

VTPP 627  
Biomedical Physiology II

Dr. J.D. Herman

Office hours: MWF — 10:00 to 11:00 a.m., TTh — 8:30 to 9:30 a.m.  
Room 316 VIDL All others by appointment (979-862-7765)  
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**Lectures:** TTh 9:35 to 10:50 a.m., Room 101A VENI

**Text:** Human Physiology; From Cells to Systems. L. Sherwood, 8<sup>th</sup> edition

**Prerequisites:** VTPP 623 and Graduate Student Classification

**Supplemental**

**Information:** <http://ecampus.tamu.edu/>

**Learning Outcomes:**

By the end of the course, the student will have:

- began to develop clinical reasoning skills
  - Recognition of common clinical pathology values
  - Understanding of implications of abnormal values
- gained further insight into homeostatic control of
  - Acid/Base balance
  - Electrolyte balance
  - GI system
  - Endocrine systems
  - Reproduction
- opportunity use computer modeling to gain insight into physiologic systems

**Classroom Communication Concerns:**

Please be advised that there is a form available in the VTPP Office (Rm 332, VMA) to express any concerns, problems, etc., you may have with this course that cannot be resolved by meeting with the instructors.

**Americans with Disabilities Act:**

The Americans with Disabilities Act (ADA) is a federal anti-discrimination statute that provides comprehensive civil rights protection for persons with disabilities. Among other things, this legislation requires that all students with disabilities be guaranteed a learning environment that provides for reasonable accommodation of their disabilities. If you believe you have a disability requiring an accommodation, please contact Disability Services, currently located in the Disability Services building at the Student Services at White Creek complex on west campus or call 979-845-1637. For additional information visit <http://disability.tamu.edu>.

Please present appropriate documentation of any disability requiring accommodation to the instructor during the first week of classes.

**Plagiarism:**

The handouts used in this course are copyrighted. By “handouts”, we mean all materials generated for this class, which includes but are not limited to syllabi, quizzes, exams, lab problems, in-class materials, review sheets, and additional problem sets. Because these materials are copyrighted, you do not have the right to copy the handouts unless we expressly grant permission.

As commonly defined, plagiarism consists of passing off as one’s own ideas, words, writings, etc., which belong to another. In accordance with this definition, you are committing plagiarism if you copy the work of another person and turn it in as your own, even if you have the permission of that person. Plagiarism is one of the worst academic sins, for the plagiarist destroys the trust among colleagues without which research cannot be safely communicated.

If you have any questions regarding plagiarism, please consult the latest issue of the Texas A&M University Student Rule, under section "Scholastic Dishonesty".

## Academic Integrity Statement

"An Aggie does not lie, cheat or steal, or tolerate those who do."

If you have any questions or concerns over Academic Integrity, please refer to the Honor Code website at <http://aggiehonor.tamu.edu>.

## Attendance

Class attendance is expected. Your arrival to the class on time will be appreciated. Should you arrive late, please enter via the door at the back of the classroom and quietly apologize to the students who you may disrupt as you take your seat in the classroom. If the first in-class quiz question has been completed, you will not have the opportunity to answer this question.

"The university views class attendance as an individual student responsibility. Students are expected to attend class and to complete all assignments." University rules related to excused and unexcused absences are located on-line at <http://student-rules.tamu.edu/rule07>."

Make-up examinations will only be given for excused absences. The format for make-up examinations will not necessarily be the same as for scheduled examinations; the format will be at the instructor's discretion (eg. short answer, essay, oral, etc.). Note: An Explanatory Absence from Class (<http://shs.tamu.edu/attendance>) does not constitute a University-approved excuse for a major exam.

The instructor will designate the date and time of make-up examinations.

## Make-up Policy:

If an absence is excused, the instructor will either provide the student an opportunity to make up any quiz, exam or other work that contributes to the final grade or provide a satisfactory alternative by a date agreed upon by the student and instructor. If the instructor has a regularly scheduled make up exam, students are expected to attend unless they have a university approved excuse.

The make-up work must be completed in a timeframe not to exceed 30 calendar days from the last day of the initial absence.

