



Australia's
Global
University

Faculty of Medicine
School of Medical Sciences

PHSL3221

Endocrine, Reproductive and Developmental Physiology

COURSE OUTLINE

TERM 3, 2020

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Please read this manual/outline in conjunction with the following pages on the

[School of Medical Sciences website:](#)

- [Advice for Students](#)
- [Learning Resources](#)

(or see "STUDENTS" tab at medicallsciences.med.unsw.edu.au)

1. COURSE STAFF

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The Department of Physiology in the School of Medical Sciences is located primarily on the 3rd floor of the Wallace Wurth building and is within the School of Medical Sciences, Faculty of Medicine.

Professor Gary Housley is Head of Department and appointments may be made directly with him (g.housley@unsw.edu.au or phone 9385 1057).

2. COURSE INFORMATION

a) General Introduction

Endocrine, Reproductive and Developmental Physiology is a 3rd year Science Course / Level III Physiology course usually undertaken upon successful completion of Physiology 1A (PHSL2101/2121/2501) and 1B (PHSL2201/2221/2502). It is worth six units of credit (6 UOC). The course usually forms part of a major in Physiology and/or Pharmacology in a Bachelor of Science or Bachelor of Medical Sciences degree.

This course has been developed with the aim of stimulating your interest and expanding your knowledge in the areas of endocrinology, reproduction, fertility and fetal development. The endocrine and reproductive physiology component builds on areas covered in Physiology 1B. The study of developmental physiology examines a wide range of organ systems and endocrine functions in the fetus, newborn and pregnant woman, and in this part of the course you will draw on your knowledge of these systems and processes from the relevant parts of Physiology 1A and 1B, and also your understanding of basic anatomy and biochemistry. The Level III Physiology subject most closely related to this course is Cardiovascular Physiology and Pathophysiology (PHSL3211).

The learning and teaching philosophy that underpins this course is our firm belief that a subject offered in the final term of your degree should not only develop a deeper understanding of physiology, but also foster the development of skills useful for your future career. All learning activities in the course are designed with this in mind.

b) Aims

This course aims to:

1. develop your understanding of the structure, function, control and pathophysiology of endocrine systems;
2. develop your understanding of the mechanisms associated with male and female reproduction and fertility;
3. provide you with an understanding of normal fetal growth and development, post-natal adaptation and survival, and maternal physiology;
4. develop your skills in teamwork, problem solving, communicating with peers, making presentations, independent learning, data analysis and report writing; and
5. stimulate an interest in and appreciation of biomedical research.

c) Science Graduate Attributes, UNSW

UNSW aims to provide an environment that fosters in you the following qualities, skills and attributes during your time here as a Science student:

Science Graduate Attributes, UNSW

- 1. Research, inquiry and analytical thinking abilities.**
Technical competence and discipline specific knowledge. Ability to construct new concepts or create new understanding through the process of enquiry, critical analysis, problem solving, research and inquiry.
- 2. Capability and motivation for intellectual development.**
Capacity for creativity, critical evaluation and entrepreneurship. Ability to take responsibility for and demonstrate commitment to their own learning, motivated by curiosity and an appreciation of the value of learning.
- 3. Ethical, Social and Professional Understanding.**
Ability to critically reflect upon broad ethical principles and codes of conduct in order to behave consistently with a personal respect and commitment to ethical practice and social responsibility. Understanding of responsibility to contribute to the community. Respect and value social, multicultural, cultural and personal diversity.
- 4. Communication.**
Effective and appropriate communication in both professional (intra and inter disciplinary) and social (local and international) contexts.
- 5. Teamwork, collaborative and management skills.**
Ability to recognise opportunities and contribute positively to collaborative scientific research, and to perceive the potential value of ideas towards practical applications. Demonstrate a capacity for self- management, teamwork, leadership and decision making based on open-mindedness, objectivity and reasoned analysis in order to achieve common goals and further the learning of themselves and others.
- 6. Information literacy.**
Ability to make appropriate and effective use of information and information technology relevant to their discipline.

The generic UNSW Graduate Capabilities can also be found at <https://teaching.unsw.edu.au/graduate-outcomes>

Endocrine, Reproductive and Developmental Physiology addresses each of these Science Graduate Attributes (SGA). Learning outcomes for the course, and the manner in which the course addresses the attributes, are outlined below.

d) Learning Outcomes

- Demonstrate an understanding of each of the three course themes:
 - function and control of endocrine systems
 - male and female reproduction and fertility
 - fetal growth, development and adaption to life after birth. [SGA 1]
- Contribute effectively in a group to discuss and offer solutions to a scientific or relevant clinical scenario. [SGA 1,2,4,5]
- Identify areas in your knowledge of physiology that could be improved, and carry out the self-directed learning necessary to “fill the gaps” [SGAs 1, 2, 6].

4. Research scientific information and communicate it to your colleagues and academic staff in written and oral format [SGAs 1, 2, 4, 6].

e) Teaching Strategies

A variety of teaching strategies are used in this course:

Lectures and Seminars introduce aspects of core material and insights into recent research and current practice. Until recently, the course convenor conducted research in fetal and developmental physiology. The course co-convenor conducts research in endocrinology and metabolism. We are also fortunate to have a large number of guest lecturers who are expert in their particular area of research or clinical practice. This means that you will gain an insight into both the basics and the latest issues relating to each of the course themes.

The **problem based learning tutorials** (PBLs) will form a large part of your study of endocrinology. These are designed not only to develop your knowledge of endocrine physiology [Learning outcome 1a], but also to encourage the development of self-directed learning, teamwork, and communication and presentation skills [Learning outcomes 2, 3, 4]. More information about PBL tutorials is given later in these notes.

Practical sessions and discussion classes are designed to give you a deeper understanding of particular aspects of the course. The practical class '*Gestational diabetes and screening in pregnancy*' enables you to carry out a glucose tolerance test, to learn more about gestational diabetes (a condition affecting about 10% of pregnant women in Australia) and to examine screening principles including sensitivity, specificity, positive predictive value and negative predictive value. You will consider the endocrine and renal control of circulating volume in the discussion class on '*Hormonal effects of water immersion*'. In '*Cross-dressing or crossing over*' you will consider sex determination in humans and the issue of intersex. The class "*Two peas in a pod*" enables you to consider twinning and aspects of paternity testing. During a *visit to the neonatal intensive care unit* at the Royal Hospital for Women, Randwick you will have a 'once in a lifetime' opportunity to see how our understanding of fetal and neonatal physiology is applied to treating preterm infants. **NOTE:** Because this course is being offered totally online for 2020, some activities have by necessity been modified. For example you will examine data from glucose tolerance tests (rather than carrying out the test yourself); a video has been made in the neonatal intensive care unit (as this site visit is not possible during the pandemic).

3. ASSESSMENT

Component	Mark allocation
Problem based learning	15%
Endocrine assignment	15%
Mid-term exam (Endocrine)	30%
Final exam (Repro, Fetal & Development)	40%
Total	100%

Details of assessment components and their rationale

The assessment components in this course are designed to help you to develop the skills outlined in the learning outcomes, as well as assessing your knowledge.

Problem Based Learning Classes. Your participation and presentations in three of the four problem based learning (PBL) classes contributes 15% to your final mark. A description of problem based learning and its assessment is included on the following pages.

