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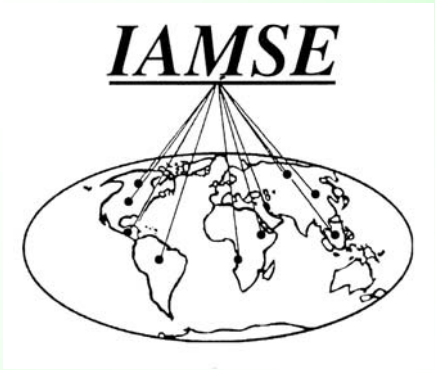

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Message from Editor-in-Chief

Uldis N. Streips, Ph.D.
Editor-in-Chief

Welcome to issue 19-3 of the JIAMSE. As we have done all 2009, this issue again has a variety of contributions from articles to innovative ideas, and also a medical education case. I hope this provides some ideas for you to contribute articles of your own, or to include the ideas into your own curriculum. I strongly encourage you do educational research. There is a lot of satisfaction to be gained and also your work can provide a reason for you to attend the national meeting of IAMSE (New Orleans next year!). I have also found that the educational research I do translates directly into improving the medical course I run at my school and the curriculum of our school. So, the benefits are multitudinous. I look forward to hearing from you.

All best,

Uldis N. Streips, Ph.D.
Editor-in-Chief, JIAMSE

INNOVATION

Technology Enhanced Scripted Scenario: A Method for Running Multiple Small Groups Simultaneously

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This innovation addresses two major difficulties related to case-based small-group learning: (1) the faculty-intensive nature of small-group learning, and (2) the inconsistencies in experiences between groups. This paper describes a method used at the DeBusk College of Osteopathic Medicine (DCOM) to run 15 small groups simultaneously using PowerPoint and the TurningPoint audience response system.[1] Technology Enhanced Scripted Scenario (TESS) cases rely on an extensive script developed by a team of faculty. Student groups take a patient history, simulate a physical exam, make a differential diagnosis, order tests and labs, make a final diagnosis and develop a treatment plan. At each step, students within a group come to a decision, and then there is a discussion between the groups until the class as a whole comes to a consensus. Using the audience response system, the student groups can be graded on each of these tasks. A unique aspect of TESS cases is that the script and PowerPoint presentation for each case has multiple pathways. The actual path followed depends on the diagnosis and treatment plan decided upon by the class. Running clinical cases using the TESS format has several benefits. A small number of faculty can facilitate multiple student groups simultaneously, making cases less faculty intensive. Since a small team of facilitators runs all the groups, the experience is the same for all students. This format also prevents facilitators from using small groups as additional lecture time. TESS cases help students develop good history-taking habits before beginning rotations. Students also get practice making a differential diagnosis, ordering appropriate labs & tests, making a diagnosis, and developing a treatment plan. Cases are more realistic, with plot-lines that follow the student decisions, and results that arrive at the appropriate point in the case. Feedback comes to the students from the case itself, not from a faculty facilitator. Analysis of student responses allows assessment of the student decision-making process, which is not easily assessed in lecture exams. Student performance can provide indications of holes in the curriculum and can guide curricular development. TESS cases can also be used to deliver cases to third and fourth year students at their rotation sites.

We have run four TESS cases for first and second year osteopathic medical students at DCOM. Student feedback was very positive, and faculty commented that the students were much more engaged than in small groups led by individual facilitators. In running these cases, it became obvious that students needed much more experience in obtaining a patient history. It was also interesting that students had no problem diagnosing a case with striking findings (e.g. aspiration pneumonia) but found it much more difficult to diagnose a more routine, less striking case (e.g. exacerbation of COPD). Results from the TESS cases are being used by systems coordinators to modify their curricula.

[1]The facilitator's room is connected to the small group rooms via a Tandberg 6000 MXP codec. Each small group room contains a Tandberg 550 MXP codec connected to a 42 inch monitor. Similar audio and video connectivity could be achieved via the internet using web conferencing software such as Adobe Acrobat Connect Pro.

INNOVATION

Professional Responsibility in Medical Practice: Online Course Pilot Study

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Professional responsibility in medicine has been increasingly recognized as a key target for raising the quality of health care provided around the world. The Professional Responsibility in Medical Error and Transparency (PRMET) course, funded through the Center for Professionalism in Business and Society, was developed to facilitate the creation of safe and effective health care systems through the continued development of physicians dedicated to the reduction of medical error and increased system transparency. Curricular content was developed to emphasize training in the major fields of patient safety, quality improvement, communication/collaboration, error science, process improvement, and ethical principles in clinical practice as it relates to medical error. This online course is intended for use in undergraduate and/or graduate medical education and was designed on the principles of collaborative learning, constructivism, and active participation, as well as sharing of thoughts and problem solving.

The PRMET course consisted of six (6) online modules, requiring approximately 5 hours to complete each module for a total of 30 hours. The modules were developed to significantly advance learners knowledge and skill related to professional responsibility, transparency and error science using a variety of approaches including patient case studies, problem-based learning, research and knowledge building, and evidence-based practice. The course was piloted twice over a two week period Winter 2009 with 30 fourth year students. Individual activities during the completion of each module included self-study, reading, and written responses to discussion questions in each module. Program evaluation utilized course logs, pre/post surveys and focus groups for the purposes of a) assessment of student achievement as compared to the learning objectives for the program; b) examination of learner and faculty satisfaction with the course, module, and teaching methods; and c) provision of feedback for course revision. Overall, students responded favorably to both the content and online, interactive delivery of the course, and students demonstrated an improvement in self-efficacy for professional responsibility related to patient safety and quality improvement. Recommendations for course revision included increased case-based discussions and a reduction of required readings. Course revision and publication will be completed after final data analysis.

INNOVATION

Use of Internet Based Chat in a "Remote Live Standardized Patient" Skills Training

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Clinical skills assessment exams using Standardized patients (SPs) are an established component of medical school education, including the current USLME Step 2 CS exam™. The authors have developed a novel method for conducting SP encounters using web-based chat technology (e.g. Google® chat). "Remote Live Standardized Patient" (RLSP) interviews are conducted entirely online. The interview requires active learner participation focused on clinical skills, and represents a unique solution to teaching specific skills and affecting behavior. We are not aware of other educators using chat for this purpose.

We have been pilot testing chat-based OSCE-style exams[1,2] since 2007. In 2009 we have begun using RLSPs to *teach* skills, as well as evaluate them. The instructional RLSP key components include an interactive SP actor, an EMR like interface, and a "Hats Off" mode, which allows the student learner to "ask a preceptor" or research information during the encounter. Each learner's performance is evaluated by standardized measures and by the RLSP actor feedback. This interactive, responsive web environment allows the RLSP interview to simulate a broad spectrum of learner/patient encounters, from initial screening through diagnosis and treatment.

Advantages include 1) decreased SP training cost and time commitment, 2) more flexible SP scheduling and recruiting since all encounters occur online, 3) elimination of face-to-face constraints of verisimilitude errors and correlation of actor physical appearance compared to the "patient", and 4) instant availability of chat conversation transcripts for review and learner feedback. Potential limitations include missing the "whole person" experience afforded by face-to-face encounters, including the ability to assess body language and demonstrate physical exam skills. Since face-to-face SP encounters also suffer from realism limitations (i.e. the actor does not typically have the "patient's" presenting symptoms), we feel that the advantages of an RLSP balance and actually outweigh the drawbacks.

Use of web-based chat or its equivalent may have significant appeal to the current generation of Internet-savvy medical students. The RLSP experience appears to be a viable alternative to the traditional face-to-face SP interview, allows cost savings, and prepares students for Step 2 CS-style exams.

ACKNOWLEDGEMENTS AND DISCLOSURE:

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2. Metcalf, M.P., Tanner, T.B., Wilhelm, S.E., and Buchanan, A. Use of a Remote Standardized Patient to Teach Clinical Skills to Undergraduate Medical Students. Poster presented at the *2009 AMIA Spring Congress*, May 28-30, 2009, Orlando, FL.

COMMENTARY

The Basic Science Learning Station: An Innovative Kinesthetic Learning Approach in one Medical School

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ABSTRACT

Students have variable and sometimes multimodal learning preferences that include kinesthetic (i.e. hands-on or tactile) learning. Yet the pre-clerkship medical school curriculum does not emphasize kinesthetic learning of basic science, particularly content in modern genetics, cell biology, immunology, pharmacology and nutrition. A new instructional strategy to enhance kinesthetic learning of basic science is the Basic Science Learning Station. At the Learning Station, students experience weekly self-guided activities which include the physical manipulation of games, puzzles and conceptual models. The Learning Station modules provide opportunities for students to integrate basic science information, reinforce concepts presented in other formats (i.e. lecture or case-based), and provide motivation and enthusiasm for basic science learning. Furthermore, the Learning Station kinesthetic strategy provides a new curricular structure in which basic science faculty can design ways to help students integrate clinical and basic science content domains. Our students report they enjoyed the hands-on approach to learning.

INTRODUCTION

The teaching of the basic science foundation in medical school has historically been divided into discipline-specific departments and courses, such as anatomy, physiology, pharmacology, etc. With the emergence of understanding in cellular and molecular-based disciplines (i.e. genetics, cell biology, immunology, pharmacology and nutrition), the expansive basic science "foundation" of medical care has been progressively squeezed into the same two-year duration of training. In an effort to integrate the traditional "silos" of information, pre-clerkship medical school curricula have also evolved, leading to cross and inter-disciplinary courses such as "Brain, Mind, Behavior"; "Metabolism and Nutrition"; and "Infection, Inflammation, and Immunity."[1]

This revised curricular structure may have achieved some degree of success integrating basic science content. But concurrently there has been an expansion of clinical experiences earlier in the pre-clerkship basic science

coursework. Consequently, there has been a progressive decline in the time devoted to basic science laboratory or hands-on experiences (e.g. dissection of cadavers, experiments involving laboratory animals, etc.). In some cases, the hands-on laboratory component of the curriculum has been largely replaced by computer simulation or eliminated altogether.[2,3] Even those laboratory sessions that still remain often prioritize visual or analytical skills with fewer and fewer hands-on approaches that rely on instructor-led demonstrations, prosections, sample patient-volunteers, or passive observations by the target learners.[2, 3]

It is these twin forces of content explosion and passive-learning methods that have diminished the hands-on basic science curriculum in many medical schools including our own small experimental program called the UC Berkeley – UCSF Joint Medical Program (JMP). The JMP enrolls 12-16 medical students per year who complete pre-clerkship years at UC Berkeley before going on to UCSF for the clerkship years. The JMP curriculum distributes the usual two-year pre-clerkship curriculum across three years because the students simultaneously pursue a master's degree in health sciences during this time. In 2002, the JMP consolidated the entire pre-clerkship basic and clinical course structure into a comprehensive Problem-Based Learning (PBL) curriculum consisting of a series of paper-based clinical cases.

In order to address the need for hands-on basic science learning, we created a new curricular concept called the Basic Science Learning Station. Medical students attended this 'Learning Station' as a parallel activity to supplement the paper cases in the first and second years of our pre-clerkship curriculum. We developed sixteen independent modules to help students integrate and learn basic science information. In this article, we describe the creation and development of the Learning Station, and provide data regarding student evaluations of the Learning Station experience.

NEW APPROACH

Beginning in 2003, we set up the Learning Station in our program during the fall semesters and report here our experiences for the first five years of its existence. The first and second-year classes of medical students (n = 24 – 32 students) were invited to voluntarily participate in the Learning Station. The weekly module at the station was presented as an optional un-graded formative activity, but attendance was encouraged by faculty. Each weekly module was designed to overlap or provide enrichment to the basic science areas related to the corresponding PBL case for that particular week. The modules were presented to first and second-year students on alternate weeks, resulting in an average of 8 learning stations per cohort per academic year. The Learning Station was available to all students for four business days per week between the hours of 8am – 5pm.

The Learning Station was designed to be a 'dry lab' physically situated in a 7'x12' cubicle with surrounding wallboard in an open suite of adjoining offices. Furniture was simple and consisted of a 24" depth L-shaped 36" high table with approximately 12 linear feet of table space and rolling chairs. Permanent equipment included a small refrigerator, microscope, slide viewer and microcomputer.

Each weekly module was arranged as a progression of 4-7 activities, consisting of items such as original hand-made models, games, puzzles, foods, containers and objects placed on the table top. An instruction booklet facilitated students through these self-guided, self-paced activities (see Table 1 for descriptions of sample Learning Station activities). The students were invited to touch or manipulate the objects in order to illustrate a scientific concept or facilitate their performance of a simple experiment. In addition, approximately 50% of the station modules contained a histology tutorial component consisting of 10-12 annotated light and electron photomicrographs (on computer, slide carousel or hard copy), as well as one glass slide for hands-on viewing with the microscope. Students could work solo, in pairs or small groups as desired. Each specific module was designed to be completed in approximately 10-20 minutes.

Many of the activities at the station involved the hands-on manipulation of objects or models which were constructed in advance using very simple materials such as paper, plastic, wood, glass containers or everyday objects. These models represented abstract molecular processes, conceptual information, or dynamic cellular activities which students performed by touching or altering the objects (see Figure 1 for examples). Students were also asked to touch hand-made puzzles or play board games comprised of tissue or organ drawings as representations (see Figure 2 for examples). Nutritional items such as vitamin supplements, food labels, pharmaceutical containers and real foods or facsimiles were also displayed.

To evaluate the Learning Station, students were required to fill out an electronic questionnaire each week, regardless of their attendance at individual stations. The questionnaire included open-ended questions, as well as items with 5-point Likert scales. One question asked respondents whether the level of complexity of the learning station was appropriate, and response options ranged from "strongly disagree" to "strongly agree." A separate question asked about the station effectiveness, and response options ranged from "poor" to "excellent." If students did not participate in individual stations, they were encouraged to answer "not applicable" to the questionnaire items.

PILOT DATA

Even though the station was a completely optional activity, roughly 3 out of 4 students regularly attended the station modules. Since 2003, the percent of students who

Table 1: Sample Learning Station modules and examples of hands-on activities

Title of module	Sample Activities
Obesity	• Touch a flip-book with obesity statistics from the US Food & Drug Administration (FDA). • Touch & examine models of 3-dimensional adipocytes. • Use a recipe to make a soft drink.
Catecholamines and the adrenal medulla	• Touch sample molecules and draw the biosynthetic pathway of catecholamines.
Cells, casts and crystals	• Manipulate tools of urinalysis such as a urinometer. • Touch models of crystals. • Manipulate molecules to form urea from an amino acid.
Capillaries are swell	• Manipulate a model with veins at different heights, and perform a simple experiment of osmotic pressure. • Touch models of different types of capillaries. • Examine daily salt quantities and foods with various salt content.
Statins	• Touch centrifuge tubes with various lipoprotein bands. • Touch drug samples of statins.
Life of a plasma cell	• Play a game to track the anatomic origins and differentiation process of plasma cells.
Sex determination	• Manipulate 5-alpha-reductase enzyme activity.
Life of a red blood cell	• Touch models of poikilocytosis in erythrocytes. • Touch models of splenic tissue.
Body fluid pH	• Perform pH measurements on various fluids.
Life of a myocardial cell	• Touch models of cardiac myocytes and connect their intercellular junctions. • Play a game with calcium ions, including extracellular entry, SR release and binding to troponin C on the thin filament.
Identifying leukocytes	• Draw progressively more complex leukocyte structures in order to understand their differences and similarities. • Feel 3-D representations of leukocytes. • Roll leukocytes along a capillary wall to find receptors.
Amino acids: the essentials	• Manipulate amino acids to create a peptide bond and the influence of different amino acid side groups. • Group foods with varying amino acid content.
Vasopressin or ADH?	• View different urine concentrations. • Perform labstix urinalysis. • Manipulate vasopressin receptor and G protein signal transduction in principal cells of collecting tubules.

interacted with the station ranged annually from 57 – 77% attendance. The 2007-08 academic year showed the lowest student participation (i.e. 57% of the students filled out evaluations) although it is not clear to us why this unanticipated drop occurred. The aggregate of student responses to the Likert questions in the evaluation questionnaire across all learning stations is provided as Table 2. Although not robustly designed to be an educational research study, these summary statistics provide some evidence in support of the educational value of the station.