The reasons absences are considered excused by the university are listed below. See Student Rule 7 for details (<http://studentrules.tamu.edu/rule07>). The fact that these are university-excused absences does not relieve the student of responsibility for prior notification and documentation. Failure to notify and/or document properly may result in an unexcused absence. Falsification of documentation is a violation of the Honor Code.

- 1) Participation in an activity that is required for a class and appears on the university authorized activity list at <https://studentactivities.tamu.edu/app/sponsauth/index>
- 2) Death or major illness in a student's immediate family.
- 3) Illness of a dependent family member.
- 4) Participation in legal proceedings or administrative procedures that require a student's presence.
- 5) Religious holy day. NOTE: Prior notification is NOT required.
- 6) Injury or illness that is too severe or contagious for the student to attend class.
  - a) Injury or illness of three or more class days:  
Student will provide a medical confirmation note from his or her medical provider within one week of the last date of the absence (see Student Rules 7.1.6.1)
  - b) Injury or illness of less than three class days:  
Student will provide one or both of these (at instructor's discretion), within one week of the last date of the absence:
    - (i.) Texas A&M University Explanatory Statement for Absence from Class form available at <http://attendance.tamu.edu>
    - or (ii.) Confirmation of visit to a health care professional affirming date and time of visit.
- 7) Required participation in military duties.
- 8) Mandatory admission interviews for professional or graduate school that cannot be rescheduled.

In cases where prior notification is not feasible (e.g., accident or emergency) the student must provide notification by the end of the second working day after the absence, including an explanation of why notice could not be sent prior to the class.

**Grades:**

- A >= 585 points
- B = 520 – 584 points
- C = 455 – 519 points
- D = 390 – 454 points

**Clinical Case Design** (50 points each) — Design two hypothetical clinical cases, one selected from the first half of the course and one selected from material presented in the second half of the course. Submit your cases including expected laboratory values along with an outline of an appropriate treatment. Each case should follow the format below:

- Signalment
- History of Present Illness
- Lab data (with abnormalities explained)
- Discussion (rationale, a physiological basis for determining the problem)
- Treatment options

Due date:            March 6 - Fluid balance, electrolyte balance, acid/base balance, general endocrine, thyroid function, growth, adrenal cortex, sexual dimorphism, gametogenesis,  
                              April 24- reproduction, pregnancy, digestion, obesity

If you have any questions, concerns, or anxieties about what topics are to be covered, the appropriateness of a topic, format to be used with respect to bonus assignments, please see Dr. Herman prior to the due date of the assignment.

**Evaluations:**

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Exam 1	100 points
Exam 2	100 points
Exam 3	100 points
Clinical Cases (2)	100 points
Final	150 points
Recitation	100 points
Total	650 points

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**Bonus points:**

A total of 50 additional points can be earned by correctly answering questions in class using the assigned iClicker system. Questions will be asked at either the beginning or end of each class period for a total of at least 1 question per day and will cover the day’s assigned material. Answering all questions correct for the day will result in earning 2 points. Answering less than all of the questions correctly will result in an appropriate fraction of points to be awarded. No points will be awarded for answering all of the questions for a class period incorrectly. In the likely event that we do not have 25 lecture days (therefore 50 points), the total number of points possible will be adjusted to 50 points.