Endocrine Assignment. This written report based on a case study in endocrinology will contribute to 15% of your final mark and should be submitted via Moodle by **9 am Monday of Week 9 (9/11/20)**. Details about this assignment are on pp.14-17. This exercise addresses the specific learning outcomes 1a, 3 and 4 (above). Please note that late submission of this assignment will incur a penalty.

Mid-term Exam. This exam is 90 min duration and will be held on **Friday 30th October (Week 7) at 10:15 am**, and covers all material relating to the Endocrinology component of the course, including the first 3 PBLs. The exam will consist of multiple choice and short answer questions.

Final Exam The final exam is 120 min duration and will be held in the official examination period. It will assess all material related to the Reproduction, Fetal and Developmental Physiology components of the course. The exam will consist of multiple choice and short answer questions.

Online formative assessment

Formative assessment questions are available online (via Moodle). These questions are multiple choice and are of a similar nature to those that will be in the summative exams. It is strongly recommended that you use these as a guide when studying for these exams and to provide feedback to help you learn.

What other feedback can I get to help my learning and to get the most out of this course?

This is a challenging course and the course convenor is very willing to help make this an interesting, satisfying way to end your 3rd year of studies. **Past exam questions** are given at the end of this outline, and you are encouraged to work through them to provide yourself with feedback on your progress. There will be a **practice exam questions and feedback session** before both the midterm and final exams. Following the exams, mark breakdown and feedback are provided on Moodle.

You are encouraged to **ask questions during lectures, seminars and discussion classes**. You will receive **feedback on your PBL participation and presentations** in the form of emailed comments and marks after both sessions of the first PBL, and you can also **ask your PBL facilitator for feedback** regarding your presentations and participation in discussions. You will receive feedback as well as marks for your assignment and presentations.

4. PROBLEM BASED LEARNING

a) Introduction

Problem based learning provides an opportunity for you, working in a group with others, to determine what you need to know in order to solve a given problem. A facilitator/tutor is present in the class and you are provided with information relating to a clinical problem. The role of the facilitator is to maintain and/or provide direction for the group discussion, but not to lead the discussion. Each group will have approximately 10-12 students. Guidelines for how individuals within the group should interact will be discussed and determined by group members with guidance from the facilitator. Each group will have a Discussion Forum on Moodle which only members of their group and their tutor can access.

Throughout the group discussions a scribe lists relevant information extracted from the information provided, and from the group discussion, under the following three headings:

- i. **Known Information:** A summary of the important facts related to the case.
- ii. **Hypotheses:** Possible hypotheses generated from the summarised information and the group discussion.
- iii. **Learning Objectives:** During the group discussion you set Learning Objectives, a list of topics/questions, which will require further investigation and later reporting to the group. This is the most important part of the exercise. At the end of the first session for each PBL case the facilitator divides the list of topics/questions among the group. Each student researches a learning topic and the following week presents the information they have researched to their group.

This entire process aims to help you not only improve your understanding of endocrine and reproductive physiology (Learning Outcomes 1 above) but also addresses outcomes 2, 3 and 4.

b) PBL presentations – how to minimise your group’s workload!

These PBL presentations will probably occupy the majority of the time away from class that you allocate to the first part of the course. You must keep in mind that you will come away from each PBL session with information from at least 9 other students. PBL content is assessed in the exam and so you need to make sure that you are providing each other with effective study materials. A big part of what makes a good presentation in this context (and this is included in the marking scheme, below) is conciseness. Think about how effective your handout will be as a study guide for the rest of the group. Once it is written, read it through and take out any unnecessary information. At the first PBL session, discuss with your group what rules you want to establish for giving presentations. These rules should be revised after the first round of presentations if necessary. Start with the following basics:

Basic rules for PBL presentations

A strict five minute time limit (shorter if possible – remember that questions take extra time and that you need to get through ~10 presentations in 90 minutes).

1. Limit each presentation to 4 slides (not including title slide and references).
2. Limit handouts to a maximum of one page of text (diagrams can be extra if necessary).
3. Handouts and presentation slides should be posted to your group’s PBL discussion forum prior to the relevant session.
4. A brief reference list is compulsory. Highlight any references you found particularly informative and which would be useful for the rest of the group to study from.

c) **Assessment Criteria for Problem Based Learning Classes**

There are two major components in the assessment of the PBL classes:

- 1) **Class interaction.** For these sessions to work well, all members of the group need to participate in the discussion **to the best of their ability**. The facilitator will assess individuals on their **participation** in the group discussion of the topic. This assessment will take into consideration the contribution of the individual to group dynamics e.g. politeness, fairness, respect for the opinions of others, genuine interest in the learning process. If you are not used to working in a group and find this process intimidating, remember that making an effective contribution to the group can be something as simple as taking the initiative to read the information sheet aloud for the rest of the group, or asking somebody to repeat something that you did not understand. This would be regarded as “participated in discussion voluntarily” (see marking scheme below).
- 2) **Reporting.** The second part of the assessment involves the reporting back and discussion of the Learning Objectives, which were allocated in the previous session. The emphasis of the assessment of this component is on how you present the information, and your ability to answer questions on your topic.

d) **Are all four PBLs assessed? How will feedback be given?**

The first PBL is to be used to become familiar with the process of problem based learning and to get to know your group. After this PBL, your tutor will send you your assessment via email along with feedback regarding your participation and presentation. **This mark will not contribute to your final assessment.** The remaining 3 PBLs will be formally assessed and we encourage you to use the feedback from your tutor after the first PBL to improve your participation and presentation skills.

e) **How is problem-based learning assessed in the exam?**

You are not expected to have an intricate knowledge of all of the material covered during each PBL class for the midsession exam. However, you should be able to demonstrate a broad understanding of the learning objectives outlined in each PBL, and be able to describe the physiology underlying each PBL case and the test results. In keeping with this, assessment of problem based learning in the exam will be largely by short answer questions, allowing you to demonstrate a broad understanding of the area, rather than by MCQs, which tend to assess specific aspects of your knowledge. Examples of questions relating to PBL classes in past exams are given at the end of this guide. MCQ questions in the formative assessment and practice exam on topics covered by the PBLs should also guide your learning.

Marking scheme:

Class Interaction – Assessed by facilitator during session 1 of PBLs 1-4

Standard	Mark (out of 5)	Required Performance
Very Poor	0-1	- no participation in class discussion; not obviously listening to other group members
Poor	2	- minimal participation; only participated in response to direct questioning
Adequate	3	- participated in discussion voluntarily;
Good	4	- voluntarily contributed to the group discussion; provided insightful comments or questions
Very Good	5	- major role in group discussion without dominating the group and still allowing other members of the group to contribute

Reporting – Presentation assessed by facilitator during session 2 of PBLs 1-4

Standard	Mark (out of 10)	Required Performance
Very Poor	0-2	- no research or preparation on allocated topic
Poor	3-4	- inadequate research on the allocated topic - explanation unclear or contains major errors
Adequate	5-7	- adequate research on the topic - mainly accurate information provided, although some errors noted - failure to comply with time limit, slide or handout requirements eg provided too much information, did not load presentation ahead.
Good	8-9	- topic researched thoroughly - information explained clearly, accurately and concisely - complied with time limit, slide and handout requirements - good understanding of topic and able to answer questions - able to relate their topic to the whole PBL
Very Good	10	- topic researched thoroughly - information explained clearly, accurately and concisely - information presented in an interesting or novel way - complied with time limit, slide and handout requirements - thorough understanding of topic and able to answer questions - able to relate their topic to the whole PBL

5. COURSE SCHEDULE AND ATTENDANCE REQUIREMENTS

The course timetable is attached at the end of these notes and can also be found on Moodle. All lectures and seminars will be recorded, but you are expected to attend online live events for their full duration. We will let you know ahead which sessions will be live, but in general this will be the Thursday afternoon seminars (4pm-6pm) and the Friday morning practical classes (10am-1pm). Attendance for activities scheduled in the practical class times are particularly important as these will not be recorded.

