heart disease (CHD)**. Stress also encourages unhealthy behaviours (smoking, poor diet) that further raise cardiovascular risk.

Evaluation

A strength of the stress–illness link is the strong supporting evidence from controlled studies. Kiecolt-Glaser's research used objective biological measures (immune-cell counts, wound-healing rates) and real-life stressors, with control groups for comparison. Because the immune effects were measured directly rather than self-reported, the findings are difficult to explain away, supporting the conclusion that stress genuinely impairs immune function. This gives the explanation real internal validity.

However, a limitation is that much of the evidence is correlational. Many stress–illness studies measure naturally occurring stress and later illness, so they cannot establish that stress *causes* illness. Third variables — such as age, diet, alcohol use or sleep — may be responsible for both the stress and the poor health. This means the direction and cause of the relationship cannot be firmly established from correlational data alone, weakening the causal claim.

A further limitation is that the stress–illness relationship is mediated by behaviour and individual differences. People under stress often smoke and drink more and sleep less, so illness may stem from these behaviours rather than from immunosuppression directly. Moreover, not everyone exposed to stress becomes ill — personality, hardiness and social support (Sections 5–6) moderate the effect. This shows a simple "stress causes illness" account is incomplete and that an **interactionist** view is needed.

On the other hand, the research has valuable real-world application. If chronic stress measurably harms health, then stress-management programmes should reduce illness — and identifying high-stress, low-control jobs allows employers to intervene. This has clear economic benefit, reducing absenteeism and lowering the burden of stress-related illness on the NHS.

3 Sources of Stress

Life Changes

Life changes are significant, relatively infrequent events that require major **readjustment** (e.g. marriage, divorce, bereavement, moving house). Even positive events are stressful because they demand adaptation. They are measured by the Social Readjustment Rating Scale (Section 4).

Daily Hassles

Daily hassles are the minor, everyday irritations of life (traffic, queues, losing keys). Individually trivial, they **accumulate** to produce significant stress, and may matter more day-to-day than rare major life changes.

Workplace Stress

- **Workload** — high job demands (too much to do, time pressure) are a major source of stress.
- **Control** — low control over one's work increases stress. **Marmot et al.'s Whitehall study** found civil servants in low-control jobs had higher rates of stress-related illness than those with more control.

Evaluation

A strength of the daily-hassles explanation is supporting research. Studies have found that scores on the Hassles Scale are better predictors of psychological symptoms and ill health than life-change scores. This suggests that the steady accumulation of minor stressors may be more damaging than rare major events, supporting the validity of daily hassles as a meaningful source of stress and giving the concept practical relevance to everyday life.

However, a limitation of life-changes research is that it relies on retrospective self-report. Scales such as the SRRS ask people to recall events from the previous year, which is vulnerable to memory error and to the possibility that an *already ill* person recalls more life events. This weakens the internal validity of the correlations between life change and illness, because the measurement itself may be biased.

A further strength of the workplace-stress research is its real-world application and high validity. The Whitehall study examined real jobs over many years, so it has strong external validity, and its finding that low job control predicts illness has directly informed workplace policy (e.g. giving employees more autonomy). This shows the explanation is not only well-evidenced but economically useful, as reducing workplace stress lowers absenteeism and improves productivity.

On the other hand, sources of stress involve individual differences that the explanations overlook. The same event — a house move or a demanding job — is experienced as highly stressful by one person and as an exciting challenge by another, depending on cognitive appraisal, personality and hardiness. This shows that "sources of stress" cannot be treated as objectively stressful in the same way for everyone, limiting the predictive power of these explanations.

4 Measuring Stress

Self-Report Scales

- **The Social Readjustment Rating Scale (SRRS; Holmes & Rahe 1967)** — 43 life events each weighted in **life change units (LCUs)**; respondents total the LCUs for events experienced in the past year. Higher totals predict greater risk of stress-related illness.
- **The Hassles and Uplifts Scale (Kanner et al. 1981)** — measures minor daily stressors (hassles) and minor positive events (uplifts) that buffer stress.

Physiological Measures

- **Skin conductance response (SCR)** — electrodes detect tiny increases in sweat-gland activity that accompany sympathetic arousal; more arousal → higher conductance.
- Other measures include **cortisol** assays (saliva/blood) and heart-rate/blood-pressure monitoring.

Evaluation

A strength of physiological measures such as SCR is their objectivity. Because skin conductance and cortisol are recorded by instruments rather than reported by the participant, they are not affected by social desirability or memory error. This makes them highly reliable and replicable, giving researchers a scientific, quantifiable index of the body's stress response that is less open to bias than questionnaires.

However, a limitation of self-report scales is that they may lack validity. The SRRS treats every instance of an event (e.g. "divorce") as equally stressful, yet the same event means very different things to different people, and the scale ignores *cognitive appraisal*. It also mixes positive and negative changes together. This means the scale may not capture the individual's actual experience of stress, reducing its construct validity.

A counterpoint, however, is that physiological measures are not a complete solution either. Arousal measured by SCR or heart rate can be produced by excitement, exercise or caffeine as well as by stress, so a raised reading does not necessarily indicate a stressed psychological state. This shows that physiological and self-report measures each capture only part of the picture, and combining them gives the most valid assessment of stress.

