

Wk9 CER: Drug effects on signaling

Your goal: Analyze your reaction-time data and explain how drugs alter neural signaling with a CER.

One product: Bar graph of baseline vs distraction average reaction time (labeled, with units) plus a CER explaining how a depressant or stimulant would alter the pattern via synaptic mechanism.

Question: What can a distraction graph show, and what independent evidence is needed to predict a drug effect?

Start the work

Preserve every trial and explain your inclusion rule; a timing difference describes the record before it explains a cause.

1. Answer both R1 warmups with actual data or labeled D1 recovery.
2. Verify the means and graph baseline versus distraction with G1.
3. Complete the assigned official analysis, or trace E1/E2 while recording the missing official input.
4. Revise a named-drug prediction and write the source-based CER; complete the check and appropriate extension.
5. Combine the graph and CER, follow P1's checks and submit or save the document.

If the section assignment is unavailable, save the document and ask the teacher for the correct assignment. After uploading, check that Schoology shows Submitted.

Words you will use

reaction time

The interval from a stimulus to a response.

stimulus

An event detected by a sensory system, such as a cue appearing.

response

The action that follows a stimulus.

baseline

The named comparison condition before adding the distraction task.

condition

One setting being compared in a task.

trial

One attempt under a stated condition.

mean

The sum of values divided by their count.

millisecond / ms

One thousandth of a second.

bar graph

A display whose bar heights represent values for named categories.

axis

A labeled reference line showing categories or a numerical scale.

error bar

A plotted interval showing a defined measure of variability or uncertainty.

neuron

A cell specialized for receiving and transmitting information.

action potential

A brief electrical signal that travels along a neuron membrane.

chemical synapse

A junction where a cell communicates with another using released transmitter.

neurotransmitter

A chemical messenger released by a neuron.

synaptic cleft

The small gap across which transmitter travels at a chemical synapse.

receptor

A protein that binds a signal molecule and influences cell activity.

excitation

Input that makes a neuron more likely to fire.

inhibition

Input that makes a neuron less likely to fire.

reuptake

Removal of released transmitter from the gap back into cells.

CNS

Central nervous system: brain and spinal cord.

depressant

A drug category associated with reduced CNS activity; the exact target depends on the drug.

stimulant

A drug category associated with increased CNS activity or alertness; this does not guarantee faster task performance.

benzodiazepine

A drug class that enhances GABA-related inhibition at GABA type A receptors.

diazepam

A benzodiazepine used here only as a paper example.

GABA

Gamma-aminobutyric acid, a neurotransmitter with inhibitory effects at many brain synapses.

GABA type A receptor

A receptor involved in inhibitory signaling and acted on by benzodiazepines.

adenosine

A signaling molecule whose brain receptors can be blocked by caffeine.

caffeine

A stimulant that blocks adenosine receptors.

mechanism

The process explaining how an effect can occur.

tolerance

A decrease in a particular effect with repeated exposure.

receptor sensitivity

How strongly receptor signaling responds under stated conditions.

analogy

A comparison used to reason; similarity does not prove the same cause.

CER

Claim, evidence and reasoning: a conclusion supported by observations or sources and an explanation.

claim

The conclusion being defended.

evidence

The specific data or source information used to support a claim.

reasoning

The explanation connecting evidence to a claim.

reflex arc

The neural pathway that mediates an automatic response.

myelin

Insulation around some axons that supports faster signal conduction.

E1: a chemical synapse is not a speed switch

At a chemical synapse, a sending neuron releases neurotransmitter into a small gap. The transmitter binds receptors on a receiving cell. Its effect depends on the receptor and cell.

Excitatory input can make a receiving neuron more likely to fire an action potential. Inhibitory input can make firing less likely. A neuron combines many inputs. Neither term means that every signal travels faster or slower.

Reuptake removes transmitter from the gap into cells. Changing release, removal or receptor action can change signaling. A drug name and target are needed to explain a particular mechanism.

Reaction time includes detecting a stimulus, processing it and producing a response. A condition difference cannot locate one synapse or prove which chemical caused it. A voluntary reaction task is not a measurement of a single reflex arc.

E2: two named drug mechanisms

NIDA identifies diazepam as a benzodiazepine, a type of CNS depressant. Benzodiazepines act at GABA type A receptors. They enhance inhibitory GABA signaling and can produce drowsiness or poor coordination.

If that drowsiness interferes with responding to a cue, a longer reaction time is a reasonable prediction. It is an inference from the mechanism, not a measured drug result from your distraction graph.