Problem based learning tutorials. PBLs form a major part of your learning for the Endocrinology and Reproductive components of this course. You are relying on other members of your group to attend all sessions, carry out the necessary research and report back to the group, and they are relying on you to do the same. For both of these reasons attendance at all PBL sessions is compulsory. Non-attendance for other than documented medical or other serious reasons, or unsatisfactory performance, will result in an additional assessment exam or in ineligibility to pass the course.

6. RESOURCES FOR STUDENTS

a) Textbooks

There are no prescribed texts for this course. '*Ganong's Review of Medical Physiology*' provides a very good coverage for the endocrine component of the course, while Harding & Bocking '*Fetal Growth and Development*' is an excellent reference for developmental physiology. Blackburn's '*Maternal, Fetal & Neonatal Physiology*' is useful for the reproduction and developmental components of the course. The others are more specialist textbooks which are held in print in the UNSW library or can be accessed online through the UNSW library catalogue or the links below and could be consulted as a reference if necessary. **(You may need to log in with your zpass)**

- Barrett KE, Barman SM, Brooks HL & Yuan JXJ. *Ganong's Review of Medical Physiology*. 26th edition, 2019. Lange. (Note: it is fine to use an earlier edition eg 23rd Edition onwards).
<https://accessmedicine.mhmedical.com/book.aspx?bookid=2525>
- Blackburn ST. *Maternal, Fetal & Neonatal Physiology*. 4th Edition, 2013. Elsevier.
<https://ebookcentral.proquest.com/lib/unsw/detail.action?docID=2072142>
- Gardner DG & Shoback D. *Greenspan's Basic & Clinical Endocrinology*. 10th edition. 2018, Lange.
<https://accessmedicine.mhmedical.com/book.aspx?bookid=2178>
- Harding, R and Bocking, AD. *Fetal Growth and Development*. 2001, Cambridge UP.
https://primoa.library.unsw.edu.au/primo-explore/fulldisplay?docid=UNSW_ALMA21119024850001731&context=L&vid=UNSW&lang=en_US&search_scope=SearchFirst&adaptor=Local%20Search%20Engine&tab=default_tab&query=any,contains,Fetal%20Growth%20and%20Development&offset=0
- Holt RIG & Hanley, NA. *Essential Endocrinology and Diabetes*. 6th Edition. Wiley-Blackwell, 2012.
<https://ebookcentral.proquest.com/lib/unsw/detail.action?docID=822511>
- Kovacs WJ & Ojeda, SR. *Textbook of Endocrine Physiology*. 6th Edition, Oxford UP, 2012.
<https://ebookcentral.proquest.com/lib/unsw/detail.action?docID=845972>

b) Other Resources

- The learning activities may involve supplementary reference articles and printed lecture notes.
- For the PBLs you may find Harrison's online (a medical database, the online version of Harrison's Principles of Internal Medicine) and the Oxford Textbook of Medicine (electronic resource) useful resources. These can be accessed via the UNSW library catalogue.
- Hammer GD & McPhee, SJ. *Pathophysiology of Disease: An Introduction to Clinical Medicine*. 8th Edition, 2019. Lange. <https://accessmedicine.mhmedical.com/book.aspx?bookId=2468>
This is likely to be helpful for the PBLs and the Endocrine Assignment
- Moodle: Lecture notes, course-related material such as timetables and outlines, as well as supplementary articles will be placed on Moodle. Marks for assessment tasks will also be posted here. Announcements will be made via Moodle and it is your responsibility to regularly check this site.
- All lectures are taped by UNSW Lecture Recordings + and can be accessed via UNSW Moodle.

See also: [Learning Resources](#) on the SoMS website.

7. CONTINUAL COURSE IMPROVEMENT

a) MyExperience

Changes are continuously being made to this course to keep it current and to make it a worthwhile experience for you. UNSW introduced MyExperience for student feedback in all courses starting from Semester 1 2017, and this tool will be used in this course. Your feedback is taken seriously, and the improvements that are made to the course are based in part on such feedback. The current course represents the outcome of many years of student feedback. In previous years many students made the comment that they wanted more feedback to help their learning. In response this, the number of opportunities for students to get feedback has been increased. These include: PBL facilitators email students individually to provide feedback on their presentations and class participation after the first PBL, which can be used to guide the remaining three PBLs; feedback on the Endocrine assignment is provided via the marking rubric and the individual assignments are annotated by the marker in Moodle; two formative assessment tools have been produced which contain a number of multiple choice questions and feedback on the answers; there is a tutorial dedicated to practice exam questions and feedback before both the midsession and final exams. Last year, with the reduction in the duration of the course due to the introduction of trimesters, students indicated that the assessment load was too high. Therefore, this year we have removed the group presentation component (15% of the mark for 2019) and adjusted the other components. The weighting for the PBLs has been increased to 15% as students felt that the previous weighting of 10% was too low for the amount of work involved. Another concern raised last year was the lack of choice in the exam questions. This year, there will be some choices in the short answer questions in both the midterm and final exams.

b) Student panel

While individual students are welcome to provide feedback to the course convenor, your views regarding the course can also be put forward by a small panel of student representatives. These representatives will have the opportunity to meet online with the course convenor during session to provide feedback on the course structure, learning activities and staff. This will enable you to make your views known while the course is running, as opposed to at the end of the course (which is a disadvantage of MyExperience evaluation).

8. GENERAL INFORMATION

Note: further advice on SoMS website:

<https://medicallsciences.med.unsw.edu.au/students/undergraduate/advice-students>

SCHOOL OF MEDICAL SCIENCES HONOURS PROGRAM

There is an Honours program conducted by the School. This program is coordinated by A/Prof Cristan Herbert (c.herbert@unsw.edu.au 9385 8679). Further information can be obtained from the SoMS website or the honours program co-ordinator.

POSTGRADUATE RESEARCH DEGREES

The Department of Physiology offers students the opportunity to pursue a Doctorate (PhD) in Physiology. For further information is available on the "Students" menu of the SoMS website.

SPECIAL CONSIDERATION

You can apply for special consideration when illness or other circumstances beyond your control, interfere with your assessment performance. Failure to sit an exam or submit an assessment without applying for Special Consideration will lead to automatic failure of the test or assessment. See

<https://student.unsw.edu.au/special-consideration>

Note that if you miss an exam and are granted special consideration, if appropriate a supplementary exam will be offered. These will be held in the Supplementary exam period for Term 3, 2020 which is 11 January to 15 January, 2021.

STUDENT SUPPORT SERVICES

For all students, there are some useful links to help you with your online learning.