A sampling of student responses to open-ended questions is provided in Table 3. Many students clearly valued the learning station as part of their learning process in this case-based curriculum. In describing their thoughts, the word 'love' appeared many times over, indicating how passionately some students felt about this experience. Although far less frequent, students also offered

constructive criticisms or neutral comments about the station which could be roughly broken down into three main categories: 1) insufficient time in their schedules to complete the station, 2) desire for a different complexity of material (either more or less), and 3) specific suggestions to improve the station by creating specific new hands-on models or materials.

Figure 1: Sample hands-on activities at the Learning Station

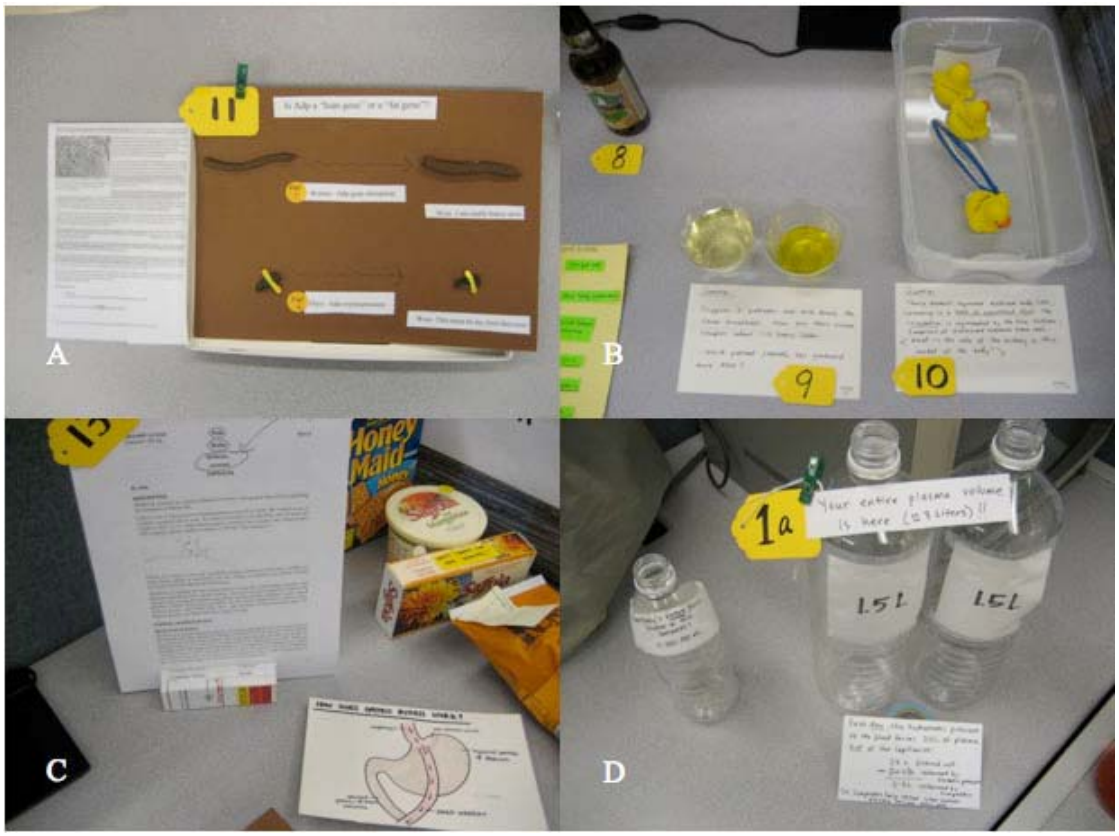

Figure 1. **A**. Obesity (touch clay models of transgenic and overexpression experiments in obese animal models). **B**. Vasopressin or ADH (fake urines; urinometer). **C**. Obesity (handle foods and read nutritional labeling of fat content, gastric bypass game board). **D**. Capillaries are Swell (observe and handle containers with entire plasma volume of adults and neonate).

LIMITATIONS

There are several limitations to our analyses. First, this effort was not robustly designed to be a scholarly evaluation or education research study. We did not, for example, include a matched cohort of students in PBL without the Learning Station opportunity. Our success in establishing the learning station may well have been influenced by our small size (n=16 students average per year), and include a variety of factors inherent in a small class per se. For instance, collective learning is a common thread and the activities that are enjoyed tend to be passed along to the group through amplification. We may also attract certain students that might be either over-or under-represented in certain ways in a larger program where there is more anonymity. Our reputation as an experimental program might skew our enrollment towards those who prefer atypical or novel approaches to learning, especially our extensive PBL curriculum. And since this was a voluntary activity, another limitation is the variable response rate for Learning Station participation and evaluation each year. Some students completed the Learning Station without evaluating the activity, while other students may have completed the evaluation without participating in the Learning Station. [4]

Figure 2: Sample hands-on games and puzzles at the Learning Station

Figure 2. **A**. Cells, casts, crystals (manipulate molecular models to form urea from amino acids). **B**. Cerebrospinal fluid (manipulate drugs moving across blood-brain vs. blood-CSF barriers; perform head injury contra-coup with physical model; examine fake CSF samples and volumes of daily CSF production. **C**. Obesity (make a cola drink from scratch). **D**. Life of a plasma cell (game of B cell production and maturation). **E**. Life of a plasma cell (touch plasma cells and play game identifying structure and function of 5 major antibody classes). **F**. Capillaries are swell (touch various capillary models; touch conceptual models of colloid osmotic pressure and Starling's forces).

During the initial year (2003), second-year medical students who had not experienced the Learning Station in their prior academic year had the lowest satisfaction rate. This suggests that initiation of a new approach such as the

Learning Station might fare better with first-year students. Additionally, students who enjoyed the station likely contributed to a response bias. In this report we did not present evaluation data of each station individually. Consequently the results in Table 2 suffer from "regressing to the mean," and therefore may not accurately represent the true variation in student opinions about each individual station.

Table 2: Summary statistics of evaluation responses

	Academic Year 2003-04 N (%)	Academic Year 2004-05 N (%)	Academic Year 2005-06 N (%)	Academic Year 2006-07 N (%)	Academic Year 2007-08 N (%)
"The level of complexity involved in this learning station was about right"					
Strongly disagree	1 (0.8)	0 (0.0)	1 (0.7)	0 (0.0)	1 (0.8)
Disagree	13 (10.5)	7 (5.2)	3 (2.1)	2 (1.8)	0 (0.0)
Agree	48 (38.7)	50 (37.0)	47 (33.6)	39 (34.5)	23 (18.6)
Strongly agree	22 (17.7)	46 (34.1)	45 (32.1)	33 (29.2)	40 (32.3)
Total number of evaluations	84	103	96	74	64
"Overall the learning station's effectiveness was"					
n/a (e.g did not attend)	39 (31.5)	31 (22.9)	44 (31.4)	39 (34.5)	60 (48.4)
Poor	1 (0.8)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Below average	4 (3.2)	0 (0.0)	1 (0.7)	2 (1.8)	0 (0.0)
Average	23 (18.6)	23 (17.0)	15 (10.7)	20 (17.7)	6 (4.8)
Above average	34 (27.4)	40 (29.6)	36 (25.7)	25 (22.2)	22 (17.7)
Excellent	23 (18.6)	41 (30.4)	44 (31.4)	27 (23.9)	36 (29.0)

As our curriculum has no formal laboratory component, the results may be irrelevant to schools with laboratory curricula. Thus, our students, as well as their responses about the Learning Station, are likely a non-representative sample of pre-clerkship medical students. Furthermore, since our evaluations were anonymous, we don't know the impact of the Learning Station on knowledge retention among individuals. Neither do we have correlative data for individual board performance or clinical skills. In spite of these limitations, we noticed a strong and consistent level of participation and satisfaction by our students over a 5-year period.

SUMMARY AND DISCUSSION

The Learning Station directly addresses medical student desires for kinesthetic learning opportunities through an innovative approach to teaching basic science. In the education literature there has been recent interest in student learning styles.[4, 5, 6, 7, 8, 9] One learning style instrument categorizes learning preferences into visual, auditory, read-write or kinesthetic. Many individuals have multimodal preferences (i.e. two or more preferences).[10] According to recent studies using this instrument, approximately 2/3 of first-year medical students have multimodal learning preferences, and most of these students have a kinesthetic preference among their multiple preferences.[11, 12] The remaining 1/3 of the medical students prefer a single mode of learning, and the largest percentage of those students favor kinesthetic learning. Our data, together with the above evidence from the literature, suggest that a great number of medical students—perhaps the majority—would benefit from or even prefer hands-on activities for learning basic science such as those presented at the Learning Station.

Our approach can be adopted and scaled up to a larger medical school basic science program. If implemented, it would provide: 1) a low-cost creative solution for active kinesthetic learning without the traditional 'wet lab', 2) reinforcement of concepts introduced in either lecture, small group, or PBL cases, 3) opportunity for enrichment in basic science areas under-represented in the lab curriculum (e.g. genetics, cell biology, immunology, pharmacology and nutrition), 4) a space-saving compact learning environment available to students at variable open-lab hours without an instructor present, and 5) self-directed learning (SDL) which provides exploration and discovery. The SDL method is an effective science learning tool [10] and has been shown to correlate with clinical performance.[11]

The Learning Station also presents new opportunities for presenting cross-disciplinary basic science content with clinical context. This approach may inspire and motivate

students to acquire and retain basic science knowledge longer than they might otherwise.[12] This is particularly valuable in light of the recent deliberations by the National Board of Medical Examiners (NBME) to convert the United States Medical Licensing Exam (USMLE) Steps 1 and 2 into a single "Gateway" exam.[13] Further work is needed to define in more detail the ways in which students benefit from the Learning Station, and whether it helps students retain basic science conceptual information during their clerkship years and beyond.

There is no doubt that current pre-clerkship basic science curricula broadly continue to rely heavily on the lecture-based didactic format. Learning of basic science through lecture may create a mismatch between student learning and instructor teaching styles, particularly for students who prefer kinesthetic learning. This raises concerns about students who fall short of program expectations[4], or have low retention of basic science content.[12] Kinesthetic activities, such as those presented at the Learning Station, may even enhance preparation of students for procedure-based clinical specialties such as surgery, obstetrics and emergency medicine.

Development of a new Learning Station by other institutions may require a significant initial time commitment by basic science faculty to acquire or create materials, and later to continually upgrade the modules. Although students and faculty may initially resist a new curricular component with a completely new structure, we are heartened by the evaluation of our learning station by one of our first year students, completed even before his/her participation in any learning station:

> *"I was very impressed by the amount of thought that goes into the learning station—it looks appealing and accessible."*

Sample learning station modules can also be obtained at the website http://jmp.berkeley.edu with Learning Station author's permission (JB).

Table 3: Representative responses to our open-ended question

"Please briefly comment on the level of complexity, the station's correlation with the unit or any other observations you might have."
"Playing with the material definitely helps make it stick a little longer."
"I really enjoyed this one."
"The hands-on approach was helpful—as usual."
"This station was fun."
"I really liked the molecular models portion. It was great to get my hands on those to reinforce structure."
"I enjoyed this learning station and felt that it achieved the goal of teaching us more about obesity from the perspective of multiple disciplines (i.e. histology, nutrition, biochemistry, etc.)"
"I love these learning stations."
"Overall, I loved the station and am excited for the next one!"
"I thought this was a great supplement to the unit. It truly helped me to understand the lungs better, and made me thirsty for more stations!"
"This station was great! I was having trouble understanding this case because the lab values overwhelmed me with numbers. This really gave me a reference that made it easier for me to understand the case. Thanks!"
"I loved it. It was like the Exploratorium for med students. Incredibly beneficial for a visual learner like myself."
"Love love love the learning station. Who knew tactile learning could be so helpful?"
"I always love the learning station. It is a huge asset to my learning."
"More complexity would have been good."
"I was overwhelmed by the amount of information and did not feel I retained it especially well."
"I would have liked a tactile model of the red blood cell wall. Otherwise, the station was helpful."
"I need schedule time to do these stations. I think a reminder email would be helpful."
"I'm still wondering what mesangial cells are. It would be nice to include them in the learning station."

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MEDICAL EDUCATION CASE STUDY

Life Doesn't Stop for School

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ABSTRACT

The personal lives of medical students routinely continue in parallel with their academic endeavors, but at times they intersect with one another. Presented is the case of a medical student whose mother was diagnosed with metastatic breast cancer during his first clinical rotation. The student subsequently experienced both personal and academic difficulties. The case raises questions about when and how faculty and course directors should proceed when these situations arise.

Background

During his first clinical clerkship, a medical student's mother—who had been experiencing chronic, disabling back pain—was diagnosed with advanced metastatic breast cancer. The student is an only child and described himself as very close to his mother, whose immediate family lives in Sweden. Because he considered his father to be a better follower than leader, he expressed concern about his father's ability to help his mother negotiate her health care needs. Shortly after her diagnosis, he was provided an opportunity to travel across country to be at home with his mother, who lived on the West Coast, and to meet with her doctors. The student had experienced no academic difficulties during his first two years of medical school (GPA = 3.43, USMLE Step 1 = 229). During his first clerkship, his academic performance was not as strong as his pre-clinical work. However, he felt he was learning and believed that staying engaged with his studies was important to his mother. He successfully completed his clerkship, scoring at the 22nd percentile nationally on his NBME subject exam.

Family Medicine Clerkship Dilemma

Due to his mother's diagnosis, the geographic location of the student's second clerkship (family medicine) was changed at his request to a clinical site on the West Coast, in order to make it easier for him to visit her on weekends. Prior to the start of this clerkship, the student stopped in to meet with the course director to discuss options to allow him to take some additional days off to be with his mother. He articulated feeling very emotional about his mother's illness and that he had significant concerns about her health care team. He was finding it hard to be away from her and to cope with her prognosis. He further reported that he had begun to seek counseling on these issues with his personal family physician. He felt that he was able to focus on his studies, but just

needed some time to be at home. When approached with the idea of requesting a leave of absence, he was reluctant to do so since his success in school was so important to his mother. In consultation with the Office of Student Affairs, arrangements were made for him to have two, 4-day weekends of travel built into his family medicine clerkship schedule.

During the first week of his clinical training the following direct observations were made by the course director (N.B. week #1 of the 6 week clerkship occurs on the medical school campus, weeks #2-6 occur at the clinical site):

- In order to accomplish the travel arrangements due to the change in location of his clinical clerkship to the West coast clinical site, the student required significant one-on-one assistance from the course director to complete relatively simple tasks.
- His personal physician raised a concern about the student's level of tearfulness, his overall mood, as well as his inability to share his fears about his mother's illness with others.
- Throughout the first week of the training, the student repetitively expressed concerns about his mother's care team and plan. Following a required training session in patient advocacy, he was openly tearful about these worries and reported wishing that he could find doctors who would advocate for his mother.

In response, the course director asked the student to self-assess his ability to concentrate and learn under the current circumstances. He once again denied any concerns. He affirmed his desire to continue in the rotation. Due to her observations, the course director discussed her concerns about the student's well-being with the Office of Student Affairs. In addition, she contacted the student's clinical site director to make him aware of the student's ongoing personal issues and raise a concern about the potential impact of his mother's illness on his upcoming clinical work. Further, the student and course director requested that the clinical site identify someone who would serve as a counselor for the student, but who would have no direct supervisory role over him. The student had agreed that this would be an important resource for him, as well as ongoing contact with his personal physician by phone or e-mail.

Upon arrival at his clinical site on week #2 of the clerkship, the student was noted to be quiet, but always professional and eager to learn. Though initially engaged, he became progressively disengaged and distracted. His knowledge was assessed as below expectations for his level of training. He struggled with time management and confidence.

Due to a growing concern about the impact of current events on his daily work, the physician assigned to serve as his counselor and confidant was asked to meet with the student to discuss these observations. During this session, the student broke down in tears, stating that he felt overwhelmed, conflicted and uncertain what to do. He reported difficulty with concentration and tremendous grief about not being with his mother while she underwent her treatment.

How should this physician, his clinical site director and the course director proceed? Who else in the faculty and the administrative structure should be notified? Should his schedule be voluntarily modified or should it be mandatorily modified? What are the implications if he chooses to withdraw? Should anything have been done sooner?