Week	Date	Reading Assignment / Topic	Objectives
1	Day 1		<ul style="list-style-type: none"> <li>• Course introduction</li> <li>• Reminder of VTPP 423</li> </ul>
	Day 2	Pp 554 - 568, 520 - 528  Roles & Regulation of Sodium	<ul style="list-style-type: none"> <li>• Describe the receptors involved in the monitoring of ECF volume (e.g., high-pressure baroreceptors and low-pressure cardiopulmonary stretch receptors), and diagram the neural reflex regulation of renal Na<sup>+</sup> and water excretion.</li> <li>• Describe the regulation of Na<sup>+</sup> reabsorption along the nephron, including the effects of sympathetic nerves, angiotensin II, aldosterone, and atrial natriuretic peptide.</li> <li>• Describe the role of the renin-angiotensin-aldosterone system in the regulation of systemic arterial blood pressure in volume-replete and volume-depleted states and in secondary forms of hypertension.</li> <li>• Identify the normal range of dietary sodium intake, sodium distribution in the body, and routes of sodium excretion. Explain the roles of antidiuretic hormone, aldosterone, angiotensin, and atrial natriuretic hormone in the regulation of sodium balance.</li> </ul>
2	Day 3	Pp 528 – 531  Roles & Regulation of Potassium	<ul style="list-style-type: none"> <li>• Identify the normal range of dietary K<sup>+</sup> intake and major routes of K<sup>+</sup> loss from the body.</li> <li>• Describe K<sup>+</sup> distribution within the body, extrarenal K<sup>+</sup> homeostasis, and the role insulin, epinephrine, and aldosterone play in the movement of K<sup>+</sup> between intracellular and extracellular pools. Describe the K<sup>+</sup> shift caused by acidosis.</li> <li>• Identify the tubular sites of K<sup>+</sup> reabsorption and secretion.</li> <li>• Describe the factors that regulate K<sup>+</sup> secretion in the collecting duct (i.e., aldosterone, plasma K<sup>+</sup>) and distinguish these from factors that alter K<sup>+</sup> secretion at this site (i.e., luminal fluid flow rate, acid-base disturbances, anion delivery).</li> <li>• Identify the normal range of dietary potassium intake, potassium distribution in the body, and routes of potassium excretion. Explain how acute changes in aldosterone, insulin, and acid/base concentrations affect the plasma potassium concentration and the movement of potassium into and out of the intracellular compartment. Explain the chronic regulation of body potassium balance and plasma potassium levels by aldosterone through its actions on renal excretion, intestinal excretion, and dietary appetite/absorption.</li> </ul>
	Day 4	Pp 721 – 731  Roles & Regulation of Calcium	<ul style="list-style-type: none"> <li>• Identify the normal range of dietary Ca<sup>2+</sup> and phosphate intake, major storage pools of Ca<sup>2+</sup> and phosphate, and major routes of Ca<sup>2+</sup> and phosphate loss from the body. Describe the regulation of plasma Ca<sup>2+</sup> by calcitonin and phosphate by parathyroid hormone.</li> <li>• Describe the renal regulation of Ca<sup>2+</sup> and phosphate transport by PTH, calcitonin, and 1,25-dihydroxy vitamin D (calcitriol), and distinguish from other factors that alter their transport (ECF volume, acid-base disorders).</li> <li>• Describe the role of the kidney in the production of 1,25-dihydroxy vitamin D (calcitriol).</li> </ul>

- Describe the functions of the osteoblasts and the osteoclasts in bone remodeling and the factors that regulate their activities.
- Describe the regulation of parathyroid hormone secretion and the role of the calcium-sensing receptor.
  - Understand the causes and consequences of a) over-secretion, and b) under-secretion of parathyroid hormone, as well as its therapeutic use.
  - Identify the sources of vitamin D and diagram the biosynthetic pathway and the organs involved in modifying it to the biologically active  $1,25(\text{OH})_2\text{D}_3$  (1-25 dihydroxy cholecalciferol).
  - Identify the target organs and cellular mechanisms of action for vitamin D.
- Describe the negative feedback relationship between parathyroid hormone and the biologically active form of vitamin D [ $1,25(\text{OH})_2\text{D}_3$ ].
- Name the stimuli that can promote secretion of calcitonin, its actions, and identify which (if any) are physiologically important.