- Transitioning to Online Learning <https://www.covid19studyonline.unsw.edu.au/>
- Guide to Online Study <https://student.unsw.edu.au/online-study>
- UNSW Student Life Online <https://student.unsw.edu.au/hub#main-content>
- Student support and development <https://student.unsw.edu.au/support>

Those students who have a disability that requires some adjustment in their teaching or learning environment are encouraged to discuss their study needs with the course convenor prior to, or at the commencement of their course, or with the Disability Advisor in the Equitable Learning Services unit (formerly Disability Support Services) (8374 9201 or <https://student.unsw.edu.au/els>). Issues to be discussed may include access to materials, signers or note-takers, the provision of services and additional exam and assessment arrangements. Early notification is essential to enable any necessary adjustments to be made.

The UNSW Learning and Career Hub offers workshops throughout the academic year on a wide variety of Academic and Career Development skills. These include referencing, writing skills, critical thinking, exam preparation and time management. Individual assistance is available on request. Further information can be obtained using the link <https://student.unsw.edu.au/skills>

9. ENDOCRINE ASSIGNMENT

Task Description

Each of the 3 cases below describes a patient with an endocrine disorder. Choose **one** case and write a report about the patient's endocrine disorder.

In your report you should include:

- (1) a description of the biosynthesis of the main hormone(s) involved (4 marks)
- (2) a description of the normal mechanisms which control secretion of the main hormone(s) involved (4 marks)
- (3) an explanation of the mechanisms underlying each of the patient's symptoms, signs and test results (8 marks)
- (4) a brief explanation of possible causes/etiology of the disease (3 marks)
- (5) a brief explanation of possible treatments (3 marks).

Your report should be 2000 words (excluding references, figures and tables), and be properly referenced with in text references and a reference list. Guidelines for referencing are provided on p17.

You must submit your assignment electronically via Turnitin Moodle by the due date and time. Please include in the file name of your assignment which case (1, 2 or 3) you have chosen. As well you must include the word count on the front cover sheet. You will only be able to submit your assignment once you have completed the Student Declaration form.

Due date: 9 am Monday 9th November (Week 9).

A penalty of 10% per day after the deadline will be applied for late submission.

Assessment Criteria

You will be assessed on:

- the scientific content of your report
- how well you have communicated your ideas (use of clear, simple, grammatical language; clear explanations; logical structure; appropriate language; effective use of illustrations where appropriate)
- evidence of critical thinking (discussion of inconsistencies in the literature; use of logical argument)
- whether your report is appropriately referenced
- your choice of sources (range, quality, relevance).

Your assignment will be marked out of 25. Please see the specific marking criteria available on Moodle. Note that in addition to the 22 marks which are allocated to the content areas described above, 3 marks are allocated for communication and referencing. Marks will be deducted for exceeding the word limit.

Questions related to the assignment can be posted on the relevant Moodle discussion board.

Note: for this assignment, it is not a requirement to use original research sources, although you should ensure that you choose reliable resources. The resources listed on p.11-12 of this course outline would be suitable sources to start with. In general, you should not be referencing lecture slides. Also, avoid referencing websites of questionable authority.

CASE 1

A 20 year old woman presents to her doctor with increased urine production. Beginning about a month previously she had noticed that she was waking up a couple of times a night to pass urine. More recently she noticed that she was also passing urine more frequently during the day, sometimes as often as once an hour.

Her mother had suggested that this increased urine production might have been due to her high caffeine consumption. However, for the past week she had limited herself to one cup of coffee per day, but still found that her urinary frequency continued. In addition she found that she was always thirsty. She had started carrying a large water bottle with her and re-filling it several times a day. She had also noticed that the urine she passed was almost colourless, rather than yellow.

On physical examination the doctor found no abnormalities.

Blood and urine tests were ordered which showed:

Plasma sodium concentration 149 mmol/L (reference range 136-145 mmol/l)

Plasma osmolality 308 mOsm/kg (reference range 285-295 mOsm/kg)

Fasting plasma glucose 5 mmol/l (reference range 4.2-6.4 mmol/l)

Urinary osmolality 200 mOsm/kg.

Urinary glucose - negative.

Further questioning revealed that no other family members had ever displayed these symptoms. There was no history of traumatic head injury. An MRI of her brain was normal.

A two-hour water deprivation test was performed. After two hours of not being able to drink water, the osmolality of her plasma and urine were measured a second time. Her urinary osmolality remained at ~ 200 mOsm/kg, but her plasma osmolality increased to 315 mOsm/kg. She was then injected with a drug called DDAVP. One hour after the injection, the osmolality of her plasma decreased to 290 mOsm/kg and the osmolality of her urine increased to 425 mOsm/kg.

The diagnosis of idiopathic pituitary diabetes insipidus was made.

CASE 2

A 60 year old man with a history of hypertension and non-insulin dependent diabetes mellitus, presented to his doctor because, although normally an active man, he was finding that his muscles were weak and he was tiring more easily. On examination, his diastolic pressure was more elevated than usual but no other abnormalities were detected. A blood test was ordered which indicated that his potassium level was 2.8 mmol/l (reference range 3.3-4.7 mmol/l).

As the man was taking frusemide (a potassium-wasting diuretic drug) to treat his hypertension, it was initially considered that this drug was the likely cause of the hypokalaemia. Consequently, frusemide was discontinued, and the man was commenced on oral potassium supplementation.

A week later, the man had further blood tests which showed:

Plasma potassium 2.7 mmol/l (reference range 3.3-4.7 mmol/l)

Plasma sodium 144 mmol/l (reference range 137-145 mmol/l)

Plasma chloride, magnesium, bicarbonate and pH were all within normal limits.

Red blood cell and white cell counts were normal.

In view of the persistent hypokalaemia, several hormone assays were performed with blood sampled at 9.30 am, after he had been seated for one hour.

Upright plasma aldosterone concentration 38 ng/dl (reference range 4-31 ng/dl)

Upright plasma renin activity - 0.4 ng/ml/h

Plasma aldosterone concentration to plasma renin activity ratio – 95 ng/dl per ng/ml/h (a ratio > 30 is strongly suggestive of autonomy of aldosterone secretion).

Primary hyperaldosteronism was confirmed by an acute intravenous isotonic saline load test. Pre and post infusion aldosterone levels were 35 and 17 ng/dl, respectively (maximum acceptable level after infusion = 5 ng/dl).

A high resolution abdominal CT scan was performed which demonstrated enlargement of the left adrenal gland and one cortical nodule approximately 1.5 cm in diameter. A diagnosis of Conn's syndrome was made and the patient was scheduled for surgery.

CASE 3

A 37-year-old man presented to the emergency department with a five-week history of nausea, vomiting and weakness. He said that he did not weigh himself routinely, but he felt that he had lost weight because his clothes were looser and he had needed to get an extra hole in his belt so that his jeans did not fall down. He had seen his general practitioner on a couple of occasions for symptoms of fatigue, nausea and anorexia. Because of the gastrointestinal nature of his symptoms, he had been treated with cimetidine and antacids, without improvement. More recently he found he was craving salt.

On physical examination, the man looked unwell. His blood pressure was 100/47 mmHg while he was lying down but fell to 70/30 mmHg when he stood up. There were areas of blue/black discolouration on his gums, and he appeared to be tanned in patches over pressure areas on his knees and elbows. Physical examination was otherwise normal.

Laboratory results were as follows:

Plasma potassium 5.8 mmol/l (reference range 3.3-4.7 mmol/l)

Plasma sodium 127 mmol/l (reference range (136-145 mmol/l)

Morning cortisol level 69 nmol/L (reference range 138 to 635 nmol/L).

Plasma ACTH level was 54.8 pmol/L (reference range 2.0 to 11.5 pmol/L).