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Student Response

The attending physician-medical student relationship naturally fosters both a learning and a guidance seeking environment to which students are accustomed. As an individual with no administrative duties, the student's physician counselor sits in a unique position to act as a role model for the student. This counselor can provide guidance and personal advice that the student can understand and which is neutral in concern with regards to his standing in school. The student can first raise his concerns and make decisions with this person without fear of any sort of disciplinary action. He can come away prepared with what he wants to do on a personal level and next follow up with his clinical site director and university faculty.

Following a session between the student and his physician counselor, the clinical site director should reevaluate the student in a personal meeting. Prior to the meeting this individual should consider speaking to the student's university clinical affairs liaison about possibly classifying this student's month as time off (if the student does indeed get vacation months) to prevent a failing grade from being placed

on his transcript. During the meeting clear expectations should be laid down by the site director to the student with the understanding that the director's primary responsibility to the student is their medical education. The student needs to know exactly how he is not meeting standards and that he may not pass if he continues to perform below standards. It should also be made clear that personal judgment is not the issue and that the student's personal decisions about how he would like to proceed due to these life changing events are his to make. The student needs to be told that taking time off is in no way a sign of failure and is a continuing option. However, the clinical director's main role is to reinforce to the student that he is not meeting performance expectations and that there are no exceptions to standards when training to become a physician.

Finally, the student's clinical affairs office needs to be directly involved with the situation. They are in an appropriate position to develop, in consultation with the dean of clinical affairs, options for the student.

At this juncture, following two separate points of intervention, the student needs to be made aware that if he were to choose to continue in his studies, his performance must remain at or above expectations. Otherwise, university directed modification of his status will be mandatory. A "3 strike, you're out" parallel would be appropriate for this situation. The interventions that have taken place thus far have been generous in accommodating the student's requests, as well as giving him a sanctuary for free thought and guidance for him to formulate future paths.

Faculty Response

This case demonstrates the emotional and psychological toll of a devastating illness in a parent on medical students. Not only are students deeply affected by the illness itself, but they may feel compelled to take on medical decision-making for their family, even when they are not fully trained. This conflict can further contribute to the student's sense of helplessness and powerlessness.

The student in this case shows growing signs of depression but finds these difficult to accept. Clearly these feelings are affecting the performance of this competent student. One option would be for the student to meet with his most trusted advisor or his physician (meeting with three faculty members could be overwhelming) and urged to meet with a psychiatrist to address the depression. With the student's permission, perhaps this advisor or the psychiatrist could reach out to the father, and even possibly the mother, so that they would not be worried that a leave of absence would be detrimental to the student's grades or career. Reconnecting with the father might be very affirming in this difficult situation. With more information the parents might join with the physician and/or faculty in giving the student permission to spend time with his mother.

Regarding a change in the student's schedule, a voluntary change would be optimal, but with psychiatry input, if the depression remains serious, a mandatory withdrawal might have to be considered. Students fear this can impact their residency selection and career. When handled well, however, such leaves of absence can show caring and maturity. In my experience I have not seen leaves of absence in such settings detrimental, so some reassurance might be in order.

For this student, earlier counseling about grieving and the importance of taking time off would have been optimal. Administrators could have assured the student that such leaves are common when a parent has a terminal diagnosis. Clearly the school has followed the student closely, which is positive, but by the end of the case definite interventions are needed.

Administrator Response

Many students are relatively inexperienced in balancing life and work which in their case is school. Consequently, a student will often view stepping away from school as an admission of weakness or failure. It is our job, as faculty, mentors, and advisors, to keep students' best interests in mind. This means we must help them to see the long-term implications of decisions they make today. "Powering through" is not always the best answer, even if it may be the most natural reaction for a driven medical student. We should help our students consider creative ways to meet all of their needs, both personal and academic, but not always simultaneously.

It is only natural to want to help this medical student to continue on with his studies, particularly at a time when so much else in his life is going through upheaval. Given that this student had a track record of pre-clinical academic success, it seems appropriate to initially explore special accommodations that allow the student to spend more time caring for his mother.

However, the first warning sign came with the student's uncharacteristically low performance in his

first clerkship. It seems that the geographical accommodations created for the second clerkship may have been proposed without knowledge of the student's performance in his first clerkship. It would have been helpful, early on, to have a planning discussion among people familiar with the student and his situation, including the clerkship directors from the first two clerkships and members of the Office of Student Affairs. In this situation, inclusion of the first clerkship director in these early discussions would likely uncover evidence that this student would benefit from closer, local supervision at the main medical campus, particularly if these special long-distance arrangements only result in creating two weekends for the student to spend at home. These discussions may also serve as the first step toward considering a temporary leave of absence for the student.

Given that some medical schools have the option to take electives during the third year, it would be nice to explore scheduling alternatives that could convert an elective period into a short break for the student to spend with his family. In some cases, taking a short break may help to resolve the personal issues, or it may open the students' eyes to the value of a longer leave of absence. Either way, taking a break of a few weeks during an elective period is likely to be easier for the student to contemplate and carries less of the stigma of letting a parent down.

In the absence of this earlier intervention, another turning point is evident in the beginning of the second clerkship. When the student is openly struggling during the on-site week of his second clerkship, asking the student to self-assess his fitness for continued study is inadequate. The observations of the clerkship director, together with the concerns of the student's physician about his emotional state, should have triggered more serious intervention, precluding sending the student off-site. Although the appointment of a counselor at the clerkship clinical site was an important positive step, it is not a substitute for careful oversight on the main medical campus from a team of mentors who know the student well and would be able to recognize significant mental health issues as they emerged. A discussion should also have been initiated with the student about the potential of these circumstances to hurt his academic record in his required clerkships. Since these grades in particular are of significant importance when program directors evaluate potential residents[1], the student should be reminded of the consequences of poor clerkship performance on his ability to competitively match for residency. If a proposed leave of absence is framed with respect to the student's long-term career trajectory, it may be seen as a more viable possibility.

Ultimately, the overarching goals should be ones that blend the student's best interests with gentle but firm guidance. If left to his own devices, a troubled student might make a problematic situation worse. However, with appropriate direction and perspective from his mentors, he would hopefully become better equipped to make difficult decisions himself before a mandatory change in course is imposed on him.

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Student Perspectives on the Value of Lectures

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ABSTRACT

Critics of the lecture methodology see it as an outmoded device for passive transfer of factual information. Defenders view it as critical, thought-provoking discourse. There is little data clearly supporting either view. Most significantly, the health professional student viewpoint is under-represented in the literature. We have, therefore, investigated the factors that motivate medical and dental students to attend non-compulsory lectures in the basic medical sciences. First year medical and dental students were asked to explain in writing why they attended lectures in a course in which the examinations are based entirely on a set of published notes. Thematic content analysis of the student responses was performed. Ten advantages to attending lectures emerged as dominant themes. These were reformatted as statements and arranged in a questionnaire asking students to indicate on a Likert scale the extent to which they agreed with each statement. The questionnaire was distributed to the new class of the following year. Most of the advantages were highly rated by the second class of students. Those that facilitate learning, e.g. providing focus or breadth, ranked higher than those that merely support learning (anxiety reduction). The advantage that ranked the lowest was time-efficiency, suggesting that students do not view the lecture as a particularly effective way to acquire factual information. We conclude that a substantial number of medical and dental students at McGill view lectures as a valuable multifaceted aid to learning.

INTRODUCTION

For the past few decades, the lecture as a pedagogic methodology has been under attack. According to current conventional wisdom, lectures serve little purpose other than passive transfer of factual information[1]. They are inefficient, obsolete, and oppressive[2,3]. The lecture is, in part, a victim of a more general postmodernist disenchantment with the concepts of authority and hierarchy[3]. The status of the lecture has also suffered from rapidly developing technology that presumably liberates students and enables them to pursue and direct their own educations with minimal guidance from faculty.

The lecture methodology does have its partisans, albeit a minority, who are no less passionate than its detractors. They see the lecture, at its best, as a critical, thought-provoking discourse in which a seasoned expert shares knowledge, experience and insight[3]. They maintain that conceptual information is best conveyed by spoken communication and that the human presence of the lecturer, as well as the social context, contribute significantly to learning[4].

In view of the relatively scant and inconsistent data supporting either side, it is difficult to make sense of this vehement, often vitriolic dispute. Using student performance as an indicator, the majority of comparative outcome studies show no significant differences between the efficacy of lectures and a variety of other pedagogic

methods in terms of knowledge acquisition[1]. Ostensibly, lectures are less effective than other methods when the objective is the application of knowledge, development of thinking skills, or the modification of attitudes[1] Although several more recent studies support this assertion[5,6], others do not[7-9].

The lecture controversy is largely centered on the perceptions of educators. There is little in the literature on the viewpoint of students, particularly those in health professional schools. The paucity of information on student perceptions precludes a complete, balanced and meaningful evaluation of the effectiveness of the lecture as a pedagogic methodology in medical and dental programs. As the beneficiaries of the educational enterprise, the judgment of students is unquestionably as important as that of education providers. We have, therefore, set out to address this issue by exploring the factors that motivate students to attend non-compulsory lectures in a course in which the examinations are based entirely on published notes. Our results indicate that a considerable number first year medical and dental students at McGill recognize a rich variety of pedagogic advantages in the lecture method and that they prioritize these benefits according to their perceived utility.

MATERIALS AND METHODS

Phase 1

We began with a qualitative approach in order to survey student views on the value of lectures. The sample population consisted of 200 first-year medical and dental students enrolled in the neuroscience component of the basic medical science curriculum (Basis of Medicine) in the Faculty of Medicine at McGill University. The students have been informed that lectures are optional and that the examinations are based on notes distributed to the class. The sampling instrument was a single page questionnaire that asked the students to explain in their own words, on the reverse side of the page, why they attended lectures. The students were also asked to indicate their gender, prior educational experience, and level of lecture attendance. The questionnaire was pilot tested on five students from the course and distributed to the rest of the class during the last week of the course. The students were asked to return their questionnaires within a week.

Out of 200 questionnaires distributed, 50 responses were received. Thematic content analysis of the student responses was performed using a constant comparison technique adapted from grounded theory[10]. The individual responses were read independently by J.B. and M.L. in order to identify prominent themes. The readers then met to discuss their results and to reach a consensus. The list of themes was subsequently corroborated independently by C.C. who checked the themes against the student responses. In order to estimate the relative frequency of occurrence of the themes, all 3 investigators reread the responses and counted the occurrence of each theme. J.B. searched the responses for common trends and identified representative quotations.

Phase 2

Ten advantages to attending lecture emerged as prominent themes in the analysis performed in phase 1. These themes were reformatted as statements and arranged in a second questionnaire in which students were asked to indicate the extent to which they agreed with each statement using a Likert scale of 1-5. Each statement in the questionnaire was reviewed by members of the McGill Centre for Medical Education in order to insure that it best reflected the corresponding theme identified in phase 1. Students were also asked to indicate their level of lecture attendance.

The sample population was the first-year medical and dental students enrolled in the neuroscience component of Basis of Medicine in the year following the study in phase 1. The questionnaire was pilot tested on 5 students in the class and was then distributed to the whole class toward the end of the course. The students were asked to return the questionnaires by the end of the course. The average numerical response for each theme was then calculated.

RESULTS

Phase 1

The 50 responses varied in length and quality of content. Some contained no more than a few general phrases, whereas others consisted of substantive, well-structured narratives. Ten advantages of lecture attendance emerged as major themes. These themes, supported by representative quotes, are presented as follows:

1) Lectures provide focus and emphasis
"I find that I get an idea of the really important ideas….", *"I prefer lectures to other interactive formats… because lectures will cover the important points in a clear manner…."*, *"…I feel the instructor will highlight the most important issues…."*

2) Multimodality exposure reinforces learning
"Lectures provide visual and multimedia aids that help with learning….", *"The biggest reason that I attend lecture is to hear the information…."*, *"from my experience, it is better to be exposed to the material in all sensory modalities…."*

3) Lectures explain/resolve difficulties and complexities in the notes and other readings
"…speaker will explain the information in his or her own words, making it more accessible to us.", *"Attending a lecture on complex material allows you to get a different explanation…than what notes provide."*, *"…there are always a few points that I still don't understand having*

read the notes…hearing the professor explain or demonstrate it often clears up the confusion right away."

4) Lectures provide an overview, "the big picture"
"Lectures provide a way to get an overview of the topic.", "Listening to lectures lets me get the big picture….", "A prof can help you connect the dots, help you see the big picture…."

5) Lectures provide exposure to experts/role models
"Experts in the field can provide up-to-date answers to questions and stimulate interest….", "…lecture is a place where we begin to see how a doctor approaches learning the material…how someone more educated and with experience explains the information…."For me, professors are role models."

6) Lectures are a time-efficient way to learn
"…it cuts the amount of study I have to do at home.", "…our lecturers…in an hour could cover what it would take me over 2 hours to learn on my own.", , "…attending lectures is a simple and efficient way to learn about something."

7) Lectures encourage structure and discipline
"Attending lectures…gives me structure.", "…it forces me to get out of bed in the morning.", "…the class schedule encourages discipline in its approach to the material."

8) Lectures provide de pth and insi ght through examples not present in the readings
"Profs can express ideas that are difficult to convey on paper.", "It is easier to grasp concepts because of the examples given in class….", "Professors provide insight and examples which may not be conveyed in the notes.", "

9) Lectures perpetuate a habitual/traditional w ay of learning – soothe anxiety
'I am in the habit….", "I feel pressure (internal) to go and I am afraid to miss something.", "I find that lecture is a "safe method" of learning…", "fear of missing a learning experience…"

10) Lectures provide a dyna mic, inter esting w ay to learn
"I enjoy lectures because they are a more engaging way of seeing the material…." ,"lectures help me because they are active and more interesting.", "…it presents the material in a potentially interesting manner."

Themes **1)** and **2)** were encountered most frequently, **3)** – **6)** somewhat less frequently, and the remainder yet less so. Many of the narratives indicated that the value of lectures depended upon the quality of the lecturer. The most frequently stated characteristics of a good lecturer were animation, enthusiasm, passion, and clarity/organization.

Phase 2

The themes that emerged from the narrative responses were re-formatted as statements and the students were asked to indicate on a Likert scale of 1-5 the extent to they agreed with each statement (1- Strongly disagree, 2- Disagree, 3- Neutral, 4- Agree, 5- Strongly agree). We split 2 of the themes such that the second questionnaire contained 12 rather than 10 statements. One hundred and ten of the 200 students turned in answer cards. The results are presented in Table 1.

The average lecture attendance of the respondents was 4.3 on a scale in which 5 indicates attendance at all of the lectures and 4, attendance at most of the lectures.

DISCUSSION

An unexpectedly large number of perceived advantages to lecture attendance emerged from this study. Most of these advantages were highly rated when evaluated numerically in phase 2, confirming that many students regard the lecture method as a valuable multifaceted aid to learning. Advantages that facilitate learning, such as providing focus and overview ranked higher than those that that merely support learning, such as anxiety reduction and comfort, suggesting that the students regard lectures as actively contributing to the learning process. The low rating of anxiety reduction is at odds with a commonly held view that lectures are valued by students for the security that they provide. The relatively high standard deviation, however, indicates greater variability of student opinion than for most of the other themes. Inasmuch as the advantage that ranked the lowest was time efficiency, it would appear that the students do not consider the lecture to be an especially effective way to acquire factual information.

The value of the lecture method as perceived by students depends upon 2 important factors. A dominant theme emerging from the qualitative survey is the rather obvious point that the utility of a lecture depends heavily upon the quality of the lecturer. It would be valuable to know whether teachers who are highly rated by students design their lectures according to principles that correspond to the most highly rated advantages identified in this study.

Table 1

Mean ratings of the advantages of the lecture method and standard deviations.

