Case Teaching Notes

for

"The Campus Coffee Shop: Caffeine Conundrums"

by

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Introduction / Background

Since caffeine is a widely used substance, especially by college age students, it serves as a real-world connection to many students' lives. The case study is separated into sections covering background information on caffeine, cell biology and signal transduction, Parkinson's Disease, cardiovascular effects, and addiction/withdrawal. The case was designed in modular format so that each section can be used independently or as a series of selected sections dictated by topic/curriculum needs.

The case was designed for students who have already completed general/introductory biology and introductory chemistry. The sections of the case can be used for a variety of science and health related courses including anatomy and physiology, disease related courses, genetics, cell biology, molecular biology, biochemistry, and neuroscience.

Classroom Management

Generally, each section of this case study can be implemented in a 75-minute class period. However, implementation time may vary depending on what advance work/reading is assigned, if computers are available in the classroom for in-class investigation/information gathering, and desired time for discussion. For example, Question 1 of Part 3 (Parkinson's Disease) is designed as a pre-assignment for students to gather basic information about the cellular and molecular basis of Parkinson's Disease prior to class. The remaining questions in Part III can then be used in class as the basis of discussion. Alternatively, Questions 3 and 4 of Part III could be the focus of an entire class session focusing on experimental design, hypothesis testing, and data collection.

Since not all the related content is contained in the case study, many of the sections rely on students searching Internet resources and compiling information to answer the questions. It is suggested that students have access to computers with Internet connections or other reference sources to answer some of the questions. Suggested time limits for in-class data gathering depends on the depth of the question but generally should be limited to 5–10 minutes.

Variation 1: Different case sections can be used together as a thematic learning unit. For example, a neuroscience class could use the introduction, signal transduction, Parkinson's Disease, and addiction/withdrawal sections (Parts I, II, III, and V) while a physiology class might use the introduction, addiction, and cardiovascular sections (Parts I, V, and IV).

Variation 2: The introduction section (Part I) can be assigned to students as homework in preparation for one or more of the subsequent case sections. The questions can be the basis for initial classroom discussion. The instructor can implement a jigsaw configuration by creating groups of 2–3 students and assigning each

group one question from a chosen section. The students are given time to search and think about answers, then report back to the entire class.

Other sections may also be used in a jigsaw format. For example, the cardiovascular system section (Part IV) might start with a jigsaw activity for students to obtain basic information about blood pressure: What is vasoconstriction? What is MAP? What is cardiac output and peripheral resistance? From this information, the students would then work together to design the experiment in Question 1 and also work together on Question 4. This could then be followed up with a similar jigsaw activity to address questions on catecholamines and hypertension.

Variation 3: Parts II, III, IV, and V could be used for concept reinforcement and integration. Since caffeine serves as the common thread throughout all parts of the case study, using multiple modules together can illustrate connections between concepts and provide students with the opportunity to develop higher order thinking skills.

Part I: Introduction

Learning objectives:

- Identify sources of caffeine in foods and drinks.
- Describe the chemical structure of caffeine and general physiological effects of caffeine consumption.
- Identify physiological effects of caffeine
- Identify some organs and systems that are affected by caffeine
- Hypothesize biological reasons for differences in caffeine effects and metabolism in different individuals.

Part II: Cell Biology and Signal Transduction

Learning objectives:

- Explain which molecules in the nervous system interact with caffeine.
- Explain and illustrate how a G-protein receptor works.
- Compare and contrast the actions of an agonist and an antagonist.
- Predict the effects of an antagonist on receptor function.

Pitfalls to address:

Many students have trouble with the idea that an antagonist binds to a receptor, but does not produce intracellular changes. An antagonist blocks the action of the agonist (in this case, adenosine). It does not cause the opposite effect inside the cell. Adenosine increases the level of cAMP in the postsynaptic cell. When caffeine, the antagonist, binds to the receptor, it **does not** reduce the level of cAMP in the postsynaptic cell. It just does **not increase** it.

Part III—Caffeine and Parkinson's Disease

Learning objectives:

- Determine the relationship between caffeine consumption and Parkinson's Disease incidence.
- Describe the molecular and cellular basis of Parkinson's Disease.
- Hypothesize biological explanations for the correlation between caffeine and PD.

Part IV—Caffeine and the Cardiovascular System

Learning objectives:

- Explain the molecular basis of changes in blood pressure in response to caffeine.
- Calculate changes in blood pressure with changes in biological variables.

- Determine potential outcomes of interactions between caffeine and catecholamine signaling pathways.

Part V—Caffeine and Addiction / Withdrawal

Learning objectives:

- Identify symptoms of caffeine withdrawal.
- Explain why pain medication often includes caffeine as an ingredient.
- Differentiate between drug dependence and drug addiction. Categorize caffeine into one of these states.
- Explain how adenosine and dopamine contribute to caffeine dependence or addiction.

Blocks of Analysis

This case uses caffeine as an example to investigate molecular and cellular biological concepts—more specifically, how chemicals affect brain function, how cell signaling works, caffeine's association with Parkinson's Disease, how blood pressure and heart rate change as a function of caffeine consumption, and how dependence may result from caffeine intake. The case targets students' skills in information gathering, critical analysis of data, experimental design, hypothesis testing, and explaining molecular processes/ interactions.