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Day 5 Pp 563 – 578

Traditional Acid Base Balance

- Identify the normal range of pH values, and the upper and lower limits compatible with life. Describe the role of buffers in maintaining pH, including the roles of the lungs and kidneys.
- Describe the respiratory and renal regulation of the  $\text{CO}_2/\text{HCO}_3^-$  buffer system, which allows a buffer with a pKa of 6.1 to be physiologically important in the maintenance of the normal plasma pH of 7.4.
- Distinguish between  $\text{CO}_2$ -derived (volatile acid) and nonvolatile acid, the relative amounts produced each day through dietary intake and cellular metabolism, and the normal routes of loss from the body.
- Identify the major sites of reabsorption (and secretion) along the nephron, emphasizing the importance of  $\text{H}^+$  secretory mechanisms in this process. Describe the cellular mechanisms responsible for net transepithelial movement of  $\text{HCO}_3^-$ .
- Describe the adjustments in filtered load and  $\text{HCO}_3^-$  reabsorption ( $\text{H}^+$  secretion) by alterations in systemic acid-base balance and distinguish from factors that alter this process (i.e., ECF volume, aldosterone, and angiotensin II).
- Describe net acid excretion by the kidneys, titratable acid, the importance of urinary buffers, and the production and excretion of ammonium. Distinguish between the reclamation of filtered bicarbonate and the formation of new bicarbonate.

Day 6 Pp 563 – 578

Traditional Acid Base Balance

- Given a sudden increase or decrease in pH, identify the magnitude and the time course of the compensations that act to minimize change in pH of the body fluids, including a) buffers, b) respiratory adjustments, and c) renal adjustments.
- From blood values, identify simple and mixed metabolic and respiratory acid-base disturbances. Distinguish between increased and normal anion gap metabolic acidosis, chloride-sensitive and -resistant metabolic alkalosis, and acute and chronic respiratory disturbances.

4	<p>Day 7 Clinical Aspects of Electrolyte and Acid Base disorders</p> <p>Day 8 Notes &amp; Handout</p> <p>Non-traditional Acid Base Balance</p>	<p>Dr. Wolfshohl</p> <ul style="list-style-type: none"> <li>• Understand the limitations of the bicarbonate buffer system</li> <li>• Understand how the physiologic regulation of chloride, sodium, proteins and CO<sub>2</sub> impacts pH.</li> <li>• Describe the relationship between independent variables and dependent variables in non-traditional acid base regulation</li> <li>• Know the independent variables and understand physiologic control of these.</li> </ul>
5	<p>Day 9 Exam 1</p> <p>Day 10 Pp. 121 - 129, 656 – 662</p> <p>Introduction / General Principles of Endocrinology</p>	<ul style="list-style-type: none"> <li>• Define and describe the interactions between hormones, target cells, and receptors.</li> <li>• Compare and contrast hormone actions that are exerted through changes in gene expression with those exerted through changes in protein activity, such as through phosphorylation.</li> <li>• Contrast the signal transduction pathways involved in G-protein coupled receptors, receptor enzymes (i.e., tyrosine kinase), and ligand-gated ion channels.</li> <li>• Understand the effects of plasma hormone binding proteins on access of thyroid hormones and steroid hormones to their sites of action and degradation and on the regulation of hormone secretion.</li> <li>• Explain the effects of secretion, excretion, degradation, and volume of distribution on the concentration of a hormone in blood plasma. Explain the importance of patterns of hormone secretion, such as pulsatile, diurnal, and menstrual.</li> </ul>
6	<p>Day 11 Pp. 662 – 671, 679 - 682</p> <p>Hypothalamus &amp; Pineal; Neurohypophysis &amp; Adenohypophysis</p> <p>Day 12 Pp. 672 - 679</p> <p>Growth</p>	<ul style="list-style-type: none"> <li>• List the target organs and functional effects of oxytocin.</li> <li>• Name the stimuli for oxytocin release in relation to its reproductive and lactation functions.</li> <li>• List the target cells for vasopressin and explain why vasopressin is also known as antidiuretic hormone.</li> <li>• Describe the stimuli and mechanisms that control vasopressin secretion.</li> <li>• Identify disease states caused by a) over-secretion, and b) under-secretion of vasopressin and list the principle symptoms of each.</li> <li>• Describe the 3 major families of the anterior pituitary hormones and their biosynthetic and structural relationships.</li> <li>• Identify appropriate hypothalamic factors that control the secretion of each of the anterior pituitary hormones, and describe their route of transport from the hypothalamus to the anterior pituitary.</li> <li>• Understand negative feedback control of anterior pituitary hormone secretion at multiple levels.</li> <li>• Describe the relationship between growth hormone and the insulin-like growth factors and their binding proteins in the regulation of growth.</li> <li>• Understand the regulation of growth hormone secretion. Identify the roles of hypothalamic factors, glucose and IGF-I.</li> </ul>

- Identify the target organs or cell types for insulin-like growth factors that account for longitudinal growth.
- Describe the metabolic and growth promoting actions of growth hormone.