STATEMENT IN QUESTIONNAIRE MEA	N	S.D.
1. Hearing about a subject in lecture reinforces my learning.	4.2	0.80
2. Lectures help me focus on what is important in the subject at hand.	4.1	0.71
3. Lectures provide me with an overview, "the big picture" of the subject at hand.	4.0	0.85
4. Lectures provide depth and insight for me through examples not present in the notes or in the reading assignments.	3.9	0.96
5. Lectures help resolve difficulties or complexities in the notes or assigned. readings.	3.8	0.81
6. Attending lectures encourages me to be disciplined in my approach to learning.	3.7	0.97
7. Lectures provide a dynamic and interesting way for me to learn.	3.7	0.97
8. Lectures provide exposure to experts and role models which I value.	3.7	0.86
9. The lecture format is a comfortable way for me to learn.	3.6	0.95
10. Attending lectures encourages me to have a structured approach to learning.	3.6	0.96
11. Attending lectures reduces my anxiety.	3.5	1.11
12. Lectures are a time-efficient way for me to learn.	3.2	1.05

A second important factor is the availability of an excellent source of factual knowledge. In the case of our neuroscience course, this consisted of a complete set of course notes written by the instructors. In the absence of adequate notes, text, or web material, the utility of lectures may well be skewed toward information acquisition. If the potential of the lecture method is to be fully realized, it is incumbent on educators to insure an adequate alternative source for course content.

As in most surveys, our response rate was not 100% (25% for phase 1, and 55% for phase 2), leaving open the question as to the views of the non-responding students. It is likely that many of these students were simply too busy to engage in a task of no direct benefit to them. It is also possible that non-respondents were indifferent or did not value lecture as an important learning tool. Nonetheless, a 55% response rate in phase 2 represents the attitudes of 110 individual learners for whom the lecture approach is a versatile and effective teaching method.

Course directors in the basic science component of the medical curriculum are often encouraged to consider replacing lectures with more interactive methods, such as small group learning. The underlying assumptions are; 1) that the two methods are essentially interchangeable and 2) small group learning is more effective than lecture. We have identified advantages to the lecture method, perceived by students, many of which may be unique to lectures. We have no information on the student perspective on the advantages of small group learning. Thus, it is not clear that lectures can be simply replaced by small group sessions with no loss to the students. We are currently addressing this issue by applying the methodology used in the present investigation to identify students' views on the advantages of small group learning.

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Does Human Simulator-Aided Learning Improve Long-Term Retention of Autonomic Pharmacology Concepts and Facts by Year II Medical Students?

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ABSTRACT

This study was designed to assess whether a human patient simulator (*SimMan*®) improves long-term retention of autonomic pharmacology facts and principles. Twenty-six second year medical students were randomly assigned to either a facilitator-guided simulator (S)-aided or a traditional facilitator-guided paper (P)-aided small group clinical case discussion session. Scores on a pre-session quiz and on small group topic-related course examination questions were compared between the S and P groups. There were no statistically significant differences between the groups with respect to their performance on the pre-session quiz and course examination test items. The results obtained in this pilot study do not support our hypothesis that the use of a human patient simulator in case-based contexts enhances long-term retention of concepts and facts related to the pharmacology of the autonomic nervous system in comparison to traditional paper-based problem solving of the same case-based contexts.

INTRODUCTION

Interest in using human simulators to enhance student learning of concepts and principles in biomedical sciences has existed for decades[1]. Applications of patient simulations in small group teaching and learning environments have been increasingly applied to various student populations in medicine, nursing and allied health sciences during the past decade.[2-6] The use of computer-controlled human patient simulators is receiving increasing support as an educational tool in the training and evaluation of the clinical skills of medical students, residents and physicians.[7] Medical students appear to value simulation-based learning highly.[8] Students especially value the opportunity to apply their theoretical knowledge in a safe and realistic setting and to develop a systematic approach to a clinical problem.[8] Simulators are an important part in the training of medical personnel to help improve patient safety.[9]

Simulation can be integrated into problem-based curricula by simply "bringing to life" existing case material.[10] The use of simulators in preclinical courses, such as pharmacology and physiology, has been shown to help link theory and practice in medical education.[11,12] A simulator-aided learning environment appears intuitively to be superior to paper-based or video-assisted learning environment due to its applicability to real life situations. However, limited research has been published on the actual value of simulation as an educational and/or evaluation tool for pre-clinical medical students, particular in pharmacology.[7] Few studies have addressed the efficacy of patient simulators in long-term retention of new

knowledge. Morgan *et al.*[13] compared the effectiveness of simulator- and video-based, faculty-assisted learning by final year medical students at the University of Toronto participating in a short-term (6 week) anesthesia rotation. Comparison of the final examination scores of students trained by either method showed no significant differences in short-term knowledge retention between the two groups. However, the authors could not predict the comparative effects of the two teaching methods on the long-term retention (> 6 weeks) of knowledge.

The purpose of our study was to investigate the effects of a high fidelity human patient simulator (Laerdal *SimMan*®) on long-term knowledge retention by second year medical students enrolled in the Medical Pharmacology course. Our hypothesis was that second year medical students participating in a trained facilitator-guided clinical case-based small group discussion assisted by a human patient simulator would demonstrate increased knowledge retention when compared to students participating in a facilitator-guided traditional paper-based small group discussion. We assessed retained knowledge at four time points over eight months.

MATERIALS AND METHODS

Study Gr oups: All medical students enrolled in the Medical Pharmacology course, including those participating in this study, attended a series of autonomic pharmacology lectures one week before participating in the related small group session. Faculty prepared syllabi, slide sets and lectures, as well as standard reference texts were available to all students. For the purpose of this study, twenty-six second year medical students were randomly assigned to either a computerized human patient simulator group (Group S: 5M/8F) or a control paper group (Group P: 7M/5F). One student assigned to Group P was absent on the day of the small group session, and was, therefore, excluded from this study. Informed consent was obtained from all participating students in accordance with an IRB approved protocol.[14]

Facilitated Small Group Lea rning: For the past decade at the University of Cincinnati, College of Medicine pharmacology faculty have delivered a series of facilitated small group sessions (ten over eight months) in paper-based clinical case scenario format to promote pharmacology knowledge retention in second year medical students. This format encourages students to apply newly acquired knowledge in clinically relevant contexts. A trained facilitator, either a pharmacology faculty member or an advanced graduate student (MD/PhD program), guides 12-14 medical students through analysis and solutions to cases during each 90-minute session.

For the purposes of this pilot study, sessions were facilitated by two senior graduate students with equivalent levels of experience with and knowledge of this subject matter. The graduate students were directly trained by the Tutorial Director (RK) and the Simulation Center Director (GH) and certified as facilitators for both the paper-based and simulator-based small sessions.

Study Desi gn: Clinical case scenarios (3-4 per session) and related self-study questions for each small group session were posted electronically on an Intranet course system Blackboard) one week prior to the session. Students were asked to independently prepare their answers to the case-based questions prior to the facilitated discussion session with their peers. Immediately before the start of their respective facilitated discussion sessions, all study participants were surveyed (see Student Survey, Appendix 1) with respect to preferences in learning style and the use of learning aids. All students completed a graded 5-question quiz on core pharmacological facts and principles related to the autonomic pharmacology lectures and small group cases at the start of the session. Group P discussed the case materials using a traditional case presentation format guided by a trained facilitator. Group S discussed the same case materials aided by a human patient simulator (*SimMan*®) pre-programmed to simulate a patient's clinical cardiopulmonary symptoms and the physiological responses (e.g., changes in heart rate, blood pressure, respiratory rate, blood oxygen saturation, etc.) to a variety of autonomic pharmacology interventions in each of the clinical case scenarios discussed at the session. No student had prior exposure to *SimMan*®.

Subsequently, all study participants were tested using graphical, tabular, and narrative Step I USMLE format questions on key autonomic pharmacology principles and concepts and on important drug facts discussed at the small group session through a series of four cumulative course examinations administered over an 8-month period. These course examinations were held 1 week (exam 1), 5 weeks (exam 2), 22 weeks (exam 3), and 33 weeks (exam 4), respectively, following the autonomic pharmacology small group session. The number of questions that related to autonomic pharmacology decreased with each exam due to the cumulative nature of the exams. More examination items (n =14) were included on exam 1 administered most proximate to the lecture and small group session than on each of the subsequent 3 examinations; 5, 4, and 4 items, respectively. The same 27 test items, each used one time, were used to compare performance between the two study groups.

Test Item Selection: Examination questions on autonomic pharmacology (and all other course topics) were selected from a secure item bank. These test items were written by course directors and lecturers and subsequently selected based on previous validation for use in examinations by a senior faculty committee, none of whom were associated with the study design or conduct. Each exam was designed to include questions that varied in degree of difficulty based on previous students' performance on these items.

Data A nalysis: All data collection was performed in a blinded manner to maintain participant confidentiality and segregation of participant identity from individual participant scores. All quantitative data were first subjected to a test for normal distribution. Quantitative data determined to be normally distributed, were analyzed by unpaired one-tailed Student's t-test. Otherwise, the Wilcoxon Rank Sums Test was applied to data sets that were not normally distributed. The criterion of $p<0.05$ was established for identification of significant differences. Statistical analyses were performed using SAS version 9.0 (SAS Institute, Cary, North Carolina, USA

RESULTS

Surveys regarding learning styles and study-aid preferences administered before the start of the small group session revealed that students in Group S and Group P had similar "Learning Style" preferences; 67% of Group S and 58% of Group P preferred to "Learn on Their Own". There were no significant differences between the groups with respect to preference for learning in a small group context or for the use of "Learning Aids". There were also no differences between the groups with respect to age (Group P: 22-28 yrs; Group S: 21-27 yrs; 18-22 yrs = 8%; 23-25 yrs = 64%; 26-29 yrs = 28%) or gender (total: 12M/13F; Group P: 7M/5F; Group S: 5M/8F). We calculated an 83% chance of detecting a difference between the mean scores of the groups using a one-tailed t-test at a 0.05 level of significance, based on metrics of our data set.

The scores on the 5-item quizzes administered at the start of the sessions revealed no difference in performance between the Groups S and P (Table 1). Comparison of the Group S and P scores on autonomic pharmacology test items in three of four post-session course examinations also failed to reveal a statistically significant difference between the groups (Table 1). The only outcome difference we detected was student performance on the four autonomic pharmacology test items on exam 4; Group P performed better than Group S ($p = 0.04$). Analysis of the 27 autonomic pharmacology test items from all four examinations yielded no significant differences between the two groups. We also used the Wilcoxon Rank Sums Test to compare the mean scores of students in Groups S and P for the pre-session quiz and on two course exams (2 and 4) because these scores were not normally distributed. This analysis yielded similar results; i.e., no statistically significant differences between Group S and Group P for the pre-session quiz or for exam 2, but on exam 4, Group P outperformed Group S ($p= 0.04$). An unpaired Student's *t*-test showed no significant differences in performance by gender, making it unlikely that the unequal distribution of men and women in Groups S and P affected the study outcome.

DISCUSSION

Our hypothesis, that students exposed to human patient simulator-aided case presentations (Group S) would retain significantly more knowledge of autonomic pharmacology drug facts and principles over an eight-month period than students who received the traditional paper-based training (Group P), was not supported. The results of our study did not reveal improvement in long-term retention of newly acquired autonomic pharmacology knowledge by second year medical students trained using a human patient simulator (*SimMan*®). Groups S and P performed similarly on all 27 autonomic pharmacology test items included in four examinations administered over an eight month period, and on each set of autonomic pharmacology test items in individual examinations; except for exam 4, in which Group P performed better. We failed to identify a benefit for patient simulator use in acquiring and retaining new knowledge of autonomic pharmacology over the course of 8 months. Morgan *et al.*[13] previously reported no significant differences in short-term (6 week) knowledge retention between two groups of medical students completing an anesthesia rotation after receiving simulator- or video-based training. Our study suggests that simulator-aided training is neither better nor worse as an educational tool than more traditional facilitated small group discussion.

Several limitations of this study constrain the interpretation and application of the results. Because group size was small, extrapolation of our results to a larger study population cannot be reliably made. In addition, any potential benefit of the human patient simulator may have been limited by the fact that the students in Group S had only a single exposure to simulator-aided training. Feingold *et al.* suggested that repeated exposure to realistic clinical simulations over a long period of time with real-time feedback and correction of errors may be necessary to observe significant benefits with this method.[15] Our ability to detect a true difference in new knowledge retention between the groups at over an eight month assessment period may have been limited by the relatively small number of test items included on exams 2-4. Another limitation of this study is that paper examinations may not be the best method for detecting enhancement of skill or knowledge gained by using the simulator as they are contextually very different from the simulator sessions. A particular advantage of simulation-based training is its ability to incorporate elements that more closely resemble real-life patient encounters, thereby enhancing students' confidence in making decisions in these settings.[16] It is possible that follow-on assessments conducted in a more realistic setting, such as those that incorporate a standardized or simulated patient, may be better able to detect improvements in knowledge retention facilitated by simulator-aided training.[17] Consistent with this, Gilbart *et al.*[16] noted significant improvement in trauma management skills by students exposed to a computer-based trauma simulator versus students who received no additional training. This skill improvement was only evident when

Table 1:

Autonomic Pharmacology Scores for Simulator (S) Group vs. Paper (P) Group

		Group S (n=13)	Group P (n=12)
	#Test Questions	Mean Score (Std. Dev.)	Mean Score (Std. Dev.)
Pre-Session Quiz	5	4.46 (0.78)	4.43 (0.94)
Exam 1	14	11.23 (1.74)	11.92 (1.56)
Exam 2	5	3.31 (1.18)	3.83 (1.27)
Exam 3	4	3.62 (0.51)	3.25 (0.97)
Exam 4	4	2.77 (0.73)	3.50 (0.52)
Combined score for all 4 exams	32	41.6 (2.7)	39.8 (4.2)

performance was evaluated using a clinically based performance assessment. Improvement was not observed on a paper-based examination. This suggests that traditional paper-based assessment instruments may not be able to discern transfer of knowledge retention that is promoted by simulator-aided training. It is possible that a different assessment modality for evaluating students' retention of newly acquired autonomic pharmacology principles and facts knowledge, such as a case scenario using a standardized patient or patient simulator, would demonstrate a benefit of simulator-aided training not detected using traditional paper-based examinations. However, it must also be acknowledged that differences in individual motivation and learning style preferences, and the use of pharmacology texts, course syllabus, and electronic sources, as well as participation in informal peer study groups are variables beyond the control of the study conditions that potentially confound the study outcome and its interpretation.

Future studies investigating the effects of human patient simulator-aided training on the acquisition and retention of medical knowledge may benefit from incorporating multiple simulator training sessions into the study design. In addition, the use of assessment modalities that closely mimic real-life clinical settings may facilitate the detection of any benefits obtained using the simulator as an educational tool for long-term retention of new knowledge.

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Appendix 1: Student Survey

INFORMATION ON THIS SURVEY
WILL REMAIN STRICTLY CONFIDENTIAL

1. Demographics: Please *circle* the choices that apply to you.

   Male Female

2. Age group 18-22 Age group 23-25
   Age group 26-29 Age group 30-35
   Age group 36-50

3. Learning Style
   - I prefer learning through discussion in small groups.
   - I prefer to study course materials on my own.
   - I do not have a preference.

4. Learning Aids
   - I am a hands-on person when it comes to learning how to use new gadgets.
   - I like to read the manual before I use a new gadget.
   - I have no preference.

Please ***print*** your name: _____

Your identification will remain ***strictly confidential***. All information will be stored in a secure locked cabinet. Once consent has been determined, your name will be removed from the survey instrument.

An Autopsy Review Laboratory is a Valuable Teaching Tool in a General Pathology Course

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ABSTRACT

An Autopsy Review Lab was introduced into a first-year medical student General Pathology course with the intention of providing an effective learning experience for the integration of General Pathology concepts in a clinicopathologic context by utilizing "real patient"-based cases. Four autopsy cases were selected to provide integration of basic principles of General Pathology (Neoplasia, Immunopathology, Cell Injury, Hemodynamic Disorders, Inflammation, Nutritional Pathology). Fixed organs for each case were displayed at separate stations; under faculty supervision, Pathology residents discussed the pathologic findings and their clinical relevance with small groups of students that rotated among the stations. Students critiqued the Autopsy Review Laboratory in required anonymous post-course evaluations. Scores on identical pre- and post-lab tests were compared by paired t-test analysis. Class performance on Autopsy Review Laboratory-related final examination questions was also analyzed.