Caffeine is absorbed in the gastrointestinal tract and generally complete absorption occurs within one hour after consumption. Since caffeine is hydrophobic, it easily passes through cell membranes. Caffeine acts as an adenosine antagonist, competitively binding to the adenosine receptor. Adenosine is a normal cell component. Once it binds to its receptor, intracellular events occur, including changes in intracellular calcium leading to alterations in neuronal electropotentiation. Caffeine blocks these actions so that no intracellular changes occur.

Caffeine intake has been associated with decreased incidences of Parkinson's Disease (PD). Confounding variables in these studies include smoking, gender, and age. The exact molecular mechanism for the correlation between increased caffeine consumption and decreased incidence of PD is unknown but may involve increased signal transmission in cells with dopamine receptors.

Caffeine is typically thought of as a stimulant since after caffeine consumption heart rate can increase and people feel alert, are able to concentrate, and have a reduced appetite. However, different concentrations of caffeine can have different effects on different people. Thus for many people, caffeine consumption can lead to habit forming behavior or, in extreme cases, substance dependence. However, there is little evidence that caffeine consumption leads to any significant negative effects on health, in contrast to other stimulants and drugs of addiction.

Answer Key

Answers to the questions posed in the case study are provided in a separate answer key to the case. Those answers are password-protected. To access the answers for this case, go to **the key.** You will be prompted for a username and password. If you have not yet registered with us, you can see whether you are eligible for an account by reviewing our **password policy and then apply online** or write to **answerkey@sciencecases.org**.

Suggested Follow-up Assignments

1. Students can choose one of the topics presented in the sections of the case study and explore current research findings by searching for scientific research articles using PubMed (**http://www.ncbi.nlm.nih.gov/PubMed**)

and summarize the research findings in the literature found. The students can then present their summary to the class as a short presentation.

2. Students can explore and evaluate scientific data and popular science publications as to the potential relationships between caffeine consumption and coronary heart disease, osteoporosis, pregnancy, and cancer.

Caffeine has been implicated as an independent risk factor for coronary heart disease. Repeated use of caffeine that results in increased blood pressure may lead to increased risk of coronary heart disease. However, other studies indicate no correlation or risk with caffeine consumption and coronary heart disease.

Again, primary scientific literature is inconsistent and inconclusive about caffeine's connection with cancer. Some studies suggest that caffeine acts as an antioxidant, leading to a decrease in cancer incidences (shown in ovarian cancer). Some studies suggest that there is no correlation between caffeine consumption and cancer.

General conclusions about caffeine and connections with diseases need to be made with caution. Careful analyses of the types of studies performed, subjects enrolled in the study, how the data was analyzed, and other variables need to be taken into consideration.

References

American Parkinson's Disease Association Inc. **http://www.apdaParkinson's.org/user/index.asp**. Accessed May 2005.

Betarbet, R., Sherer, T.B.,and Greenamyre, J.T. (2002). Animal models of Parkinson's disease. *Bioessays* 24(4): 308–318.

Fredholm, et al. (1999). Actions of caffeine in the brain with special reference to factors that contribute to its widespread use. *Pharmacological Reviews* 51(1): 83–133. Available online at **http://www.pharmrev.org.**

G-protein animations—the following websites are not specific for adenosine, but have good animations for G-proteins in general. Accessed May 2005.
http://www.blackwellpublishing.com/matthews/neurotrans.html
http://dkc.jhu.edu/~teal/gprotein.html
http://w3.uokhsc.edu/human_physiology/presentation/G-prote.gif

Hartley, T.R. et.al. (2000). Hypertension risk status and effect of caffeine on blood pressure. *Hypertension* 36: 137–141.

How does caffeine affect cardiovascular health? **http://my.cardiovalens.com/articles/articledisplay.asp?article_id=mrf&articledetail_id=mrf_cofe**. Accessed May 2005.

Kirchheimer (2004). Caffeine Withdrawal Is Real. CBSNEWS.com **http://www.cbsnews.com/stories/2004/09/30/health/webmd/main646620.shtml**. Accessed May 2005.

Kluger, et al. (2004). The buzz on caffeine. *Time*, December 20, 2004.

The Michael J. Fox Foundation for Parkinson's Research. **http://www.michaeljfox.org/** Accessed May 2005.

National Institute of Neurological Disorders and Stroke (NINDS). 2004 Caffeine Information Summary. Available online at **http://www.ninds.nih.gov/funding/research/parkinsonsweb/drug_summaries/caffeine_pr.htm**. Accessed May 2005.

Reid, TR. (2005). Caffeine. *National Geographic*. January.

Ross, G.W. et al. (2000) Association of coffee and caffeine intake with the risk of Parkinson's disease. *JAMA* 283(20): 2674–2679.

Suleman, A., Siddiqui, N.H. Haemodynamic and Cardiovascular Effects of Caffeine **http://www.priory.com/pharmol/caffeine.htm**. Accessed May 2005.

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