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Day 13 Pp. 686 - 692

Thyroid

- Identify the steps in the biosynthesis, storage, and secretion of tri-iodothyronine (T3) and thyroxine (T4) and their regulation.
- Describe the absorption, uptake, distribution, and excretion of iodide.
- Explain the importance of thyroid hormone binding in blood on free and total thyroid hormone levels.
- Understand the significance of the conversion of T4 to T3 and reverse T3 (rT3) in extra-thyroidal tissues.
- Describe the physiologic effects and mechanisms of action of thyroid hormones.
- Understand the causes and consequences of a) over-secretion and b) under-secretion of thyroid hormones. Explain what conditions can cause an enlargement of the thyroid gland.

Day 14 Pp. 692 - 704

Adrenal

- Identify the functional zones (one medullary and three cortical zones), innervation, and blood supply of the adrenal glands and the principal hormones secreted from each zone.
- Describe the biosynthesis of the adrenal steroid hormones (glucocorticoids, mineralocorticoids, and androgens) and the key structural features that distinguish each class.
- Understand the cellular mechanism of action of adrenal cortical hormones.
  - Identify the major physiological actions and therapeutic uses of glucocorticoids
  - Describe the components of the neuroendocrine axis that control glucocorticoid secretion.
  - Identify the causes and consequences of a) over secretion and b) under secretion of glucocorticoids and adrenal androgens
- List the major mineralocorticoids and identify their biological actions and target organs or tissues.
  - Understand the differential regulation of cortisol versus aldosterone release.
  - Describe the principal physiological stimuli that cause increased mineralocorticoid secretion. Relate these stimuli to regulation of sodium and potassium excretion
  - Identify the causes and consequences of a) over secretion and b) under secretion of mineralocorticoids
- Identify the chemical nature of catecholamines, their biosynthesis, mechanism of transport within the blood, and how they are degraded and removed from the body
  - Describe the biological consequences of activation of the adrenal medulla and identify the target organs or tissues for catecholamines along with the receptor subtype that mediates the response. Understand the mechanism by which epinephrine and norepinephrine can produce different effects in the same tissues.

		<ul style="list-style-type: none"> <li>○ Name the key stimuli causing catecholamine secretion. List the factors that can modulate a) the secretory response and b) the responses of target tissues</li> <li>○ Describe the interactions of adrenal medullary and cortical hormones in response to stress</li> <li>○ Identify disease states caused by an over secretion of adrenal catecholamines.</li> </ul>
8	<p>Day 15 Pp. 739 - 751, 760 – 761</p> <p>Gametogenesis &amp; Sexual Dimorphism</p> <p>Day 16 Exam 2</p>	<ul style="list-style-type: none"> <li>● Describe spermatogenesis and the role of Sertoli cells, Leydig cells and the basement membrane in this process.</li> <li>● Describe the endocrine regulation of testicular function: the role of the GnRH pulse generator, FSH, LH, testosterone, and inhibin.</li> <li>● Describe oogenesis and its relationship to changes in the ovarian follicle. Explain the roles of FSH, LH, estradiol, and inhibin in oogenesis and follicular maturation.</li> <li>● Describe ovulation and the formation and decline of the corpus luteum and the roles of hormones in each of these processes.</li> <li>● Compare and contrast the actions of testosterone, dihydrotestosterone, estradiol, and Müllerian inhibitory factor in the development of the male and female reproductive tracts.</li> <li>● Describe developmental changes in the male and female reproductive systems, including the mechanisms responsible for these changes, during <i>in utero</i> development, and in childhood through puberty.</li> </ul>
9	<p>Day 17 Pp. 736, 743 -757</p> <p>Male Reproduction</p> <p>Day 18 Pp. 757 - 773</p> <p>Female Reproduction</p>	<ul style="list-style-type: none"> <li>● Describe the physiological functions of the major components of the male reproductive tract.</li> <li>● Describe the, biosynthesis, mechanism of transport within the blood, metabolism and elimination of testosterone and related androgens.</li> <li>● List the major target organs and cell types for testosterone and other androgens.</li> <li>● Describe the actions and cellular mechanisms of testosterone and related androgens.</li> <li>● Describe the neural, vascular, and endocrine components of the erection and ejaculation response.</li> <li>● Identify the causes and consequences of over-secretion and under-secretion of testosterone for a) prepubertal and b) postpubescent males.</li> <li>● Understand aging- related changes in the hypothalamo-pituitary-gonadal axis that lead to puberty, reproductive maturity, and reproductive senescence (andropause).</li> <li>● List the major target organs and cell types for estrogen action and describe its effects on each.</li> <li>● Describe the actions and cellular mechanisms of estrogen.</li> <li>● List the principal physiological actions of progesterone, its major target organs and cell types, and describe its effects on each and the importance of “estrogen priming.”</li> <li>● Describe the actions and cellular mechanisms of progesterone and other progestins.</li> </ul>