A screening test for adrenal function was performed.

Plasma cortisol levels at 30, 60 and 90 minutes after injection of cosyntropin (250 µg) were 72 nmol/L, 74 nmol/L and 55 nmol/L respectively.

At 90 minutes, the plasma aldosterone level was less than 28 pmol/L (the normal incremental increase is >111 pmol/L).

A CT scan showed a severe reduction in the size of the adrenal glands bilaterally. His chest X ray was normal, except that the heart size seemed rather small. The tuberculin skin test was negative. His blood contained autoantibodies against 21 hydroxylase.

A diagnosis of Addison's disease was made and steroid replacement therapy with hydrocortisone and fludrocortisone acetate was begun.

Guidelines for referencing

These guidelines have been adapted from the School of Medical Sciences Honours Manuscript – Instructions to Authors.

Note: for this assignment, it is not a requirement to use original research sources, although you should ensure that you choose reliable resources. Examples of reliable resources – peer-reviewed journal articles, online textbooks. Non-reliable web resources – Wikipedia, healthdirect, mayoclinic, patient.info, etc.

In the text, references to other work should take the form: (Bolton and Kitamura, 1983) or 'Bolton and Kitamura (1983) showed that...' When a paper written by two authors is cited, both names are given; for three or more authors only the first name is given, followed by 'et al.'

The reference list at the end of the manuscript must be arranged alphabetically according to the surname of the first author. When the names of first authors are identical, the alphabetical order of the surnames of subsequent authors takes precedence over the year of publication. The authors' names are followed by the year of publication in brackets. If more than one paper by the same authors in one year is cited, a, b, c, etc. are placed after the year of publication, both in the text and in the list of references. All authors should be quoted for papers with up to seven authors; for papers with more than seven authors, the first six should be quoted followed by *et al.*

The format for references to papers and books, and to chapters in books, is as follows:

Lipp P, Egger M & Niggli E (2002). Spatial characteristics of sarcoplasmic reticulum Ca²⁺ release events triggered by L-type Ca²⁺ current and Na⁺ current in guinea-pig cardiac myocytes. *J Physiol* **542**, 383-393.

Adrian ED (1932). *The Mechanism of Nervous Action*. Humphrey Milford, London.

Buchan AMJ, Bryant MG, Polak JM, Gregor M, Ghatei MA & Bloom SR (1981). Development of regulatory peptides in the human fetal intestine. In *Gut Hormones*, 2nd edn, ed. Bloom SR & Polak JM, pp. 119-124. Churchill Livingstone, Edinburgh.

For those articles published online ahead of print, that have not been assigned full publication details the DOI (digital object identifier) should be used. See example below:

Dickerson MT, Dadi PK, Butterworth RB, Nakhe AY, Graff SM, Zaborska KE *et al.* (2020). Tetraspanin-7 regulation of L-type voltage-dependent calcium channels controls pancreatic β -cell insulin secretion. *J Physiol*; DOI: 10.1113/JP279941

10. PAST EXAMINATION SHORT ANSWER QUESTIONS (last 5 years)

MIDSESSION EXAMINATION, SEPTEMBER 2015

Question 1. (15 minutes)

The following test results were taken from a 5 year old child with a history of precocious sexual development:

	Patient level	Normal level
<u>Plasma:</u>		
Cortisol	30 nmol/L	280-550 (morning)
11-deoxycortisol	20 µg/100 mL	<0.1 µg/100 mL
ACTH	500 pg/mL	30-120 pg/mL (morning)
Aldosterone	3 ng/100 mL	5-20 ng/100 mL
<u>Urine:</u>		
17-ketosteroids	15-20 mg/24h	<0.5 mg/24h
Tetrahydrocompound S	1 mg/h	<0.5 mg/h

- (i) What is the likely diagnosis?
- (ii) Explain why each of the above test results might be abnormal.
- (iii) Why does this child have precocious sexual development?
- (iv) What treatment would you recommend and why?

Question 2.(15 minutes).

A 12 year old girl, Elise, has been brought unconscious into the emergency department by her concerned mother. She says that Elise had been complaining of feeling unwell for the past few days. She had been drinking lots of water and kept having to urinate. Blood tests, renal function tests and a physical examination are carried out.

Provide a **very brief** explanation for each of the results below, using only the space provided. The table continues over the page.

	Patient result	Normal result	Brief explanation
Blood glucose concentration	70 mmol/L	<8 mmol/L	
Glucosuria	++++	-	
Urine flow rate	30 mL/min	0.3 – 15 mL/min	
Ketonuria	++++	-	
Arterial pH	6.95	7.35-7.45	

*****The remainder of this question has not been released *****

Question 3. (5 minutes)

Clomiphene is used in the treatment of infertility. Describe the mechanism of action of this drug and what type of infertility can be treated with it.

Question 4. (5 minutes)

Explain how it is possible for an individual to have 46XY karyotype but not be phenotypically male.

Question 5. (5 minutes)

Write notes on ONE anorexigenic and ONE orexigenic gut hormone.

In your answer include (i) the site of their synthesis, (ii) the stimulation for their release and, (iii) the signaling mechanism through which these hormones act on hypothalamic pathways for appetite control.

Question 6. (5 minutes)

What is a pheochromocytoma? Why is a 24 h urine collection used to make the diagnosis? What is measured in this collection?

Question 7. (5 minutes)

Paul, 26 years old, undergoes fertility assessment with the following results:

Gynaecomastia

Testicular volume: 11 mL (reference range: 15-30mL)

Semen analysis: Azoospermia (i.e. no sperm detected)

Urinary Testosterone:Epitestosterone ratio: 10.1 (normal range: 0.1-3.99)

Subsequent discussions with the doctor revealed that Paul, an amateur athlete, had been self-administering synthetic testosterone esters for the past two years. Briefly explain why the abnormal findings described above could be caused by his testosterone use.

Question 8. (5 minutes)

Describe the production, regulation and effects of parathyroid hormone.

FINAL EXAMINATION, NOVEMBER 2015

Question 1. (15 minutes)

- Name three shunts present in the fetal circulation. For each shunt indicate where it is located and describe its function.
- Describe other ways in which the fetal cardiovascular system (both the heart and the circulation) differ from that of the adult.

Question 2. (15 minutes)

Jenny is a 30 year-old woman who is 8 weeks pregnant with her first child. At her first antenatal visit she expresses concern that her friend had gestational diabetes during her pregnancy and needed to have insulin injections. Jenny doesn't like needles and hopes she won't need injections as well.

- What is gestational diabetes and why does it develop during pregnancy?
- How is gestational diabetes usually diagnosed, and at what stage of gestation?
- Why is it important to treat gestational diabetes?
- Indicate 6 risk factors which increase the likelihood of women developing gestational diabetes.
- What are the long-term consequences for a mother with gestational diabetes?

Question 3. (5 minutes)

Compare and contrast andropause and menopause.

Question 4. (5 minutes)

Describe the fluid fluxes into and out of the amniotic cavity in the second half of gestation.

Question 5. (5 minutes)

“Human chorionic gonadotropin is the most important hormone in early pregnancy.” Discuss.

Question 6. (5 minutes)

What are fetal breathing movements? Briefly describe the major factors that control them.

Question 7. (5 minutes)

Describe the production of oxytocin and its role in human labour.

Question 8. (5 minutes)

What is epigenetics? Describe how epigenetics relates to the “Barker hypothesis”.