The average score on the post-laboratory test was significantly higher than the pre-test ($p<0001$). On the final examination, the entire class ($p=0.08$) and the lowest quartile ($p=0.05$) of the class answered more Autopsy Review Laboratory-related questions correctly than unrelated questions. Students also favorably rated the Autopsy Review Laboratory for reinforcing concepts of General Pathology by application to real patients (4.18 out of a 5-point Likert score); for illustrating the value of an autopsy (4.12/5); and for encouraging autopsy requests when they care for patients in the future (3.93/5). Pathology faculty and residents observed that the exercise provided a valuable supervised teaching experience for resident training.

The Autopsy Review Laboratory provides medical students with an effective and enjoyable learning experience for clinicopathological correlation and integration of General Pathology concepts. In addition, the Autopsy Review Laboratory enhances student appreciation of the value of an autopsy and allows Pathology residents to cultivate their teaching skills in a supervised environment.

INTRODUCTION

Recent innovations in medical education have featured early integration of clinical medicine into the curriculum as a strategy to engage medical students' interest and facilitate recognition of the relevance of basic sciences to clinical practice. [1] In our first-year General Pathology course, illustrative patient-based case presentations are offered whenever possible in lectures and in practical laboratories to demonstrate principles of General Pathology. An Autopsy Review Laboratory (ARL), utilizing actual autopsy cases, was designed to provide an effective learning experience for the integration of General Pathology concepts in a clinicopathological context. Autopsy cases illustrating the basic concepts previously

introduced in the course were pre-selected for use in this educational endeavor.

MATERIALS AND METHODS

The six-week, 22-hour General Pathology course at Mount Sinai School of Medicine, given during the Spring semester of the first year, is divided into weekly modules covering the basic concepts of General Pathology: Cell Injury, Inflammation and Repair, Hemodynamic Disorders, Immunopathology, and Neoplasia. Content is provided in lectures, with practical laboratories and team-based learning exercises designed to reinforce the concepts covered. The ARL, an educational exercise given in the final week of the General Pathology course, was introduced in 2006. This one-hour laboratory was repeated in two sequential sessions, to accommodate the entire class of 120 students. Four autopsy cases were selected to highlight and integrate specific basic principles of General Pathology previously presented in the course: 1) Metastatic adenocarcinoma of the breast [Neoplasia]; 2) Tuberculosis with secondary amyloidosis [Inflammation, Immunopathology]; 3) Obesity with deep vein thrombosis and pulmonary embolus [Cell Injury, Hemodynamic Disorders, Nutritional Pathology]; and 4) AIDS with multiple infections [Inflammation, Immunopathology]. Fixed organs for each case were displayed at four separate stations in a large lab. Under the supervision of faculty instructors, senior Pathology residents briefly presented the clinical history of each case and discussed the pathologic findings and their clinical relevance with small groups of 15 students that rotated among the stations at 15-minute intervals. In addition, students were encouraged to identify how autopsy findings contributed to the clinicians' understanding of the clinical course for each case (Figure 1).

Pathology faculty provided the participating residents with a pre-laboratory review of teaching methods and required content. Faculty emphasized key points in the case histories and the salient gross findings and their differential considerations. While the ARL was designed to be informal and interactive, the Pathology residents were provided in advance with a written syllabus that included pertinent questions (along with suggested "answers") to incorporate into the discussions for each case. All syllabus topics in the autopsy review laboratory had been covered in previous modules of the course and were considered critical concepts of General Pathology. During the laboratory, one or two senior faculty members rotated among the stations and observed the resident-student interactions and participated occasionally in the discussion, as needed. After the exercise, the faculty solicited the residents' feedback about the exercise and provided informal feedback to the residents about their teaching efforts with suggestions for future encounters.

Students' response to the Autopsy Review Laboratory was solicited in required, anonymous, post-course evaluations in 2006 and 2007. Questions regarding the ARL were assessed by a 5-point Likert score. Voluntary free-form comments were also reviewed.

In the second year after the introduction of the ARL, its effectiveness as a short-term learning aid for the students was assessed in 2 ways, using (1) "closed-book" pre- and post-ARL multiple-choice quizzes and (2) the final examination. The quizzes consisted of 12 multiple-choice questions covering basic concepts, which had been highlighted in the 4 autopsy cases. All students took the required online, pre-ARL quiz during the weekend before the Laboratory. The post-ARL quiz was administered in-class to the students participating in an elective final course review conducted by the course director two days after the ARL. Sixty-six students took the post-ARL quiz. Performance improvement for the 66 students who took both the pre- and post-ARL quizzes was assessed using a paired t test. In addition, student performance on seven ARL-related final exam questions ("related" questions) was compared with seven randomly selected, unrelated questions (the "unrelated" questions). Specifically, the numbers of related and unrelated questions each student answered correctly were compared using the Wilcoxon signed ranks test.[2] Under the hypothesis that educational interventions may be most effective for students who do not perform as well, we also repeated the above analysis on students who scored in the lowest quartile on the final examination as a whole.[3]

The Associate Dean of Undergraduate Medical Education, Mount Sinai School of Medicine, submitted and received Institutional Internal Review Board (IRB) waiver to use any student survey data for publication.

RESULTS

Statistical analysis of the means of the pre- and post-ARL quizzes (66 students) showed that the average score was 1.4 points higher on the post-test (8.6 vs. 7.2 out of a possible 12 points), and that this difference was highly significant ($p<0.0001$). Evaluation of the final examination showed that for the class as a whole, students on average correctly answered more of the related questions than the unrelated questions, although this difference was not statistically significant ($p=0.08$). For the students scoring in the lowest quartile on the final examination, this tendency was greater ($p=0.054$).

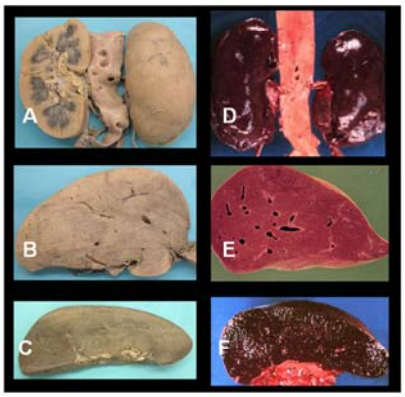
Students were generally enthusiastic (Likert scores $\geq 4/5$) about the ARL as a teaching exercise for reinforcing concepts of General Pathology and for illustrating the value of an autopsy (Figure 2). In addition, students provided many positive, free-form comments about the ARL in response to an open query in the course evaluation asking for examples of course strengths. Examples of such comments included: "The autopsy lab was phenomenal in

FIGURE 1A:

SAMPLE CASE: IMMUNOPATHOLOGY

A 42-year-old Peruvian female immigrant presents with complaints of dyspnea and increased abdominal girth. She was treated empirically for pulmonary tuberculosis several years ago in Peru but was non-compliant with her medications. Physical examination is remarkable for clubbing of the fingers, hepatosplenomegaly, rhonchi on auscultation of the chest, ascites and lower extremity edema. Laboratory studies are remarkable for renal function abnormalities. Chest x-ray reveals cavitary destruction of the upper lobe of her right lung. Early during her work up, she complains of palpitations and is then found unresponsive the following morning.

Patient's lung with cavitary tuberculosis



Patient's kidneys (A), liver (B), and spleen (C) showing amyloidosis, with normal counterparts in pictures D-F

FIGURE 1B:

SAMPLE CASE: IMMUNOPATHOLOGY

Sample Questions Posed to the Students:

- ***How do you describe the pathologic changes of the lung?***
- ***What is your differential diagnosis for the underlying pulmonary disorder?***
- ***How do you describe the pathologic changes of the patient's kidney, liver, and spleen? What is your differential diagnosis for the underlying disorder in these organs?***
- ***Why does this patient have generalized cutaneous edema?***

Course Objectives Reinforced:

- ***Inflammation and Repair: Causes, types, and consequences of chronic inflammation***
- ***Cell Injury: Major patterns and causes of necrosis***
- ***Immunopathology: Types, pathogenesis, and consequences of amyloid***

Value of the Autopsy: Confirmed the presence of pulmonary tuberculosis and diagnosed the unexpected presence of secondary (reactive) amyloidosis

FIGURE 1: An example of one of the autopsy cases utilized in the Autopsy Review Laboratory. **A.** This patient died with chronic pulmonary tuberculosis and previously undiagnosed systemic amyloidosis. The illustrated gross organs (patient's and normal controls) were available for examination by the students. **B.** Sample questions were provided to the Pathology residents to include in their discussions with the students. General Pathological concepts illustrated by this case relate to Immunopathology, Inflammation & Repair, and Cell Injury. The case also demonstrates the value of the autopsy, which revealed unsuspected amyloidosis.

integrating all of the material that we have learned;" "The autopsy at the end was very good to tie things together;" "I

Figure 2

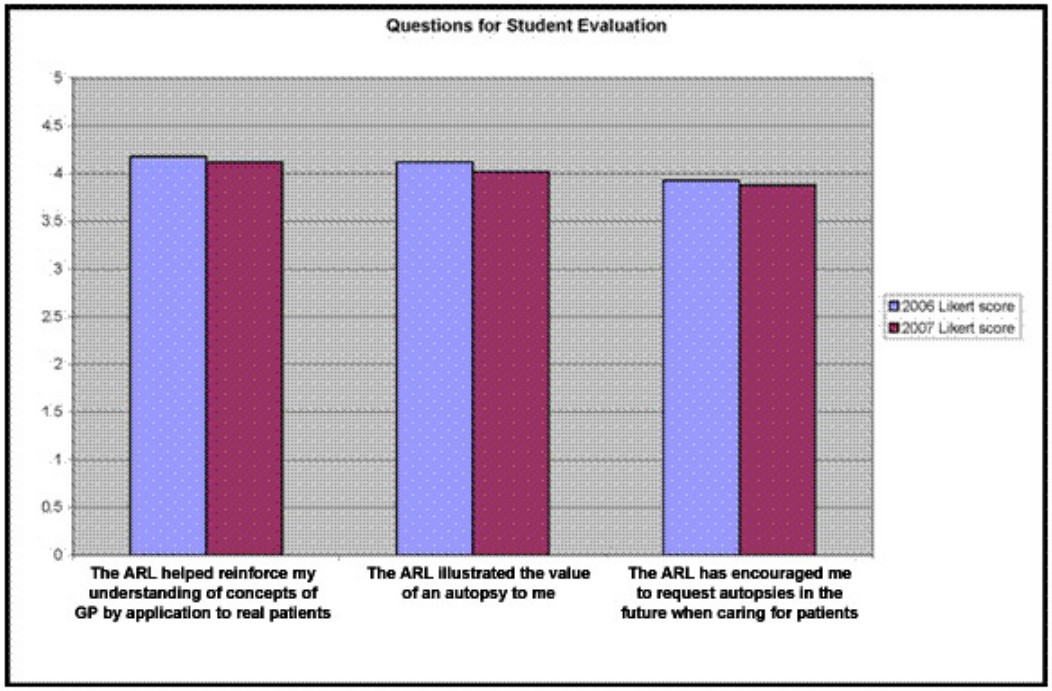


FIGURE 2: Student responses expressed as Likert scores to questions about the Autopsy Review Laboratory in required anonymous post-course evaluations.

also really enjoyed the autopsy review lab." No spontaneous negative comments about the ARL were submitted.

Pathology residents reported that they enjoyed the experience and believed that they improved their teaching skills via pre- and post-exercise suggestions provided by the faculty.

DISCUSSION

The autopsy has traditionally been valued as an important tool in medical education, representing the ultimate example of clinicopathological correlation. Despite the decline in autopsy rates in the U.S. over the past three decades, a number of studies have shown that the autopsy continues to yield valuable information. [4-6] In addition, medical literature continues to support the essential didactic role of the autopsy, [7-11] despite the trend toward computer-generated medical education in recent years.

Myriad factors have contributed to the decline in autopsy rates.[12-14] Junior physicians and physicians-in-training have been swept up in a culture change in Medicine that relies, often erroneously, on the perception that autopsies will not reveal disorders that are not already documented by sophisticated imaging and technological medical advancements. Without an autopsy, the final chapter in our understanding of any patient's medical course is left unwritten. From a practical standpoint, medical students are often no longer afforded the opportunity to participate in and learn from the autopsy.

While we strive to provide the students in our General Pathology course with the opportunity to attend the performance of actual autopsies, the short length of the course makes it impossible for all students to participate in this opportunity. Our eagerness to utilize the autopsy for its educational value in our course was a principal factor in the development of the Autopsy Review Laboratory. Our ARL differs from previously described programs that incorporated viewing autopsies during the medical school years. [9-11] These programs generally involved small groups of students observing ongoing autopsy cases in rotation.

Our use of selected cases allows focused, practical reinforcement of basic pathological principles covered in the course. The use of such "real" patients represents, not only the ultimate demonstration of clinicopathological correlation, but also the clinical relevance of general pathological principles in patient care.

Enlarging medical class sizes and increasing use of digital imaging in medical education, in recent years, have diminished the use of tangible specimens in the teaching of Pathology. The disappearance of gross specimens and microscopic slides from the curriculum has served to divorce students' exposure to the practice of Pathology. Although this issue was not assessed, we also hoped that such a "hands-on" review of pathological specimens coupled with the clinical significance of the findings might illustrate one aspect of the working discipline of Pathology to the students.

By solicited anonymous evaluations, our students appear to have enjoyed the educational experience and found it a valuable learning opportunity. Statistical analysis of objective testing also shows that the ARL had a beneficial effect on short-term learning, perhaps most effectively for the lowest quartile of the class. Others have previously shown a similar disproportionately beneficial effect of supplemental educational exercises on students in the lowest academic quartile.[3] We believe that the educational value of the ARL lies in its reinforcement of concepts previously presented. Its role in long-term learning has not yet been assessed.

While post-course evaluations suggest that students recognized the value of the autopsy to clinical practice, it is too early to assess the long-term effect of the ARL on these students' future utilization of the autopsy in their own practice of medicine.

Resident instruction in effective teaching methods is an essential component of residency training at any academic medical center. Our ARL served as a valuable teaching experience for our Pathology residents by providing a structured forum for them to lead small-group instruction with constructive feedback by senior faculty.

CONCLUSIONS

The Autopsy Review Laboratory provides medical students with an enjoyable and effective learning experience by utilizing a multi-systemic, practical exercise for integration of General Pathology concepts in a clinicopathological context. In addition, the ARL enhances student appreciation of the value of an autopsy and allows Pathology residents to cultivate their teaching skills in a structured and supervised learning environment.

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SPECIAL COMMUNICATION

Design and Implementation of Core Knowledge Objectives for Medical Microbiology and Immunology

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ABSTRACT

Academic curriculum subcommittees of the Association of Medical School Microbiology and Immunology Chairs (AMSMIC) have developed a series of core knowledge objectives for courses in medical microbiology and immunology. Detailed and specific objectives were created by separate subcommittees on Fundamental Microbiology, Host Defenses and Pathogenesis. The academic subcommittees consisted of meeting conferees and distinguished faculty that met at biennial meetings. In 2006 the faculty developed a practical wiki site for membership guidance and revision of the objective documents, allowing changes, contributions and corrections to the core objectives. The wiki afforded the identification of problematic areas and provided a process for ranking objectives, using a numerical rating scale, which provided quantifiable information. The wiki site greatly facilitated the evaluation of core knowledge objectives and was formulated into a condensed set of parameters listing specific academic areas of importance. The final documents contain core competency objectives and provide a format for academic medical microbiology and immunology departments on a national and international level.