			<ul style="list-style-type: none"> <li>Graphically illustrate the timing of changes in blood levels of FSH, LH, estradiol, progesterone, and inhibin, and correlate these with structural changes in the endometrium and the ovary seen during the menstrual cycle.</li> </ul>
10	Day 19	Pp. 757 - 773  Female Reproduction	<ul style="list-style-type: none"> <li>Describe how the changes in ovarian steroids produce the proliferative and secretory phases of the uterine endometrium and menstruation and the changes in basal body temperature during the menstrual cycle.</li> <li>Understand aging- related changes in the hypothalamo-pituitary-gonadal axis that lead to puberty, reproductive maturity, and reproductive senescence (menopause).</li> </ul>
	Day 20	Pp. 773 – 792  Pregnancy & Parturition	<ul style="list-style-type: none"> <li>Describe the process of fertilization, including capacitation and the acrosome reaction, and the movement of the blastocyst to the uterus.</li> <li>Describe the process of implantation.</li> <li>Describe the development and the major physiological functions of the placenta.</li> <li>List the protein hormones secreted by the placenta and describe the role of human chorionic gonadotropin (hCG) in the rescue of the corpus luteum in maintaining pregnancy early post-implantation.</li> <li>Describe the interactions between the placenta and the fetus in the pathway for production of estrogens during pregnancy.</li> <li>Discuss the roles of sex steroids, oxytocin, relaxin, and prostaglandins in the initiation and maintenance of parturition.</li> <li>Explain the role of hormones in mammary gland development during puberty, pregnancy, and lactation.</li> <li>Explain the basis for the inhibition of milk secretion during pregnancy and the initiation of lactation after parturition.</li> <li>Describe the neuroendocrine regulation of milk secretion and milk ejection.</li> <li>Explain the physiological basis of steroid hormone contraception.</li> <li>Understand other methods of contraception</li> </ul>
11	Day 21	Notes & Handouts  Reproductive Problems	<ul style="list-style-type: none"> <li>Describe common problems associated with the male reproductive tract and function, including erectile dysfunction, priapism, hypospadias, varicocele, Peyronie’s disease, retrograde ejaculation, etc.</li> <li>Understand anatomic and physiologic abnormalities of the female reproductive tract including virilization (androgenital syndrome), ovarian cyst formation, luteal cyst formation, retrograde uterus, endometriosis, failure of ovulation, etc.</li> <li>Discuss possible causes of failure of ovulation, conception, implantation, etc.</li> <li>Investigate current techniques to assist in fertility</li> </ul>
	Day 22	Exam 3	
12	Day 23	Pp. 581 – 632  Anatomical aspects of the digestive system and their	<ul style="list-style-type: none"> <li>Understand the integrated regulation (neural, endocrine, luminal) that drives digestion and absorption of nutrients after a meal and the temporal sequence of regulatory events during digestion.</li> </ul>

effect on control of digestion. Neural, hormonal, and enzymatic control of digestion