Caffeine is a stimulant. The Institute of Medicine describes its blocking of adenosine receptors as important to its brain effects. Blocking an inhibitory adenosine influence can change activity in neural circuits.

MedlinePlus states that caffeine stimulates the central nervous system and can make someone feel more awake. This is a source fact about alertness, not proof of a specific improvement on a reaction task.

A shorter reaction time is a conditional caffeine prediction if increased alertness improves that task. Neither source guarantees that everyone responds faster. Do not treat all stimulants or depressants as identical mechanisms.

D1: original practice inputs

These are the March 17 RT1 practice rows for fictional subject F1, reused for arithmetic and graph practice. No person was tested. They represent five baseline trials and five distraction trials, all in milliseconds (ms).

Baseline trials 1 through 5 (A01 to A05): 220, 215, 225, 360, 230 ms.

Distraction trials 1 through 5 (B01 to B05): 270, 280, 265, 285, 275 ms.

All values are included, including A04, which keeps its March 17 review flag. There is no drug condition, no observed receptor measurement and no claim that five trials are five people. Use D1 only when the actual approved table is missing.

Mean equals sum divided by trial count. Find each mean, then subtract baseline mean from distraction mean. A positive difference means a longer average interval. A negative difference means a shorter one. Zero means equal means.

Check after trying: each sum uses all five values in its condition, A04 stays in the baseline sum, and each divisor is 5. This is a practice pattern, not observed impairment.

G1: graph and evidence rule

Make a bar graph with condition on the horizontal axis and mean reaction time (ms) on the vertical axis. Use equal intervals starting at zero. Give it a title and label both bars.

For D1, a vertical scale from 0 to 300 ms in 50 ms intervals fits both means. Mark each bar at the mean you calculated. State that each mean represents five invented trials.

For your actual table, preserve its units and verified averages. Ruler distance in cm is not time in ms. Obtain the approved conversion before labeling distance values as reaction times.

Add an error note naming a limitation of your method or data. For D1, note that the values are invented and contain no measured device error. Do not invent error bars. Use them only with a defined, verified calculation.

The graph supports a comparison of the two named conditions. It cannot establish a drug mechanism, predict a drug effect in milliseconds or show statistical significance by itself. Cite E2 separately for any drug-mechanism claim.

X1: tolerance without a guaranteed outcome

Tolerance means that a particular effect becomes smaller with repeated exposure. It does not establish that every effect disappears or that a person can safely drive.

A 1994 cell study by Klein and colleagues found reduced interaction between GABA and benzodiazepine binding sites after exposure in engineered cells. It studied receptor behavior, not human reaction time over weeks.

For the extension, consider an explicitly hypothetical person whose receptors become less responsive to a benzodiazepine over several weeks. Hold the task and exposure conditions the same in this paper model.

If reduced receptor responsiveness weakens the relevant inhibitory effect, the added reaction-time delay might become smaller. This conditional chain does not establish that tolerance occurred, when it occurred or how much delay changed.

Receptor sensitivity is a possible part of the explanation, not a measurement in D1. Keep the human prediction separate from the cell-study evidence.

R1: warmups, recovery and checks

Before graphing, answer both warmups. Was your actual distraction mean faster, slower or equal to baseline? State both means and the signed difference with units. If missing, do this with D1 and label it practice.

Then predict whether a depressant could shift reaction time in the same direction as your distraction pattern. Keep this first prediction. After E2, revise it to name diazepam, its receptor mechanism and one uncertainty.

If your actual pattern is unchanged or faster under distraction, report it honestly. Do not rewrite the values to match an expected story. An unexpected graph does not overturn a receptor mechanism or prove a drug effect.

For support, build a CER in separate sentences: name the drug and a conditional prediction; state the graph comparison; cite the independent mechanism source; explain the link and its limits.

Self-check scenario: a hypothetical distraction condition adds 60 ms. Predict the possible direction for caffeine, name the adenosine-receptor mechanism and explain why 60 ms cannot give its drug effect size.

Check method: use E2 for the possible direction and the receptor action, keep the direction conditional, then ask whether the 60 ms record measured caffeine at all.

Work alone using the complete cards and model if a partner is absent. Restart at the first unfinished step: verify means, graph, read the mechanism, draft CER, then check the combined record.

P1: Work and submission checks

Recover the March 17 reaction-time table and its approved method locally. Keep actual observations distinct from D1. No drug testing, personal medication report or treatment change is part of this lesson.