INTRODUCTION

In 1986 the Association of Medical Schools Microbiology and Immunology Chairs (AMSMIC) hosted an inaugural educational conference at the Ocean Creek Conference Center in Myrtle Beach, South Carolina. The meeting brought together a wide range of plenary sessions and provided a number of afternoon workshops, covering specific topics on General and Molecular Microbiology, Immunology, Pathogenic Bacteriology, Virology, Parasitology and Mycology. The purpose of these sessions was to provide a format to discuss teaching modalities in medical education. The meetings have continued on a biennial basis since the inaugural session, with the concept of similar workshops facilitated by a variety of speakers and the objective to share experiences in teaching and implementing educational outcomes in medical microbiology and immunology. Additional evening

workshops were also conducted on "Optimal Course Content." In 1991 Cantor and coworkers, in a survey of 1369 medical educators, observed a strong endorsement of the need for "fundamental changes" or "thorough reform" in medical education.[1] In the mid to late 1990's a major shift in the modes of medical student education was underway. The distinct levels of cognition, organized into a taxonomy of general objectives by Bloom, provided a basis for higher education.[2] Other avenues of concern, modifications and changes in the medical curriculum have occurred more recently.[3-6] As modification of the medical curriculum occurred, the "Myrtle Beach" meetings focused on curriculum change, innovative techniques and evaluation formats in medical education. At the 1998 meeting the former course categories were condensed to Pathogenesis/Infectious Disease and Immunology/Host Defenses. Since that time the meetings have, in addition to curriculum discussions, centered on bioterrorism, computerization techniques and up-to-date innovations. The fields of medical microbiology and immunology are rapidly evolving with new research and the appearance of unrecognized pathogens and discovery of new immunological diseases. There is a need therefore to provide a regularly updated resource for core knowledge objectives to aid in the development and improvement of existing discipline-based and integrated medical school curriculums. At the 2006 meeting the curriculum sessions were divided into three distinct areas: (1) Fundamental [Basic] Microbiology, (2) Host Defenses [Immunology] and (3) Pathogenesis [Infectious Disease]. Following the meeting a wiki website was created[7] and made available for the membership to establish core knowledge objectives in these areas. This paper describes the outcome and current status of the medical microbiology and immunology core knowledge objectives project. However, the project is a continuum and the objectives are continuously open for modification at the wiki website and at biennial AMSMIC meetings.

MATERIALS AND METHODS

Beginning in 1998, three groups of faculty at the Myrtle Beach meeting were charged to provide a computerized listing of core knowledge objectives for the disciplines medical microbiology and immunology, including the broad areas of virology, medical parasitology and mycology. In May 2006, at the 11th Educational Strategies Meeting in Myrtle Beach, the learning objectives were further refined to three main academic areas, namely Fundamental Microbiology, Host Defenses and Pathogenesis. Each section subcommittee, directed by faculty facilitators, was responsible for compiling and documenting core knowledge objectives and a wiki website was created following the meeting to facilitate revision of the proposed objectives. The website carried the following general instructions:

> "*After you have logged in, click on your name next to the logoff link and change your password. Please remember to click "Update Password" link to change your password. You may then enter and exit the website.*
>
> *To make corrections or changes in any of the Workshop articles click on the button located on the left side. If you then click on "Edit" you can add your changes to the page. You must scroll down and click the "Save" button for changes to become effective. For major changes to the page please deselect the "minor changes" option before you click "Save."*
>
> *To create sub pages within each article enclose the word you wish to become a link to a sub page with double brackets e.g. [[link]]. After you save the page a new link will appear in the article. Click on the link to create the new page and to start adding content. This same technique can be used to create links to other sub pages within each article. A complete list of acceptable syntax within the wiki is located below the save button.*
>
> *The Education Committee requests that you not change information in any more than two Workshops. If you have questions or need additional information please contact the Workshop Director.*
>
> *Thank you for your efforts and assistance in designing the knowledge objectives for Medical Microbiology & Immunology.*"

In addition, instructions for each academic area, including Fundamental [Basic] Microbiology, Host Defenses [Immunology] and Pathogenesis [Infectious Diseases], are available at: http://mmi.creighton.edu/CoreObjectives/Default.aspx?tabid=53, http://mmi.creighton.edu/CoreObjectives/Default.aspx?tabid=54, and http://mmi.creighton.edu/CoreObjectives/Default.aspx?tabid=55, respectively.

RESULTS

To date 117 responses have been obtained, representing 56 different medical schools in the United States, Canada, Dominica and Grenada. Individual responses totaled 1,847, resulting in 63, 15 and 55 revisions for the sections on Fundamental [Basic] Microbiology, Host Defenses [Immunology] and Pathogenesis [Infectious Diseases], respectively. For the section on Fundamental Microbiology, the working group ranked each item based on importance in the curriculum using a scale of *'3'* for

knowledge that was essential for inclusion in the curriculum, *'2'* for important knowledge that should be included in the curriculum if time is available, and *'1'* for information that was deemed trivial and therefore not required in the curriculum. This version of the core knowledge objectives is shown in Table 1 (*see Appendix*), with an average of the individual rankings. In addition, Table 1 represents the four major divisions of microbiology, including Basic Bacteriology, Basic Mycology, Basic Parasitology and Basic Virology. The division within Basic Bacteriology is divided into 9 subdivisions that represent (A) Structure and Function of Bacteria, (B) Nutrition and Growth, (C) Microbiological Techniques, (D) Physiology and Metabolism of Bacteria, (E) Microbial Genetics, (F) Antibiotic Susceptibility Testing, (G) Antibiotics, (H) Physical and Chemical Agents for Control of Microbial Growth and (I) Host-Parasite/Pathogen Relationships. The second division entails Basic Mycology and includes subdivisions on (A) Principles, (B) Fungal Classification and (C) Antifungal Agents. The third division entails Basic Parasitology and includes subdivisions on (A) Principles and (B) Classification. The fourth and last division entails Basic Virology and includes subdivisions on (A) Principles of Structure and Function, (B) Virus Multiplication and Infectivity and (C) Antiviral Agents.

A second section on Host Defenses Core Knowledge Objectives is represented in Table 2 (*see Appendix*). The rankings, far right-hand column, are similar to Table 1, with a value of *'3'* for essential knowledge, *'2'* for important knowledge and *'1'* for information that was found to be trivial and not an absolute requirement for the curriculum. As indicated, Table 2 is composed of three major divisions, including Division I: Basic Concepts in Immunology, Division II: The Immune System and Disease and Division III: Applied Immunology. Division I on Basic Concepts in Immunology consists of 9 sections that represent (A) General Principles, (B) Development of Cells and Function of Organs, (C) Innate Immunity, (D) Antigens and Antibodies, (E) Antigen Receptor Diversity, (F) MHC, Antigen Processing and Presentation, (G) B and T Lymphocyte Activation, (H) Regulation of the Immune Response and (I) Cell Mediated Immunity. Division II on The Immune System and Disease consists of 6 sections that represent (A) Hypersensitivities, Allergy and Asthma, (B) Autoimmunity, (C) Transplantation Immunology, (D) Immunodeficiencies – Congenital and Acquired, (E) Tumor Immunology and (F) Immunity to Microbes and Vaccines. The final division, Division III: Applied Immunology, is subdivided into 2 sections that represent (A) Immunotherapeutics and (B) Immunodiagnostics.

The final major division on core knowledge objectives for medical microbiology and immunology, Pathogenesis, is represented in Table 3 (*see Appendix*). The right-hand column represents values of *'3'* for information that is essential knowledge to be included, *'2'* for information that is important knowledge to be included if there is time in the curriculum and *'1'* for information that is trivial knowledge not required in a curriculum on Pathogenesis/Infectious Diseases. In addition, Table 3 includes two major divisions, Essential Concepts in Infectious Pathogenesis and Systems-Based Diseases. The division on Essential Concepts in Infectious Pathogenesis is subdivided into 4 subdivisions that represent (A) Encounter with Pathogen, (B) Invasion and Dissemination, (C) Outcomes of Infection, (D) Treatment and Prevention. The second major division on Systems-Based Diseases consists of eleven subdivisions that represent (A) Upper Respiratory Tract Infections, (B) Lower Respiratory Tract Infections, (C) Cardiac Infections, (D) Gastrointestinal Infections, (E) Genitourinary Infections, (F) Genital Tract, (G) Musculoskeletal Infections, (H) Infections of the Nervous System, (I) Degenerative Brain Diseases, (J) Zoonotic Diseases and (K) Opportunistic Infections.

DISCUSSION

Over the past two decades the professional curriculum for medicine and other allied health professions has continued to change.[8-11] In the specialty of medicine the Liaison Committee for Medical Education (LCME), which accredits complete and independent M.D.-granting programs, is recognized as the reliable authority by the nation's medical schools and the U.S. Department of Education for this purpose.[12] In effect, all U.S. and Canadian medical schools operate under the auspices of the LCME accreditation program. Since the LCME inception in 1942, numerous changes have altered and shaped the medical curriculum. In recent years, periodic review and amendment of the standards for the institutional setting, educational program for the M.D. degree, medical students, faculty and educational resources have all played a significant role in modification of the modern day medical school. In addition, new and progressive methods of teaching and changes in the curriculum over the past century have led to a variety of approaches.[13] In general, didactic lectures and paper examinations have been replaced in favor of problem-based learning[14], team-based learning[15], e-based small group, simulation-based learning[16-18] and a shift to the use of computerized[19] and block testing modalities[20], respectively. For medical microbiology and immunology, a single or dual course presentation under direction of the respective faculty remains preferable to provide an appropriate foundation in the period required for basic science. To the contrary, several medical schools have been able to integrate "some" or "a large portion" of medical microbiology and immunology into other coursework. The purpose of developing core knowledge objectives was to provide, not regarding either stand-alone or integrated coursework, guidelines for those minimal concepts and principles that are essential for the integration of medical microbiology and immunology into the practice of medicine. These guidelines provide a yardstick with which all institutions can measure the mastery of basic principles as well as to evaluate understanding and competency of their students, regardless

of the curriculum used. The continuum of core knowledge objectives will be facilitated by future meetings and underscored by faculty participation at the wiki website.

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APPENDIX

Table 1. Fundamental/Basic Microbiology Core Knowledge Objectives

DIVISION I: BASIC BACTERIOLOGY	
A. STRUCTURE AND FUNCTION OF BACTERIA	
1. Compare and contrast prokaryotic and eukaryotic cells, particularly with respect to nuclear membranes, DNA structure, ribosomes, and cell walls	*fn 3.0
2. Describe the morphology and arrangement of bacterial cells using acceptable scientific terms (cocci, bacilli, etc)	3.0
3. Explain the use of the Gram and acid-fast stains	3.0
4. List some important gram-positive, gram-negative, and acid-fast bacteria and their morphology and arrangement	3.0
5. Explain how the Gram stain works (why are gram + bacteria blue and gram - bacteria red?)	2.5
6. Describe the structure and arrangements of flagella	1.0
7. Describe the functions of flagella	3.0
8. State another name for flagella (H-antigen)	1.8
9. Describe the structure of pili/fimbriae	1.0
10. Describe the functions of pili/fimbriae	3.0
11. Explain antigenic (phase) variation of pili or other cell surface proteins and describe its clinical significance	2.8
12. Describe the structure bacterial capsules	3.0
13. Describe the role of bacterial capsules in pathogenicity	3.0
14. List other terms used to describe capsules (e.g., K antigen, slime layer)	2.4
15. Describe the quellung reaction	1.3
16. Describe the formation and importance of a bacterial biofilms	3.0
17. Compare and contrast the structure of Gram-positive and Gram-negative cell walls	3.0
18. Describe the importance of peptidoglycan to bacteria	3.0
19. Explain the importance of peptidoglycan as a target for some antibiotics	3.0
20. Describe the biological activities in humans of Peptidoglycan	2.2
21. Explain what lysozyme is, where it is found, and its biological activity	2.0
22. Describe where teichoic acids are found and their importance	2.7
23. Describe the components and functions of the outer membrane of Gram-negative bacteria	3.0
24. Describe porins of Gram-negative bacteria and their importance	2.9
25. Discuss the structure and biological activities of endotoxin	3.0
26. Describe the Type III secretion system, including where it is found and its importance to pathogenicity	1.5
27. Describe the type IV secretion system and its importance to pathogenicity	2.9
28. Describe how to prepare protoplasts and spheroplasts and their importance	1.5
29. Explain the term “L-form”	1.1
30. Describe why mycoplasmas are unique among the bacteria	1.1
31. Describe the structure and functions of cytoplasmic membranes in bacteria	3.0
32. Describe a mesosome and where they are most commonly found	2.0
33. Explain the term “penicillin-binding protein”	1.0
34. Give another name for this protein (transpeptidase)	2.9
35. Explain its function in bacteria	2.6
36. Describe the major contents of bacterial cytoplasm	2.8
37. Describe the structure and functions of endospores	1.2
38. Name the two major genera of bacteria which produce endospores (*Clostridium*, *Bacillus*)	3.0
39. Describe the primary similarities and differences between the *Clostridium* and *Bacillus* (e.g., Gram reaction, oxygen requirements)	2.8
40. Describe the methods used to classify bacteria	2.8
41. Describe the methods used to identify bacteria in the clinical laboratory	1.4
42. List some important gram-positive, gram-negative, and acid-fast bacteria and their morphology and arrangement	3.0 3.0
B. NUTRITION AND GROWTH	
1. Explain the function of siderophores	3.0

2. Explain the term "fastidious" with respect to bacterial nutrition	2.2
3. Describe the classification of bacteria based upon oxygen requirements	2.9
4. List examples of each	3.0
5. Describe the classification of bacteria based upon temperature requirements	1.8
6. Describe the importance of proper pH for microbial growth	1.8
7. Describe the importance of proper osmotic pressure for microbial growth	1.2
8. Explain the term "halophile"	1.7
9. Name two halophilic pathogens (*V. vulnificus, V. parahaemolyticus*)	2.2
10. Explain the importance of growing in a high salt concentration with respect to *S. aureus* and *Enterococcus*	1.9
11. Explain the term "generation time" and the various factors that can affect it	3.0
12. Describe the four growth phases of bacteria and explain the importance of each	3.0
13. Describe the concept of quorum sensing and its importance	2.7
C. MICROBIOLOGICAL TECHNIQUES	
1. Describe how to perform a Gram stain, explaining the purpose of each manipulation or reagent	2.3
2. Explain how the Gram stain works (i.e., why are gram-positive bacteria blue and gram negative bacteria red?)	2.7
3. Explain how to obtain a pure culture of bacteria	2.0
4. Demonstrate or describe the use and care of a bright-field microscope	1.1
5. Describe microscopic methods used to observe microbial pathogens	1.6
6. Differentiate between nonselective, selective, and differential media	2.9
a. list common examples	2.6
D. PHYSIOLOGY AND METABOLISM OF BACTERIA	
1. Explain the following terms:	
a. glycolysis	2.9
b. fermentation	2.9
c. aerobic respiration	3.0
d. anaerobic respiration	3.0
2. Using the terminology used to classify bacteria based upon oxygen requirements, list examples of bacterial species which typically perform the above metabolic processes	2.6
3. In general terms (not exact numbers) describe the amount of energy (ATP) generated by each of the above metabolic processes	2.3
4. Explain how the metabolic capabilities of bacteria can relate to pathogenicity	2.5
5. Explain how the metabolic capabilities of bacteria relate to identification of genera/species	2.2
6. Discuss the transport processes: active, passive, etc.	1.8
E. MICROBIAL GENETICS	
1. Describe the use of the terms "transcription" and "translation"	2.9
2. Define:	
a. mutation	3.0
b. base substitution	2.7
c. frame-shift mutation	2.7
d. genotype	2.6
e. phenotype	2.7
3. Describe an operon and its regulation mechanisms	2.6
4. Describe DNA repair mechanisms in bacteria	1.6
5. Describe transformation as it occurs in bacteria	3.0
6. Define:	
a. transfection	2.7
b. homologous recombination	2.6
c. nonhomologous recombination	2.5
d. donor	3.0