- Know the major excitatory and inhibitory motor neurotransmitters and major digestive hormones in the GI tract and how these biomediators affect function in GI tissues and cells.
- Identify the cell type and anatomical location of the endocrine cells secreting major GI hormones, such as gastrin, secretin, cholecystokinin (CCK), GLP-1, GLP-2, leptin, and motilin.
- Define the “incretin” concept, and as an example, describe the glucose-dependent release and action of an incretin from the gut.
- Understand how the physical and chemical compositions of luminal contents are sensed and the cellular and systemic responses to luminal stimuli.
- Describe the major anatomical characteristics of the enteric nervous system and the major cellular divisions of enteric ganglia (sensory nerves, interneurons, and motor neurons). Given either a cross section or whole mount of the bowel wall, identify the anatomical positions and major characteristics of the myenteric and submucosal plexi.
- Know how afferent and efferent extrinsic nerves (sympathetic and parasympathetic) interact with the enteric nervous system and regulate the functions of the GI tract.
- Describe the storage, digestion, and motility roles of the stomach.

Day 24 Pp. 581 – 632

Digestion

- Understand how the composition of gastric luminal fluid is affected by intake of a meal, as well as variable gastric secretions of acid, alkali, and attendant salts.
- Identify the proteins secreted into the gastric lumen by chief cells, parietal cells, and mucous cells. Contrast the functions and regulation of these secretions.
- Identify the gastric cell types secreting gastrin, somatostatin, histamine, and gastrin releasing peptide. Describe the stimuli that promote and inhibit release of these peptides, and their cellular targets.
- Describe the role of HCl in the gastric digestion of carbohydrates and protein, and how pepsinogen is activated.
- Describe the luminal pH of the stomach in the basal fasted state versus the time course of changes in luminal pH after a mixed meal.
- Describe how parietal cells H-K-ATPase activity can be inhibited physiologically and pharmacologically.
- List the stomach cell types and secreted substances that contribute to regulation of gastric acid secretion via paracrine, hormonal, and neuroendocrine pathways. Understand the integrated feedback regulation of acid secretion via these pathways during a meal.
- Describe the sequential digestion of ingested starch by enzymes of the salivary glands, pancreas, and the intestinal apical membrane.

## Digestion Continued

- Describe the sequential digestion of ingested proteins by gastric pepsin, pancreatic enzymes, and enzymes at the intestinal apical membrane. Make sure to include the role of duodenal enteropeptidase.
- Compare the membrane transport mechanisms responsible for uptake of sugars, amino acids and di-peptides by intestinal epithelial cells.
- Describe the mechanisms and molecules mediating the solubilization and digestion of lipids in the small intestine.
- Describe the mechanisms for the uptake, processing and release of lipids by the small intestinal epithelium and consequences of their malabsorption.
- Describe how liver blood flow and liver architecture impact liver function.
- List the water, ionic, bile salt, and bilirubin components of bile as secreted by the liver and after modification by the gallbladder.
- Describe the mechanisms whereby the gall bladder concentrates bile, and the endocrine mechanism stimulating gall bladder contraction and the secretion of bile through the sphincter of Oddi into the small intestine.
- Describe the amphipathic structure of bile salts, and describe how this property assists the solubilization and digestion of fats.
- Describe the enterohepatic circulation, including any different handling among primary and secondary bile salts, and bile acids.
- Describe the composition and formation of chylomicrons, their movement across the enterocyte basolateral membrane, and the route of entry into the cardiovascular system.
- Describe common causes of steatorrhea, and predict effects of steatorrhea on absorption of fat-soluble vitamins.