MIDSESSION EXAMINATION, SEPTEMBER 2016

Question 1. (15 minutes)

There are many variants of congenital adrenal hyperplasia (CAH). Cameron Jones had 11 β -hydroxylase deficiency and was hypertensive. On the other hand, 21 β -hydroxylase deficiency is associated with excessive loss of salt in the urine (‘salt wasting’).

- (i) Explain the mechanism underlying hypertension in 11 β -hydroxylase deficiency.
- (ii) Explain why salt wasting occurs with 21 β -hydroxylase deficiency.
- (iii) Cameron Jones was treated with cortisone. Explain the underlying basis for this treatment.
- (iv) Why might an infant born with CAH have ambiguous genitalia?

Question 2.(15 minutes).

A 12 year old girl, Elise, has been brought unconscious into the emergency department by her concerned mother. She says that Elise had been complaining of feeling unwell for the past few days. She had been drinking lots of water and kept having to urinate. Blood tests, renal function tests and a physical examination are carried out.

Provide a **very brief** explanation for each of the results below, using only the space provided. The table continues over the page. See Table 2015.

Question 3. (5 minutes)

Describe

- (i) how the hormones found in combination oral contraceptives alter hypothalamic-pituitary function, and
- (ii) how this prevents conception.

Question 4. (5 minutes)

- (i) What are Tanner stages? Which features are examined (a) in boys and (b) in girls to assess Tanner stages? What range of values can be assigned to these features?
- (ii) What are the normal age ranges for the onset of puberty (a) in boys and (b) in girls?
- (iii) List two differences in body composition between boys and girls which become apparent by the end of puberty.

Question 5. (5 minutes)

Explain what is meant by the incretin effect. Give an example of two forms of incretin-based treatment for Type 2 diabetes.

Question 6. (5 minutes)

Describe the hormonal effects of head-out water immersion. What are the mechanisms that underlie these hormonal changes?

Question 7. (5 minutes)

Describe the production and actions of vitamin D.

Question 8. (5 minutes)

Describe the hormonal changes that occur in the hypothalamic-pituitary-ovarian axis as a result of menopause. Your answer should indicate why these changes occur.

FINAL EXAMINATION, NOVEMBER 2016

Question 1. (15 minutes)

- (a) Describe the fetal response to an acute fall in oxygen availability.
- (b) List and briefly describe 4 methods that have been used in animal studies to induce fetal hypoxia.

Question 2. (15 minutes)

Describe the maternal changes that occur during pregnancy in

- (a) blood volume and composition
- (b) the cardiovascular system

Question 3. (5 minutes)

Describe the role of the prepartum cortisol surge in fetal maturation.

Question 4. (5 minutes)

Describe the human placenta. Explain how placental transfer increases in late gestation even though placental weight remains fairly constant.

Question 5. (5 minutes)

“It doesn’t matter if the fetal kidneys don’t function *in utero*, so long as they function after birth”. Discuss.

Question 6. (5 minutes)

Suckling by the infant causes elevation of maternal plasma levels of two hormones important in lactation. Complete the following table for these two hormones.

Hormone Name		
Chemical structure		
Site of synthesis		
Site of release		
Role in lactation		
Other function		

Question 7. (5 minutes)

Briefly describe the changes that occur in the neonate’s cardiovascular system after birth.

Question 8. (5 minutes)

What do you understand by the term “fetal programming”? Include examples in your answer.

MIDSESSION EXAMINATION, SEPTEMBER 2017

Question 1. (15 minutes)

Physical examination and blood and urine tests taken from a child with a history of precocious sexual development revealed the following:

<u>Examination</u>	Patient level	Normal level
Height	130 cm	95 th Percentile = 125 cm
Weight	33 kg	95 th percentile = 27 kg
Blood pressure	150/90	~100/70
<u>Plasma:</u>		
Cortisol	30 nmol/L	280-550 (morning)
11-deoxycortisol	20 µg/100 mL	<0.1 µg/100 mL
ACTH	500 pg/mL	30-120 pg/mL (morning)
Aldosterone	3 ng/100 mL	5-20 ng/100 mL
<u>Urine:</u>		
17-ketosteroids	15-20 mg/24h	<0.5 mg/24h
Tetrahydrocompound S	1 mg/h	<0.5 mg/h

- What is the likely diagnosis?
- Explain why each of the above findings might be abnormal.
- Why was this child given glucocorticoid treatment?

Question 2.(15 minutes).

A 12 year old girl, Elise, has been brought unconscious into the emergency department by her concerned mother. She says that Elise had been complaining of feeling unwell for the past few days. She had been drinking lots of water and kept having to urinate. Blood tests, renal function tests and a physical examination are carried out.

Provide a **very brief** explanation for each of the results below, using only the space provided. The table continues over the page. See Table 2015.

Question 3. (5 minutes)

- Which hormone(s) is/are used in male hormonal contraceptives?
- Describe how the hypothalamic-pituitary-gonadal axis is modulated by the hormone(s) to control male fertility.

Question 4. (5 minutes)

What factors influence the timing of puberty and what is the normal age of onset?

Question 5. (5 minutes)

Briefly explain why glucagon-like peptide-1 (GLP-1) is called "ileal brake and incretin gut hormone".

Question 6. (5 minutes)

What are hot flushes and why do they occur in menopausal women? What factors trigger them in affected women?

Question 7. (5 minutes)

Describe the production and actions of vitamin D.

Question 8. (5 minutes)

What are the clinical features of carcinoid tumour and what are they due to? When a 24h urine sample is collected to make the diagnosis, what foods should the patient avoid?

FINAL EXAMINATION, NOVEMBER 2017

Question 1. (15 minutes)

“The fetus is a miniature adult”. Discuss with reference to the cardiovascular system and one other organ system.

Question 2. (15 minutes)

Explain the mechanisms underlying each of the following conditions which occur commonly in pregnant women:

- (a) appetite stimulation
- (b) supine hypotension
- (c) warm hands and feet even in cold weather
- (d) ankle swelling
- (e) anaemia
- (f) glycosuria
- (g) increased risk of thrombosis

Question 3. (5 minutes)

- (a) List the main fluxes of fluid into and out of the amniotic cavity during fetal life.
- (b) What is likely to happen to amniotic fluid volume if fetal swallowing is impaired?

Question 4. (5 minutes)

Why does the newborn, especially the premature newborn, have difficulty maintaining body temperature?

Question 5. (5 minutes)

Explain why removal of the maternal ovaries would cause abortion of a human pregnancy at 5 weeks LMP, but not at 12 weeks LMP.

Question 6. (5 minutes)

Briefly describe the adaptations that enable fetal survival, despite an arterial oxygen tension that is only about 20% of healthy adult values.

Question 7. (5 minutes)

Lisa, a mother who is breast feeding her 6 month old baby, is concerned that her periods have not yet returned. Her friend Joanna, who delivered her baby only 4 months previously, has already had two normal periods. Joanna is bottle feeding her baby. Use your knowledge of the physiology of lactation to explain Lisa's amenorrhea.

Question 8. (5 minutes)

What is epigenetics? Describe why an altered epigenetic state is proposed to be the mechanism that explains the “DOHaD hypothesis”. Give examples.