e. recipient	3.0
f. transformant	3.0
7. Describe conjugation as it typically occurs in Gram-negative bacteria when the donor is:	
a. F+	2.3
b. Hfr cell	2.1
c. F'	1.7
8. Define:	
a. male and female bacteria	2.6
b. F factor	2.7
c. plasmid	3.0
d. sex pili	3.0
e. Hfr cell	2.2
f. episome	1.6
9. Describe resistance transfer factors and discuss their significance to human medicine	3.0
10. Describe the environmental pressures which favor the development of multiply antibiotic resistant bacteria	3.0
11. Describe pathogenicity islands	2.6
12. Define "insertion sequence" and "transposon" and discuss their importance	2.8
13. Discuss selective pressures that can lead to antibiotic resistance	3.0
14. Describe the essential features of bacterial viruses	3.0
15. Define:	
a. bacteriophage	3.0
b. capsid	2.4
c. capsomere	1.9
16. Describe, in words or by a sketch, the lytic cycle as it occurs in bacteriophage infected bacteria	3.0
17. Define "lytic", "virulent", and "temperate" phages	3.0
18. Describe the lysogenic cycle (lysogeny)	3.0
19. Define "prophage"	3.0
20. Define "lysogenic conversion" and discuss the clinical significance	3.0
21. Describe transduction as it occurs in bacteria	3.0
22. Define generalized transduction	2.4
23. Define specialized transduction	2.1
F. ANTIBIOTIC SUSCEPTIBILITY TESTING	
1. Discuss the basis on which antibiotics are selected	3.0
2. List side effects of antimicrobial agents and describe what is meant by each	1.6
3. Discuss the use of antibiotic susceptibility testing	3.0
4. Describe the basic procedures used to perform antimicrobial susceptibility testing and to interpret the test results:	
a. broth dilution	1.7
b. agar plate dilution	1.7
c. agar disk diffusion (Kirby-Bauer)	1.9
d. gradient diffusion (E-test)	1.4
e. colorimetric (chromogenic)	1.2
5. Discuss the pros and cons of each of the above methods of susceptibility testing	1.1
6. Define MIC and MBC	3.0
G. ANTIBIOTICS	
1. Define the following terms as they apply to antimicrobial agents:	
a. broad-spectrum	3.0
b. narrow-spectrum	2.1
c. expanded-spectrum	1.9
2. For the following antimicrobial agents, discuss the primary mode of action, mechanisms of bacterial resistance, spectrum of activity, and any unique characteristics:	

a. sulfonamides	2.9
b. trimethoprim	2.9
c. dapsone	2.1
d. daptomycin	2.1
e. isoniazid	2.6
f. ethambutol	1.7
g. pyrazinamide	1.6
h. beta-lactams (list examples)	3.0
i. cephalosporins (list examples; indicate differences in generations)	3.0
j. cephamycins	1.3
k. carbapenems (imipenem)	2.4
l. monobactams (aztreonam)	2.3
m. vancomycin	3.0
n. cycloserine	1.1
o. bacitracin	2.2
p. polymyxin	2.2
q. quinolones and fluoroquinolones (list examples)	3.0
r. rifampin	2.7
s. aminoglycosides (list examples)	3.0
t. tetracyclines	3.0
u. chloramphenicol	2.7
v. macrolides	3.0
w. lincosamides (clindamycin)	2.7
x. streptogramins and oxazolidinones	2.1
y. nitrofurans	1.3
z. metronidazole	3.0
3. Define bactericidal and bacteriostatic drugs	3.0
4. Explain why cell wall and membrane active agents are usually bactericidal	3.0
5. Draw the essential features of a beta-lactam antibiotic	1.2
6. Explain how a beta-lactamase works	1.3
7. Explain what clavulanic acid, tazobactam, and sulbactam have in common and what they are used for in clinical medicine	2.6
8. Explain why some antimicrobial agents (e.g., cell wall active) are most effective against rapidly growing cells while other agents (e.g., membrane active) are active against both rapidly growing and resting cells	2.6
9. Explain the mechanisms of the following inherent resistances to antimicrobial agents:	
a. mycoplasma resistance to cell wall active antibiotics	3.0
b. anaerobe resistance to aminoglycosides	1.1
c. aerobic resistance to metronidazole	2.6
d. gram-negative resistance to vancomycin	2.6
10. Explain tolerance to beta-lactam antibiotics	2.2
H. PHYSICAL AND CHEMICAL AGENTS FOR CONTROL OF MICROBIAL GROWTH	
1. Define:	
a. antiseptic	3.0
b. aseptic	3.0
c. bactericidal	3.0
d. bacteriostatic	3.0
e. disinfectant	3.0
f. germicide	2.2
g. sepsis	3.0
h. sterilization	3.0
i. pyrogen-free	2.9
2. Describe the general effects chemical and physical agents have on membranes, proteins, and nucleic	

acids which are lethal to cells	2.0
3. Describe the major effect moist heat has on cells	2.2
4. Discuss the pros and cons of using boiling, pasteurization, and autoclaving to control microbial growth	2.3
5. Discuss the use of filtration for sterilization	2.1
6. Describe the effects of ionizing and nonionizing radiation on microbes	2.1
7. Give examples of each type of radiation and its use	1.7
8. Define a thymine dimer	1.8
9. Explain the difference between cationic and anionic detergents (list examples)	1.2
10. Explain the effects surfactants have on bacteria	1.7
11. Explain the mechanism of action and uses of the following in controlling microbial growth:	
a. quaternary ammonium compounds	2.3
b. phenol and derivatives of phenol	2.1
c. alcohols	2.4
d. heavy metals	1.5
e. halogens	1.9
f. iodine	2.0
g. tincture of iodine	2.0
h. iodophor	1.9
i. chlorine and its various forms	1.9
j. hydrogen peroxide	2.1
k. alkylating agents	1.4
l. formaldehyde	1.4
m. glutaraldehyde	1.3
n. ethylene oxide	1.4
o. beta-propiolactone	1.1
p. mineral acids	1.1
q. organic acids	1.1
r. alkalis	1.1
I. HOST-PARASITE/PATHOGEN RELATIONSHIPS	
1. Explain Koch's Postulates	1.7
2. Explain how pathogenic microbes can evade the non-specific first-line defenses of the body	2.8
3. Describe the components of the non-specific second-line defenses of the body and their function as a barrier to disease	2.8
4. Compare and contrast true pathogens and opportunistic pathogens	2.8
5. Explain the difference between a toxigenic and an invasive pathogen	2.9
6. Compare and contrast exotoxins and endotoxins	3.0
7. Define superantigen	3.0
8. List several infectious diseases mediated by superantigens	3.0
9. Explain the biological activities of endotoxin and superantigens	2.9
10. Describe AB toxin structure	2.9
11. Explain the attributes of a microbe which can contribute to invasiveness	3.0
12. Explain the roles played in health and disease by the body's normal flora	2.3
13. List the major normal flora microbes and where they are found	2.9
14. List the major normal flora microbes that are important opportunistic pathogens	2.9
15. Describe where they are normally found and the disease associations	2.9
16. Describe the major mechanisms of transmission of infectious diseases	3.0
17. Define the following:	
a. bacteremia	3.0
b. carrier	3.0
c. communicable disease	3.0
d. endemic	3.0
e. endotoxin	3.0
f. enterotoxin	3.0

g. epidemic	3.0
h. exotoxin	2.9
i. fomite	2.9
j. infectious dose	2.9
k. latent infection	3.0
l. nosocomial infection	3.0
m. opportunistic pathogen	3.0
n. pandemic	3.0
o. pathogenicity	1.9
p. pyemia	3.0
q. pyogenic	3.0
r. pyrogenic	3.0
s. septicemia	3.0
t. subclinical infection	2.9
u. superinfection	3.0
v. systemic	3.0
w. toxoid	3.0
x. virulence	3.0
y. zoonosis	3.0
18. Discuss proper specimen collection from various anatomical sites.	2.0
DIVISION II: BASIC MYCOLOGY	
A. PRINCIPLES	
1. Compare the structure of fungal cells to other eukaryotic cells and to bacteria	3.0
2. Compare and contrast yeasts, molds, and dimorphic fungi	3.0
3. Describe the basis for fungal taxonomy	1.4
4. List the major attributes of the following:	
a. Deuteromycetes (fungi imperfecti)	1.0
b. Zygomycetes	1.4
c. Ascomycetes	1.7
d. Archiascomycetes (*Pneumocystis*)	1.3
e. Basidiomycetes	1.8
5. Define:	1.3
a. hyphae	3.0
b. septate	2.5
c. nonseptate	2.5
d. pseudohyphae	2.5
e. mycelium	2.5
f. rhizoids	2.8
g. zygospores	1.3
h. ascospores	1.2
i. ascus	1.3
j. basidiospores	1.2
k. conidia	2.0
l. arthroconidia	1.8
m. chlamydoconidia	1.2
n. blastospores	1.2
o. sporangiospores	1.2
p. macroconidia	1.8
q. microconidia	2.0
r. dimorphism	3.0
B. FUNGAL CLASSIFICATION	
1. Describe the classification of human mycoses:	

a. superficial (e.g., Malassezia furfur, etc.)	3.0
b. cutaneous (e.g., microsporum, trichophyton, etc.)	3.0
c. subcutaneous (e.g., *Sporothrix schenckii*, etc.)	3.0
d. systemic/endemic (e.g., *Histoplasma, Coccidioides*, etc.)	3.0
e. opportunistic (e.g., *Aspergillus, Candida*, etc.)	3.0
2. Describe the laboratory identification of fungi	3.0
3. Define:	
a. KOH preparation	3.0
b. Sabouraud's agar	1.5
C. ANTIFUNGAL AGENTS	
1. Describe the mechanism of action and clinical use of:	
a. nystatin	2.7
b. amphotericin b	3.0
c. itraconazole	3.0
d. voriconazole	1.4
e. fluconazole	2.8
f. ketoconazole	2.9
g. butoconazole	1.0
h. clotrimazole	2.0
i. miconazole	2.0
j. sertaconazole	1.0
k. econazole	1.0
l. tioconazole	1.0
m. terconazole	1.0
n. caspofungin	2.7
o. anidulafungin	1.2
p. micafungin	1.2
q. terbinafine	1.5
r. naftifine	1.2
s. flucytosine	2.5
t. griseofulvin	3.3
u. tolnaftate	1.3
v. potassium iodide	1.3
DIVISION III: BASIC PARASITOLOGY	
A. PRINCIPLES	
1. Define:	
a. cyst	3.0
b. trophozoite	3.0
c. oocyst	2.8
d. schizogony	2.0
e. vector	3.0
f. intermediate host	2.8
g. definitive host	2.8
B. CLASSIFICATION	
1. Describe parasite classification	2.8
a. protozoa	2.8
(1) rhizopods (entamoeba, etc.)	3.0
(2) ciliates	1.0
(3) flagellates (trypanosoma, leishmania, etc.)	2.6
(4) sporozoa (plasmodium, toxoplasma, etc.)	3.0
b. helminths	3.0

(1) nematodes (ascaris, enterobius, etc.)	3.0
(2) cestodes (taenia, echinococcus, etc.)	3.0
(3) trematodes (schistosoma, clonorchis, etc.)	3.0
DIVISION IV: BASIC VIROLOGY	
A. PRINCIPLES OF STRUCTURE AND FUNCTION	
1. Compare a virus to a cell	3.0
2. Discuss/define the following features of viruses:	
a. size	3.0
b. shape	2.4
c. nucleic acid	3.0
d. capsid	3.0
e. capsomere	2.8
f. nucleocapsid	2.8
g. capsid symmetry	1.1
h. icosahedral	1.7
i. helical	1.7
j. envelope	3.0
k. peplomer	1.2
3. Describe virus classification	3.0
a. list the DNA virus families, including the following features	3.0
(1) enveloped or naked	3.0
(2) DNA structure (DS, SS, linear, circular)	3.0
(3) replication site (cytoplasm, nucleus)	3.0
(4) list medically important examples from each family	3.0
b. list the RNA virus families, including the following features	3.0
(1) enveloped or naked	3.0
(2) RNA structure (DS, SS, linear, circular)	3.0
(3) positive-, negative-sense	3.0
(4) capsid symmetry	1.2
(5) replication site (cytoplasm, nucleus)	3.0
(6) list medically important examples from each family	3.0
4. Describe the following agents, including their replication cycle	3.0
a. defective virus	1.7
b. pseudovirion	1.0
c. viroid	1.0
d. prion	2.7
B. VIRUS MULTIPLICATION AND INFECTIVITY	
1. Describe virus multiplication, including	3.0
a. adsorption	3.0
b. hemagglutinin	2.0
c. entry	2.8
d. naked viruses	3.0
e. enveloped viruses	3.0
f. uncoating	2.8
g. site	2.8
(1) cytoplasm	2.8
(a) RNA viruses	2.8
= (+) ssRNA	2.8
= (-) ssRNA	2.8
= dsRNA	2.8
(2) nucleus	2.8
(a) RNA viruses	3.0

=Retroviruses	3.0
=Influenza virus	3.0
(b) DNA viruses	3.0
h. role of reverse transcriptase	3.0
i. viral protein synthesis	3.0
j. assembly	3.0
(1) lysis	3.0
(2) budding	3.0
k. release	3.0
l. virus load	3.0
2. Discuss the infectivity of naked virus:	2.7
a. DNA	2.8
b. RNA	2.8
3. Define	
a. conditional mutants	1.4
b. recombination	3.0
c. reassortment	3.0
d. complementation	1.8
e. phenotypic mixing	1.7
4. Describe	
a. antigenic drift	3.0
b. antigenic shift	3.0
5. Describe how viruses are cultivated in the laboratory	
a. Describe and/or define the following	
(1) cell culture	2.6
(2) one-step growth experiment	1.7
(3) cytopathic effect	2.6
(4) syncytia	2.7
(5) plaque	2.6
(6) hemagglutination assay	2.1
6. Describe the use of viruses in gene therapy	3.0
7. Explain and give examples of the following viral vaccines	
a. live	3.0
b. inactivated	3.0
c. recombinant	3.0
8. Discuss and/or define	
a. malignant transformation	3.0
b. oncogene	3.0
C. ANTIVIRAL AGENTS	

1. Describe the mechanism of action and clinical use of	
a. acyclovir	3.0
b. famciclovir	1.8
c. valacyclovir	1.8
d. penciclovir	1.8
e. docosanol	1.2
f. trifluridine	1.2
g. ganciclovir	3.0
h. valganciclovir	1.0
i. foscarnet	2.0
j. cidofovir	1.5
k. fomivirsen	1.0
l. amantadine	2.7
m. rimantadine	2.6
n. oseltamivir	3.0
o. zanamivir	2.2
p. ribavirin	3.0
q. adefovir dipivoxil	1.8
r. entecavir	1.3
s. imiquimod	1.5
t. interferon alpha	3.0
u. HAART	3.0
v. nucleoside reverse transcriptase inhibitors	3.0
(1) abacavir	3.0
(2) didanosine	3.0
(3) emtricitabine	3.0
(4) lamivudine	3.0
(5) stavudine	3.0
(6) zalcitabine	3.0
(7) zidovudine	3.0
w. nucleotide reverse transcriptase inhibitors	3.0
(1) tenofovir	3.0
x. nonnucleoside reverse transcriptase inhibitors	3.0
(1) delavirdine	3.0
(2) efavirenz	3.0
(3) nevirapine	3.0
(4) etraverine	3.0
y. protease inhibitors	3.0
(1) amprenavir	3.0
(2) atazanavir	3.0
(3) fosamprenavir	3.0
(4) indinavir	3.0
(5) lopinavir	3.0
(6) nelfinavir	3.0
(7) ritonavir	3.0
(8) saquinavir	3.0
(9) tipranavir	3.0
z. fusion inhibitors	2.3
(1) enfuvirtide	2.3
aa. integrase inhibitors	3.0

*Numbers represent a scale of *'3'* for essential knowledge that was essential for inclusion in the curriculum, *'2'* for important knowledge that should be included in the curriculum if time is available, and *'1'* for information that was deemed trivial and therefore not required in the curriculum.