## Day 26 Notes &amp; Handouts

## Disorders

- Describe how dysfunction in the spatial or temporal characteristics of the esophageal pressure wave and/or sphincter relaxation can lead to swallowing defects and disorders such as heart burn, achalasia and aspiration of food.
- Describe the role of stomach functions in preventing pernicious anemia and peptic ulcer disease.
- List the mechanisms contributing to gastric mucosal defense and how they can be compromised by drugs or pathogens.
- Describe the function and dysfunction of gastric peristalsis, the pyloric sphincter, and duodenal feedback in controlling gastric emptying rate.
- Describe the causes of peptic ulcer disease.
- Relate the clinical characteristics of end-stage acute and chronic liver disease to the normal functions of the liver, and describe how fibrosis affects liver function.
- Describe the basis for studying liver enzymes in the circulation as a measure of liver injury.
- List the diseases of enzyme and transport deficiencies leading to osmotic diarrhea.
- Describe the role of short chain fatty acids in colonic sodium absorption and in both colonic and body energy metabolism.

14	Day 27 Pp. 635 – 653	Describe the related roles of fluid malabsorption in the small intestine versus colon on the potential to cause diarrheal disease.
	Energy Balance & Temperature Regulation	<ul style="list-style-type: none"> <li>• Diagram the thermal balance for the body, including heat production (metabolism, exercise, shivering) and heat loss (convection, conduction, radiation, and evaporation). Identify those mechanisms that shift from heat production to heat loss when environmental temperature exceeds bodycore temperature.</li> <li>• Define the thermoregulatory set point. Diagram the negative feedback control of body core temperature, including the role of the hypothalamic set point. <ul style="list-style-type: none"> <li>○ Contrast the stability of body core with that of skin temperature. Include the control and mechanisms of cutaneous blood flow and sweating on skin temperature.</li> <li>○ Identify the mechanisms for maintaining thermal balance in the following environments: desert (120°F), snow skiing (10°F), falling through ice into a lake (water temp 37°F), and snorkeling in 80°F water.</li> </ul> </li> <li>• Describe the role of appetite and metabolic rate in the maintenance of long-term energy balance and fat storage. Identify the factors that regulate appetite and fuel oxidation.</li> <li>• Identify the normal range of plasma glucose concentrations, and list the chemical forms and anatomical sites of storage pools for glucose and other metabolic substrates. <ul style="list-style-type: none"> <li>○ Identify the hormones that promote the influx and efflux of glucose, fat, and protein into and out of energy storage pools and their impact on the uptake of glucose by tissues. Establish specific roles for insulin, glucagon and catecholamines.</li> </ul> </li> <li>• List the major target organs or cell types for insulin, the major effects of insulin on each, and the consequent changes in concentration of blood constituents.</li> <li>• Identify the time course for the onset and duration for the biological actions of insulin.</li> <li>• Understand the relationship between blood glucose concentrations and insulin secretion.</li> <li>• Describe the roles of neural input and gastrointestinal hormones on insulin secretion. List the factors that modulate the secretory response.</li> <li>• Describe the control of glucagon secretion.</li> <li>• Identify disease states caused by: a) over-secretion, b) under-secretion of insulin, or c) decreased sensitivity to insulin, and describe the principal symptoms of each.</li> </ul>
	Day 28 Notes & Handouts Obesity	<ul style="list-style-type: none"> <li>• Understand the physiologic implications of obesity</li> <li>• Understand the economic and clinical impact of obesity</li> <li>• Identify some of the possible causes for obesity</li> </ul>
Finals	Final Exam	

VTPP 627  
Biomedical Physiology

- Recitation Goals:** To develop critical thinking, problem solving and scientific skills in preparation for a career in biomedical science. To gain experience in the evaluation of modern physiological principles discussed in lecture sessions. To reinforce physiological principles discussed in lecture sessions.
- Recitation Grading:** A total of 100 points are possible in the recitation portion of this course. Grades will be based upon review of 5 articles posted on eCampus. Review and discussion of the articles is worth 20 points each. Part of the review will involve asking and answering questions in a public format on eCampus. A rubric will be provided. Two weeks will be available for the reading of the article and answering questions.
- Article Selection:** Primary research articles focusing on area of understanding in physiology will be used.