The 25–45 minute block is reserved for the assigned signaling analysis. Today's official work is Activity 2.1.4 The Secret to Signals, with optional Activity 2.1.5 Neuropharmacology. Confirm the assigned screens and stopping point.

The official signaling section also needs the teacher Action Potential handout, eight description cards matched to its graph, and three membrane diagrams with ion directions and charges. Its notebook prompts and knowledge check remain separate from this graph and CER.

If the official task is unavailable, record what is missing and use the block to trace E1 and E2 and compare their evidence. This is original practice. It does not complete official screens.

No new homework is due before this class. The March 17 prerequisite was Reflex arc and myelin, 25 minutes, in Lab Notebooks. Ask for the missing diagram; do not invent its contents.

Weekly requirements include actual reflex/reaction data analysis, the teacher-posted data/model packet, the tracker and online evidence through Activity 2.1.7 Here Comes the Hammer. Patient-case clues for the unit challenge are not supplied here.

The March 22 weekend item is Submit reflex evidence. Assemble the reflex-arc diagram, actual reaction-time table, comparison graph and CER when available. Confirm with the teacher where to submit it.

For March 18, combine the graph and CER in one document. Use Wk9 CER: Drug effects on signaling in your section. If access fails, save the document and tell the teacher. Check Schoology after uploading.

M1: a different invented record with no mean distraction gap

This worked model is invented. Its baseline trials are 300, 320, 280 and 300 ms. Its distraction trials are 300, 280, 320 and 300 ms. No measurements occurred.

Each condition sums to 1200 ms. Dividing by four gives 300 ms for each mean. The distraction-minus-baseline difference is 0 ms.

Graph equivalent: title, M1 invented mean reaction time; horizontal axis, baseline and distraction; vertical axis, mean reaction time (ms), from 0 to 350. Both bars reach 300 ms.

The graph shows no mean difference in this invented record. It measures no drug response. E2 independently explains that diazepam enhances inhibitory GABA signaling at GABA type A receptors.

A defensible claim is that diazepam-related drowsiness could lengthen a response on a task if it interferes with responding. That prediction does not come from the zero gap.

Reasoning connects the named receptor mechanism with possible drowsiness and task performance. It does not assert a measured delay, prove the same mechanism as distraction or predict every person's response.

Try the decision

What evidence supports the graph-plus-CER conclusion?

- Describe the graph's conditions, then use E2 receptor evidence for a conditional named-drug prediction with uncertainty.
- Use the distraction gap as the number of milliseconds any depressant will add.
- Wait for personal drug-testing results before writing any mechanism explanation.

Explain your choice using the reading. Discuss it or say it to yourself. This practice does not add another submission.

Your 80-minute flow

0–8 minutes	Calculate the starting comparison and initial prediction.
8–25 minutes	Verify means and create the labeled graph with limitation note.
25–45 minutes	Complete assigned screens if available; otherwise trace E1/E2 and compare receptor mechanisms.
45–55 minutes	Revise the prediction; complete source mechanism and caffeine self-check.
55–75 minutes	Draft and revise CER; complete conditional tolerance extension if core work is secure.
75–80 minutes	Use the check record to identify your next action, then submit or save if access fails.

Your response record

Use these labels on paper or in your document. The space below also works when this page is printed.

Both warmups and the numerical starting point

Use R1 to state your actual or D1 means, signed difference and faster/slower/equal result. Make the initial depressant-direction prediction, then revise it after E2.

Label actual or invented practice. A positive distraction-minus-baseline difference means longer reaction time. Keep both versions of your prediction.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.



Labeled condition graph and error note

Verify the two means and graph baseline versus distraction using G1. Include title, axes, units, scale, both bars, trial counts, data origin and an error or limitation note.

Use D1 only for marked recovery. Calculate its means from all five values in each condition. Do not add a drug bar or fabricate error bars. Ask for missing class data; do not invent it.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.



Source-based mechanism and conditional prediction

Choose diazepam or caffeine from E2. Name its drug class, receptor target and signaling action. State a possible reaction-time direction and why the graph cannot establish that drug effect.

Use E1 to distinguish activity from conduction speed. Cite the named source in E2. D1 supports only its two invented task conditions.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.

Graph plus signaling CER

Write a CER with your graph comparison, the independent receptor evidence and a conditional prediction for the named drug. Explain the mechanism and explicitly limit the graph's role. Combine it with the labeled graph in one document.