5 Individual Differences in Stress

Personality Types A, B and C

Type	Characteristics & stress link
Type A	Competitive, time-urgent, hostile. Greater sympathetic arousal; **Friedman & Rosenman (1974)** found Type A men had roughly double the rate of coronary heart disease over 8.5 years.
Type B	Relaxed, easy-going, tolerant. Lower physiological stress response and lower associated health risk.
Type C	Suppresses emotions (especially negative ones) and is conflict-avoidant. Linked in some research to a raised vulnerability to cancer.

Hardiness (Kobasa 1979)

> **THE 3 CS OF HARDINESS**
>
> **Commitment** (active involvement in life), **Challenge** (seeing change as an opportunity rather than a threat) and **Control** (feeling in charge of one's life). Hardy individuals appraise stressors more positively and stay healthier under stress.

Evaluation

A strength of the Type A explanation is the strength of its founding evidence. Friedman and Rosenman's Western Collaborative Group Study followed around 3,000 men prospectively for 8.5 years, so it measured personality *before* illness developed rather than afterwards. Finding that Type A men had roughly double the incidence of CHD gives the link real predictive validity and reduces the problem of reverse causation.

However, a counterpoint is that later research has failed to replicate the strong Type A–CHD link. Subsequent studies found the relationship to be much weaker and suggested that it is mainly the **hostility** component of Type A — not competitiveness or time urgency — that predicts heart disease. This indicates the original Type A construct is too broad, and that a more precise focus on hostility better explains the data.

A strength of the hardiness concept is its useful real-world application. Because hardiness can be learned, "hardiness training" programmes teach people to reinterpret stressors as challenges and to focus on what they can control. The success of such programmes supports the idea that appraisal style affects health, and offers an economically valuable way to reduce stress-related absence in the workplace.

On the other hand, individual-differences research raises issues of measurement and gender bias. Personality types and hardiness are usually assessed by self-report questionnaires that are open to social-desirability bias, and much of the foundational work (e.g. the Western Collaborative Group Study) used **only male participants**. This androcentric, beta-biased basis means the findings may not generalise to women, limiting the validity of these explanations as universal accounts of who copes with stress.

6 Managing and Coping with Stress

Drug Therapy

Anti-anxiety drugs reduce the **physiological** arousal of stress — for example by enhancing the calming neurotransmitter GABA, or by blocking adrenaline's effects on the heart. *(The named drugs benzodiazepines and beta blockers were removed from the 2025 spec but remain valid examples.)*

Stress Inoculation Therapy (SIT; Meichenbaum 1985)

A **cognitive-behavioural** approach in three phases: **conceptualisation** (understanding the stressor), **skills acquisition and rehearsal** (learning coping techniques such as relaxation and positive self-talk), and **real-life application** (using the skills in increasingly stressful situations).

Biofeedback

The person receives real-time feedback about a bodily function (e.g. heart rate) and, through relaxation and operant conditioning, learns to bring it under **voluntary control**.

Gender Differences and Social Support

- **Gender differences** — Taylor et al. (2000) proposed women are more likely to **"tend and befriend"** (protect offspring, seek social contact) rather than fight-or-flight.
- **Social support** — **instrumental** (practical help), **emotional** (comfort) and **esteem** (encouragement) support buffer the effects of stress.

Evaluation

A strength of drug therapy is that it is fast-acting and effective. Anti-anxiety drugs reduce physiological symptoms quickly and require little effort or motivation from the person, which makes them suitable for someone who is acutely unwell or unable to engage with talking therapy. Research shows such drugs are more effective than placebo at reducing stress symptoms, supporting their use as a first-line option to bring symptoms under control.

However, a major limitation of drug therapy is that it treats the symptoms rather than the cause, and carries side effects. Once the drugs stop, the original stressor remains and symptoms can return, and long-term use risks dependence. In contrast, SIT and biofeedback aim to change how the person handles stress, so although they are slower, they may produce more durable benefits. This shows drug therapy is best seen as a short-term aid rather than a complete solution.

A strength of stress inoculation therapy is that it tackles the cause and builds lasting resilience. Because SIT changes the way a person appraises and prepares for stressors, the coping skills transfer to *future* situations, "inoculating" the person against later stress. This gives it an advantage over drugs in the long term. The trade-off is that SIT is time-consuming, expensive and demands high commitment, which may not suit everyone — so the best approach is often to combine methods.

Finally, social-support research has valuable application but also limitations. Evidence supports the **buffering hypothesis** — support protects against the impact of stress — and this underpins support groups and workplace wellbeing schemes that bring economic benefits through reduced absenteeism. However, gender differences (Taylor's tend-and-befriend) suggest social support may not work identically for everyone, and simply having people around is not the same as receiving the *type* of support that is actually needed.

These notes were prepared for Simply Psychology and cover spec section 4.3.7 of the AQA Psychology 2025 specification (A-level only, Paper 3). The named drugs (benzodiazepines, beta blockers) were **removed** in 2025; generic drug therapy remains. For deeper coverage, see simplypsychology.org.