MIDSESSION EXAMINATION, SEPTEMBER 2018

Question 1. (15 minutes)

The following blood and urine test results were taken from a 5 year old child with a history of precocious sexual development:

	Patient level	Normal level
<u>Plasma:</u>		
Cortisol	30 nmol/L	280-550 (morning)
11-deoxycortisol	20 µg/100 mL	<0.1 µg/100 mL
ACTH	500 pg/mL	30-120 pg/mL (morning)
Aldosterone	3 ng/100 mL	5-20 ng/100 mL
<u>Urine:</u>		
17-ketosteroids	15-20 mg/24h	<0.5 mg/24h
Tetrahydrocompound S	1 mg/h	<0.5 mg/h

- (i) What is the likely diagnosis?
- (ii) Explain why each of the above blood and urine results might be abnormal.
- (iii) Would you expect this child to have normal blood pressure? Why/why not?
- (iv) What treatment would you recommend and why?

Question 2. (15 minutes).

A 12 year old girl, Elise, has been brought unconscious into the emergency department by her concerned mother. She says that Elise had been complaining of feeling unwell for the past few days. She had been drinking lots of water and kept having to urinate. Blood tests, renal function tests and a physical examination are carried out.

Provide a **very brief** explanation for each of the results below, using only the space provided. The table continues over the page. See Table 2015.

Question 3. (5 minutes)

Define what is meant by the term 'insulin resistance'. List 3 factors that cause insulin resistance and describe how insulin resistance is involved in the pathogenesis of type 2 diabetes.

Question 4. (5 minutes)

An athlete self-injects synthetic testosterone for two months prior to a competition and is asked to provide a urine sample for testing. Describe two methods which could be used to detect this performance enhancing steroid use, providing an explanation of the underlying mechanisms.

Question 5. (5 minutes)

Name a gut hormone that is involved in the regulation of appetite and indicate whether it has a stimulatory or inhibitory effect. List four unique features of gut hormones that distinguish them from other hormones.

Question 6. (5 minutes)

A patient is seen by his doctor because of recurrent headaches. He also complains of episodes of palpitations during which he looks very pale and feels uneasy. On examination he is hypertensive (blood pressure 190/100). Describe how these clinical features (underlined) are consistent with a pheochromocytoma. Indicate two ways to confirm the diagnosis.

Question 7. (5 minutes)

How is it possible for an individual to have a 46XY karyotype but not be phenotypically male?

Question 8. (5 minutes)

Describe the effects of growth hormone. How do the effects of excessive growth hormone secretion differ according to whether the patient is a child or an adult. What are the names of these two conditions?

FINAL EXAMINATION, NOVEMBER 2018

Question 1. (15 minutes)

- (a) Name three shunts present in the fetal circulation. For each shunt indicate where it is located and describe its function.
- (b) Describe other ways in which the fetal cardiovascular system (both the heart and the circulation) differ from that of the adult.

Question 2. (15 minutes)

(a) Suckling by the infant causes elevation of maternal plasma levels of two hormones important in lactation. Complete the following table for these two hormones.

Hormone Name		
Chemical structure		
Site of synthesis		
Site of release		
Role in lactation		
Other function		

(b) Why do breastfeeding women experience amenorrhea of a longer duration after delivery than women who formula-feed their infant?

(c) Briefly discuss the reliability of lactation as a method of contraception.

(d) If a lactating woman decides to go on the oral contraceptive pill, what type of pill should she be prescribed and why?

Question 3. (5 minutes)

Compare and contrast menopause and andropause.

Question 4. (5 minutes)

What is gestational diabetes mellitus? Why is it important that all pregnant women are screened for this condition and what are the adverse consequences if it is not adequately treated?

Question 5. (5 minutes)

What is meant by the term “myometrial activation”? Describe the changes that occur during myometrial activation. What is responsible for myometrial activation?

Question 6. (5 minutes)

Describe the composition, formation and importance of lung liquid.

Question 7. (5 minutes)

Fetal growth can be influenced by fetal, maternal and placental factors. Briefly describe how these factors affect fetal growth, giving specific examples.

Question 8. (5 minutes)

Briefly define epigenetics. Describe why an altered epigenetic state is proposed to be the mechanism that explains the “DOHaD hypothesis”. Give an example of an epigenetic change and its mechanism of action.

MIDSESSION EXAMINATION, SEPTEMBER 2019

Question 1. (15 minutes/15 marks)

There are many variants of congenital adrenal hyperplasia (CAH). Cameron Jones had 11 β -hydroxylase deficiency and was hypertensive. On the other hand, 21 β -hydroxylase deficiency is associated with excessive loss of salt in the urine (‘salt wasting’).

(a) Explain the mechanism underlying hypertension in 11 β -hydroxylase deficiency. (3 marks)

(b) Explain why salt wasting occurs with 21 β -hydroxylase deficiency. (3 marks)

- (c) What would you expect a blood test for ACTH to show in untreated CAH? Why? (2 marks)
- (d) Cameron Jones was treated with cortisone. Explain the underlying basis for this treatment. (3 marks)
- (e) Why might a newborn infant with CAH have ambiguous genitalia? (2 marks)
- (f) Would you expect a newborn male infant (XY karyotype) with CAH to have ambiguous genitalia? Justify your answer. (2 marks)

Question 2. (15 minutes/15 marks).

A 12 year old girl, Elise, has been brought unconscious into the emergency department by her concerned mother. She says that Elise had been complaining of feeling unwell for the past few days. She had been drinking lots of water and kept having to urinate. Blood tests, renal function tests and a physical examination are carried out.

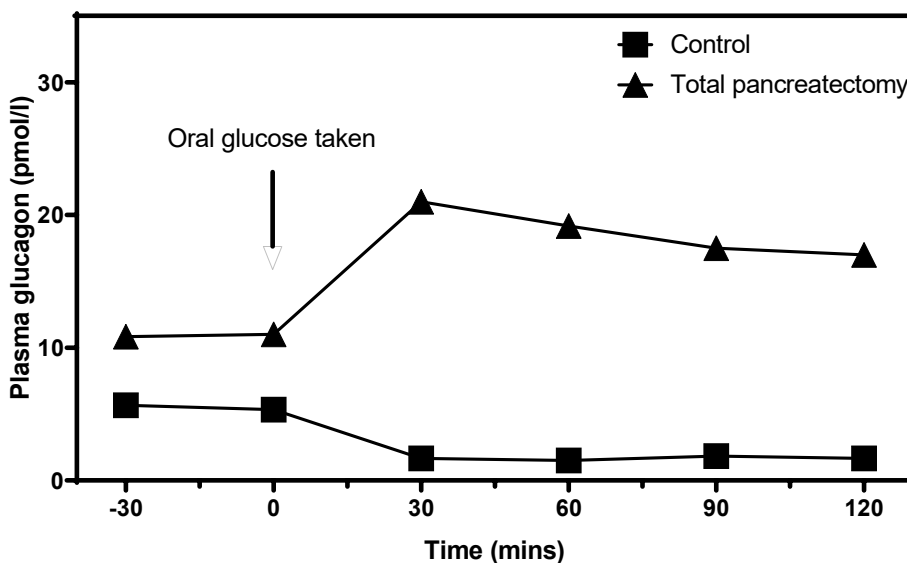
Provide a **very brief** explanation for each of the results below, using only the space provided. The table continues over the page. See Table 2015.