Use the R1 sentence sequence if needed. M1 models a different record. Neither the sign nor size of a distraction gap proves a drug mechanism or effect.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.

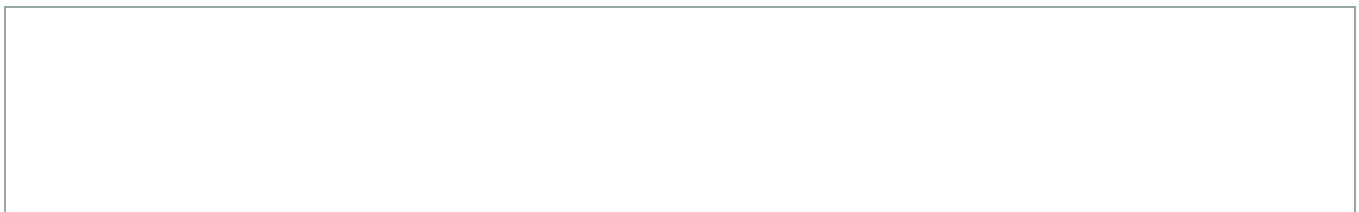


Caffeine self-check

Answer the R1 hypothetical 60 ms question. Explain the possible direction, adenosine-receptor mechanism and why the drug effect cannot be calculated from 60 ms.

A source fact about feeling more awake does not guarantee quicker responses. Check your answer against the R1 check method after trying.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.



Tolerance extension

For X1's hypothetical reduced receptor sensitivity over weeks, predict how an added delay might change. Explain the receptor link and why the cell study cannot prove that human result.

Make the chain conditional. A smaller relevant inhibitory effect could mean a smaller added delay. Do not infer safety or a time course.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.

Check your evidence and assignment

Identify the table and diagram you used. Include evidence from the assigned official analysis only after doing it, and ask for missing inputs. Check P1's weekly and weekend items separately. Save or submit the single graph-plus-CER document. After uploading, check that Schoology shows Submitted.

Original recovery is not actual lab completion. Confirm where the weekend item goes. No personal health information is requested.

Use this space or the same labeled section in your existing document. Add paper when your table, graph or drawing needs more room.

Check your product

- The graph preserves the data origin, condition means, labels and units.
- The CER names a drug and independently sourced receptor mechanism.
- The prediction is conditional; the distraction gap establishes no drug effect.
- Label the supplied practice clearly. Keep evidence from your actual lab and official task separate, and check Schoology after uploading.

Keep the limit: The reaction-time comparison supplies no drug exposure evidence. Mechanism-based predictions are conditional and cannot establish personal diagnosis, drug effect size or safe activity.

Turn in your work

Submit graph and CER as a single combined document. Section 1292 uses assignment 8544580087; section 6792 uses <https://clevelandmetro.schoology.com/assignment/8544580109>.

If the section assignment is unavailable, save the document and ask the teacher for the correct assignment. After uploading, check that Schoology shows Submitted.

[Section 1292: Wk9 CER: Drug effects on signaling](#)

[Section 6792: Wk9 CER: Drug effects on signaling](#)

Carry forward

Use the graph for its measured conditions and independent receptor evidence for a bounded drug prediction.

Keep the graph and signaling CER with evidence from your actual reflex work; request missing prior or official-task materials. Which signaling claim requires evidence beyond a reaction-time graph?

Sources and resources

[Open today's page](#)

OS-SYN · Communication Between Neurons, section 12.5

OpenStax Anatomy and Physiology 2e

<https://openstax.org/books/anatomy-and-physiology-2e/pages/12-5-communication-between-neurons>

NIDA · Misuse of Prescription Drugs, pages 13–14

National Institute on Drug Abuse, December 2018

<https://nida.nih.gov/sites/default/files/2609-misuse-of-prescription-drugs.pdf>

IOM-CAF · Pharmacology of Caffeine: Caffeine and Adenosine Receptors

Institute of Medicine, 2001, NCBI Bookshelf

<https://www.ncbi.nlm.nih.gov/books/NBK223808/>

NLM-CAF · Caffeine

MedlinePlus, National Library of Medicine

<https://medlineplus.gov/caffeine.html>

KLEIN · Benzodiazepine treatment causes uncoupling of recombinant GABAA receptors expressed in stably transfected cells

Klein, Whiting and Harris, 1994; PubMed abstract, PMID 7964756

<https://pubmed.ncbi.nlm.nih.gov/7964756/>