Question 3. (5 minutes/5 marks)

- (a) Explain why head out water immersion causes an increase in urine flow rate. (4 marks)
- (b) List two other manoeuvres which have a similar effect. (1 mark)

Question 4. (5 minutes)

A clinical research team was studying glucose metabolism in patients who had undergone a total pancreatectomy (removal of the pancreas due to adenocarcinoma; n=8), versus age and weight-matched control subjects (n=8). As expected, patients in the total pancreatectomy group showed impaired glucose metabolism following a standard oral glucose tolerance test (75 grams of glucose), due in part to insulin deficiency. However, they had some unexpected results during this test, and the research team would like your insight as an experimental physiologist.



- (a) What conclusions can you make from this data? (4 marks)
- (b) What is the likely source of glucagon in the total pancreatectomy group? (1 mark)

Question 5. (5 minutes/5 marks)

- (a) What is the incretin effect? (2 marks)
- (b) Briefly describe the mechanism by which sitagliptin exerts its hypoglycaemic effect, making it appropriate for treatment in type 2 diabetes mellitus. (3 marks)

Question 6. (5 minutes/5 marks)

- (a) Describe the sources and production of vitamin D in humans. (3 marks)
- (b) Indicate four groups of people at risk of vitamin D deficiency. (2 marks)

Question 7. (5 minutes/5 marks)

Compare and contrast precocious puberty and precocious pseudopuberty. In your answer include two examples of each condition.

Question 8. (5 minutes/5 marks)

A recreational athlete has been injecting himself with a synthetic testosterone over the last 5 years. Describe what potential side-effects he could have developed and provide an explanation of the mechanisms for 2 of the most common side-effects.

FINAL EXAMINATION, NOVEMBER 2019

Question 1. (15 minutes/15 marks)

Describe the maternal changes that occur during pregnancy in

- (a) the cardiovascular system
- (b) blood volume and composition.

Question 2. (15 minutes/15 marks)

- (a) Describe the fluid fluxes into and out of the amniotic cavity in the second half of gestation. (8 marks)
- (b) List the functions of amniotic fluid. (4 marks)
- (c) What is polyhydramnios? How is it detected? What problems may result from polyhydramnios? (3 marks)

Question 3. (5 minutes/5 marks)

The fetus consumes twice as much oxygen per kg body weight as the adult, and yet the P_{O_2} of fetal blood is low compared with the adult. Describe how the fetus manages to survive and grow in a low oxygen environment.

Question 4. (5 minutes/5 marks)

Describe the human placenta. In what ways does the sheep placenta differ from that of the human?

Question 5. (5 minutes/5 marks)

Describe the production of oxytocin and its role in human labour.

Question 6. (5 minutes/5 marks)

- (a) How long is gestation in humans and how are “pre-term” and “post-term” defined? (2 marks)
- (b) List 6 **important** risk factors that predispose for premature labour. (3 marks)

Question 7. (5 minutes/5 marks)

Describe the changes that occur in the neonate’s cardiovascular system in the first few hours after birth.

Question 8. (5 minutes/5 marks)

Describe what “developmental programming” is and how altered nutrition can lead to programming.

11. TIMETABLE 2020

PHSL3221 Endocrine, Reproductive and Developmental Physiology

Lectures: Tuesdays 10 am - 12 pm
 Seminars: Thursdays 4 pm - 6 pm
 Pracs/PBLs: Fridays 10 am - 1 pm

WEEK 1			
Tuesday 15/9	10 am	Course information and assessment requirements	Dr K Gibson
Tuesday 15/9	11 am	Concepts in endocrinology	Dr K Gibson
Thursday 17/9	4 pm	Insulin action and nutrient metabolism	Prof N Turner
Thursday 17/9	5 pm	The role of glucagon in metabolism and disease	Dr G Smith
Friday 18/9	10 am	PBL 1.1	PBL tutors
WEEK 2			
Tuesday 22/9	10 am	Gut Hormones	Dr L Liu
Tuesday 22/9	11 am	Hypothalamic regulation of body weight	Dr K Ip
Thursday 24/9	4 pm	Growth	Dr K Gibson
Thursday 24/9	5 pm	Hormonal effects of water immersion	Dr K Gibson
Friday 25/9	10 am	PBL 1.2/PBL2.1	PBL tutors
WEEK 3			
Tuesday 29/9	10 am	Endocrine functions of white adipose tissue	Dr A Brandon
Tuesday 29/9	11 am	Calcium metabolism	Dr K Gibson
Thursday 1/10	4 pm	Update on the renal angiotensin system	Dr K Gibson
Thursday 1/10	5 pm	Hypersecretion: catecholamines and serotonin	Dr K Gibson
Friday 2/10	10 am	PBL 2.2/PBL 3.1	PBL tutors
WEEK 4			
Tuesday 6/10	10 am	Oocyte development and maturation	Dr Rodrigues Paris
Tuesday 6/10	11 am	Androgens and anabolic steroids	Dr V Birzniece
Thursday 8/10	4 pm	Puberty	Dr K Gibson
Thursday 8/10	5 pm	Cross-dressing or crossing over?	Dr K Gibson
Friday 9/10	10 am	PBL 3.2/PBL 4.1	PBL tutors
WEEK 5			
Tuesday 13/10	10 am	Regulation of Fertility	Dr A Finch
Tuesday 13/10	11 am	Fertility and Assisted reproductive Technology	Dr A Clark
Thursday 15/10	4 pm	Menopause and Andropause	Dr K Gibson
Thursday 15/10	5 pm	Practice exam questions and feedback	Dr K Gibson
Friday 16/10	10 am	PBL 4.2	PBL tutors
WEEK 6			
Flexi week		No classes	

WEEK 7			
Tuesday 27/10	10 am	Introduction to Fetal Physiology	Dr K Gibson
Tuesday 27/10	11 am	Maternal physiology	Dr K Gibson
Thursday 29/10	4 pm	Fetal circulation	Dr K Gibson
Thursday 29/10	5 pm	Fetal response to hypoxia	Dr K Gibson
Friday 30/10	10 am	Midsession Exam	KG, VB
WEEK 8			
Tuesday 3/11	10 am	Placenta A	Dr K Gibson
Tuesday 3/11	11 am	"Ghost in your genes" - Moodle	
Thursday 5/11	4 pm	Developmental Origins of Health and Disease	Dr C Maloney
Thursday 5/11	5 pm	Epigenetics	Dr C Maloney
Friday 6/11	10 am	Gestational Diabetes & Screening in pregnancy	KG, VB
WEEK 9			
Monday 9/11	9am	<i>Endocrine assignment Due (Submit in Moodle)</i>	
Tuesday 10/11	10 am	Placenta B	Dr K Gibson
Tuesday 10/11	11 am	Fetal endocrinology	Dr K Gibson
Thursday 12/11	4 pm	Fetal breathing	-
Thursday 12/11	5 pm	Regulation of fetal fluids	Dr K Gibson
Friday 13/11	10 am	Two peas in a pod	Dr K Gibson
WEEK 10			
Tuesday 17/11	10 am	Parturition	Dr K Gibson
Tuesday 17/11	11 am	Adaptation to life after birth	Dr K Gibson
Thursday 19/11	4 pm	Lactation and early infant nutrition	Dr K Gibson
Thursday 19/11	5 pm	Practice exam questions and feedback	Dr K Gibson
Friday 20/11	10 am	Neonatal Intensive Care /Neonatal nursery	Dr K Lui/VB

PBL tutors

Dr Vita Birzniece
Amrutha Patkunarajah
Nathan Luque
Mallory Luke
Deepak Sharma