Table 2. Immunology/Host Defense Core Knowledge Objectives

DIVISION I: BASIC CONCEPTS IN IMMUNOLOGY	
A. GENERAL PRINCIPLES	
1. Describe in overall terms what the host defense system is, why we need it, what it does and how it does it	*fn 3.0
2. Explain the difference between self and non-self	3.0
3. Describe characteristics of active versus passive immunity	3.0
4. Compare and contrast innate and adaptive immunity	3.0
5. Be familiar with the cells of the innate immune response – neutrophils, macrophages and NK cells – know their general function in terms of recognition of microbes, production of cytokines and destruction of microbes	3.0
6. List several examples of physical barriers to infection (e.g., skin, mucous, etc.)	3.0
7. List examples of physiological barriers to infection (temperature, pH, etc.)	3.0
8. Describe the role that complement plays in innate immunity	3.0
9. Describe the phagocytic barrier to infection	3.0
10. Understand the concept of innate pattern recognition of microbes by phagocytic cells	3.0
11. Describe in overall terms the major components of the inflammatory response	3.0
12. Know the local and systemic effects of the innate immune response as they relate to TNF-alpha, IL-1 and IL-6	2.0
13. Understand the transition from innate to adaptive immunity	3.0
14. Describe the essential characteristics of humoral and cell-mediated immunity	3.0
15. List the features of the adaptive immune response – specificity, diversity, specialization, self limitation, memory	3.0
16. Describe 'generation of diversity' in the immune system	2.0
17. Explain the essential role of gene families in the evolution of antigen recognition in the immune system	3.0
18. Describe the theory of clonal selection	3.0
19. Describe the cells involved in the adaptive immune response – T cells, B cells and antigen presenting cells	3.0
20. List the phases of the adaptive immune response – recognition, activation, effector, decline and memory	3.0
21. Describe the basic aspects of T and B cell activation and the role of antigen presenting cells in this process	3.0
22. Describe the basic effector function of T and B cells in an immune response	3.0
23. Describe the significance of immunology in medicine by:	
(A) Listing several examples of disorders affecting the immune response	3.0
(B) Listing several benefits of immunology to medicine	3.0
B. DEVELOPMENT OF CELLS AND FUNCTION OF ORGANS	
1. Describe in general terms the development of white blood cells from stem cells to progenitor cells to mature cells	3.0
2. Describe the different maturational stages of B and T cells (small lymphocyte, blast cell, plasma cell, etc.)	3.0
3. List the markers used to distinguish different lineages, subsets and maturational stages of lymphocytes. Be familiar with the functional significance and/or cellular distribution of the following: CD2, 3, 4, 5, 8, 11b, 14, 16, 19, 21, 23, 25, 40, 45, 56, TCR, BCR, B220	3.0
4. Describe the characteristics and functions of monocytes and macrophages (phagocytosis, antigen processing, etc.)	3.0
5. Describe the characteristics and functions of the granulocytic cells (neutrophils, eosinophils, basophils), mast cells, dendritic cells and natural killer cells	3.0
6. Describe the role of the bone marrow in lymphocyte origin	3.0
7. Describe the role of the thymus in maturation and selection of T lymphocytes	3.0
8. Recall the developmental pathway of T cells in the thymus	1.0

9. Recall that the thymus is the major site of selection and maturation of both helper T cells and CTLs	3.0
10. Explain the processes of positive and negative selection in the thymus. Describe the process of programmed cell death (apoptosis) and its role in the thymus	3.0
11. Describe the overall structure of the TCR (alpha/beta and gamma/delta) and associated polypeptides	3.0
12. Describe the development of TCR and CD4/CD8 expression in maturing T cells	2.0
13. Describe the sequence of events in the B cell developmental pathway from stem cell to immature B cell in the bone marrow	2.0
14. Diagram the order of rearrangement and expression of Ig heavy chain and light chain genes during development of the B cell	2.0
15. Describe the structure of the BCR and associated polypeptides	2.0
16. Recall the antigen-independent and antigen-dependent phases in B cell ontogeny	2.0
17. Recognize that tolerance is the antigen-induced, immunologically specific inactivation of lymphocytes	3.0
18. Recall that two basic mechanisms exist to induce tolerance: (a) clonal deletion and (b) clonal anergy (or functional inactivation)	3.0
19. Describe the major T cell effector populations in the periphery (helper, cytotoxic, regulatory)	3.0
20. Describe the different T helper subpopulations and their role in controlling the immune response	3.0
21. Describe the different peripheral B cell subpopulations (e.g. follicular, marginal, B1 vs. B2)	1.0
22. Recall the role of the germinal center in B cell responses to antigen	3.0
23. Describe the role of the lymphatic system in the transport of antigen and immune cells in the body	2.0
24. Recall the distribution of lymph nodes in the body	2.0
25. Describe the function of the secondary lymphoid organs in trapping and processing of antigens	
26. Recall the functions of different regions of the spleen and lymph nodes in the adaptive immune Response	3.0
27. Recall the location and function of specialized lymphoid tissues such as the mucosal-associated-lymphoid tissues (GALT, BALT, etc.)	2.0
28. Recall the recirculation of lymphocytes and the role of adhesion molecules in lymphocyte trafficking	2.0
C. INNATE IMMUNITY	
1. Recall the role of physical barriers in innate immunity	3.0
2. Recall the role of physiological barriers in innate immunity	3.0
3. Discuss the role of the acute phase response and associated soluble effector proteins in the innate immune response	2.0
4. Discuss the inflammatory process and its role in innate immunity	2.0
5. Recall the key inflammatory cytokines and their local as well as systemic role in innate immunity	3.0
6. Recall definition of complement	3.0
7. Recall principles of complement nomenclature	1.0
8. Describe basic components of complement system	3.0
9. Describe the complement receptors, their expression pattern and their function	2.0
10. Discuss the three main complement pathways – classical, lectin and alternative	3.0
11. List the effector molecules of complement activation and their biologic function	3.0
12. Describe the biological function of complement - chemotaxis, anaphylatoxins, clearance of immune complexes, B cell activation, opsonization, enhancement of phagocytosis, respiratory burst, and cytokine production	3.0
13. Recall the role of complement in bacterial clearance and lysis	3.0
14. Recall the use of plasma CH50 levels in the assessment of disease processes	2.0
15. Recall the regulation of C3 convertase and MAC	2.0
16. Describe various phagocytic cells in the body	3.0
17. Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members and accessory molecules	3.0
18. Recall the role of endothelial cell activation in leukocyte recruitment	2.0
19. List the chemotactic factors involved in the recruitment of various inflammatory cells	3.0
20. Discuss the steps involved in phagocytic cell recruitment and migration into sites of inflammation – rolling, activation, tight adhesion and transendothelial migration	3.0

21. Define the role of Toll Like Receptors in recognition of pathogen associated molecular patterns (PAMPS) and the activation of innate immune cells	3.0
22. Define the role of Fc receptors and complement receptors in opsonization, phagocytosis and activation of phagocytic cells	3.0
23. Describe the stages of phagocytosis – ligand binding, activation, engulfment, fusion of endosome with lysosome and bacterial destruction	3.0
24. Discuss the role of macrophages in antigen processing and presentation	3.0
25. Describe the possible effect of nitric oxide in inflammatory cell-mediated tissue injury	2.0
26. Recall the pathways involved in reactive oxygen burst and the formation of reactive oxygen metabolites following tissue injury	3.0
27. Recall the role of antimicrobial peptides such as defensins or cathelicidins in innate immunity	2.0
28. Discuss the role of natural killer (NK) cells in mediating antiviral immunity and the role of activating and inhibitory receptors in the control of their function	3.0
D. ANTIGENS AND ANTIBODIES	
1. Compare and contrast antigenicity and immunogenicity.	1.0
2. List the chemical classes of antigens.	2.0
3. Define antigen, antigenic determinant, epitope and hapten and give examples of each.	3.0
4. Describe the fundamental difference between B cell and T cell epitopes.	3.0
5. Describe antigen-antibody interaction as a subset of receptor-ligand type interactions.	2.0
6. Define affinity, avidity, and describe their role in immune processes.	3.0
7. Describe the difference between soluble and insoluble immune complexes.	1.0
8. Describe the basic structure of the immunoglobulin molecule.	3.0
9. Recall the overall chain structure of the major classes and subclasses of immunoglobulins.	3.0
10. Recall the different types and subtypes of light-chains.	2.0
11. Explain the differences between isotype, allotype and idiotype.	3.0
12. Recall the overall structure of the major Ig fragments (e.g., Fab, Fc) and describe the enzymatic digestion used to obtain these fragments.	3.0
13. List the major regions of the Ig molecule (e.g., hinge, variable hypervariable, etc.) and describe their overall structure and function.	3.0
14. Diagram the basic domain structure of the Ig molecule and the essential features of the tertiary structure. Describe constant, variable and hypervariable regions with respect to antibody structure.	2.0
15. Explain the differences between isotype, allotype and idiotype.	2.0
16. Recall the specialized functions of the human Ig isotypes. List examples of the specific role of different Ig isotypes in host defense, e.g. IgA may neutralize toxins in the gut.	2.0
17. Describe the composition and function of secretory immunoglobulins.	3.0
18. Diagram the process by which IgA crosses the epithelium and recall the role of the poly-Ig receptor in IgA secretion.	3.0
E. ANTIGEN RECEPTOR DIVERSITY	
1. Describe and explain the molecular genetic mechanisms involved in the generation of antibody diversity (e.g. multiple V region gene elements, variable recombination, junctional diversity etc.).	3.0
2. Diagram the organization of the BCR heavy chain gene locus.	1.0
3. Diagram the organization of the BCR light chain gene loci.	2.0
4. Discuss variable gene recombination at BCR heavy and light chain loci.	1.0
5. Describe reasons for junctional diversity during V gene recombination.	2.0
6. Explain allelic exclusion with respect to immunoglobulin gene expression.	2.0
7. Describe the genetic mechanism used to produce membrane-bound and secreted forms of Ig.	3.0
8. Explain isotype switching and its functional significance.	1.0
9. Describe the mechanism used to regulate expression of IgD.	3.0
10. Describe somatic hypermutation and its functional significance.	3.0
11. Describe the overall structure of the TCR molecule.	2.0
12. Differentiate between the two types of TCR (alpha/beta, gamma/delta) and distinguish their subsets.	2.0
13. Recall the molecular genetic mechanisms used to generate diversity in the TCR.	1.0

14. Compare the gene organization of the TCR loci with that of the BCR loci.	1.0
F. MHC, ANTIGEN PROCESSING AND PRESENTATION	
1. Describe the function of MHC molecules in antigen presentation and in cell-cell interactions in the immune system.	3.0
2. Diagram the genetic organization of the HLA complex.	2.0
3. Describe the three major classes of MHC gene products.	1.0
4. List the major structural features of the MHC gene products (e.g., Class I molecules are two chains, a heavy chain and a beta-2 microglobulin chain.	3.0
5. Identify the tissue distribution of class I and class II MHC.	3.0
6. Recall several examples of MHC/disease correlations and provide a hypothesis to account for this correlation	3.0
7. Explain MHC polymorphism and the likely selective advantage of such a system.	2.0
8. Describe the concept of MHC restriction and provide examples of the functional consequences of the "restriction" of T cell recognition.	3.0
9. Define: haplotypes, genotypes, phenotypes, alleles, linkage disequilibrium.	2.0
10. Diagram a proposed model of MHC-Ag-TCR interaction.	3.0
11. Recall that T lymphocytes recognize antigen bound to MHC molecules and define aggretope versus epitope.	2.0
12. Recall that T lymphocytes primarily recognize protein antigens and in fact recognize linear determinants as opposed to the conformational determinants recognized by B cells.	3.0
13. Recall that T lymphocytes recognize antigen on the surface of other cells (antigen-presenting-cells (APC's) or target cells).	3.0
14. List several examples of APC's (e.g. dendritic cells, macrophages, B cells, etc.).	3.0
15. Describe the phenomenon of MHC-restricted antigen recognition and diagram this recognition process for both helper and cytotoxic T lymphocytes.	3.0
16. Describe in overall terms the conversion of polypeptides to peptide antigens via antigen processing (e.g. the role of acidic intracellular compartments, proteases, etc.).	2.0
17. Diagram the pathway of processing of an exogenous protein antigen.	2.0
18. Diagram the pathway of processing of an endogenous protein antigen.	2.0
19. Describe the selectivity of binding of peptide antigens to Class I and Class II MHC.	1.0
20. Compare and contrast the presentation of exogenous and endogenous antigens to T lymphocytes.	2.0
G. B AND T LYMPHOCYTE ACTIVATION	
1. Discuss the steps involved in lymphocyte activation – protein synthesis, proliferation, differentiation, homeostasis and memory cell formation.	2.0
2. Describe the overall structure of the B cell antigen receptor.	2.0
3. Discuss the fact that the B cell antigen receptor recognizes a wide range of antigens.	1.0
4. Describe the BCR complex including CD79a/b and the role of these associated polypeptides.	2.0
5. Recall the functional significance of Immunoreceptor Tyrosine-based Activation Motifs (ITAMs).	2.0
6. Describe the overall structure of the TCR molecule.	2.0
7. Describe the composition and function of the CD3 complex including the zeta:zeta homodimer.	2.0
8. Describe the activation of T cells, e.g. the interactions between APCs and T cells leading to T cell activation.	3.0
9. List several examples of biochemical events triggered in T cells by antigen recognition.	2.0
10. Discuss the functional role of the T cell accessory protein CD4 and CD8 in recognition of antigen and T cell activation.	3.0
11. List examples of cell adhesion molecules, e.g. ICAM, LFA-1 and discuss their role in T cell activation.	2.0
12. Diagram the mechanism of superantigen activation of T cells.	2.0
13. Describe the mechanism of antigen induced B lymphocyte activation.	2.0
14. List several examples of biochemical events triggered in B cells by antigen recognition.	2.0
15. Compare and contrast the effects of T-independent and T-dependent antigens on B cell activation.	3.0

16. List several examples of T independent antigens and describe the typical characteristics of such antigens (e.g., polyclonal activators, BcR cross-linkers).	3.0
17. Recall the role of B lymphocytes as antigen-presenting cells (APCs).	2.0
18. Describe the mechanism of TH-B cell collaboration and explain the observation known as the "hapten-carrier effect."	3.0
19. Diagram the cell-cell interactions in a humoral immune response to a protein antigen (e.g., TH, cytokines, APCs interact with T cells, activated T cells interact with antigen-specific B cells, etc.).	1.0
20. Define the two-signal model of T cell activation and the role of costimulatory CD28 molecules.	2.0
21. Recall that most B cell responses require CD40-dependent signals in addition to that provided by antigen alone – discuss the functional role of CD40 signaling.	2.0
22. Recall that antigen-dependent signaling in the absence of co-stimulation leads to induction of anergy in T and B cells and what this means.	3.0
H. REGULATION OF THE IMMUNE RESPONSE	
1. Lymphocyte Tolerance and Selection	
a. Describe the concept of central tolerance as it pertains to T cell development in the thymus.	3.0
b. Define the roles played by mechanisms leading to apoptosis and anergy in the regulation of T cell development in the thymus.	2.0
c. Discuss positive versus negative selection of thymocytes.	3.0
d. Describe the concept of central tolerance as it pertains to B cell development in the bone marrow.	1.0
e. Recall the role that receptor editing plays in B cell selection.	2.0
f. Discuss the factors that control T and B cell selection and tolerance including avidity and affinity of interactions between the antigen receptor and antigen.	2.0
g. Discuss the concept of peripheral tolerance induction for T and B cells.	2.0
h. Recall the elimination of autoreactive B cells during the transitional phase in the spleen.	1.0
i. Discuss the role of regulatory T cells in mediating peripheral tolerance.	3.0
j. Discuss the function of tolerogenic dendritic cells in the periphery.	2.0
2. Regulation of Lymphocyte Activation Response	
a. Recall how the process of activation induced cell death relates to feedback control of T cell activation.	2.0
b. Discuss the role of Fas and Fas ligand in mediating apoptosis of activated T and B cells.	2.0
c. Understand the major pathways that lead to apoptosis in lymphocytes (i.e. the mitochondrial pathway and the death receptor pathway).	3.0
d. Understand the role of CTLA-4 in attenuation of T cell activation.	3.0
e. Discuss the role of PD-1 and PD-1 ligand in regulating T cell activation.	1.0
f. Discuss the role of Baff/BLyS and April and their receptors BAFF-R/BR3, TACI and BCMA in regulating B cell survival, activation and differentiation.	1.0
g. Understand the process by which B cell co-receptors modulate antigen receptor signaling through the recruitment of effector proteins to ITIMs, and ITAMs.	2.0
h. Understand the process of antibody-dependent feedback in negative regulation of B cells and the role played by the FcR-gamma-IIb, ITIM.	3.0
i. Discuss the role of CD19 in positively regulating activation of B cells.	2.0
3. Cytokines	
a. Be familiar with cytokine nomenclature and major classifications of cytokines.	3.0
b. Recall basic functions of cytokines in cell-to-cell communication.	2.0
c. Describe the basic properties of cytokines.	2.0
d. Discuss the basic families of cytokine receptors.	2.0
e. Describe the general functions of cytokines (IL-1, 2, 3, 4, 5, 6, 8, 10, 12, IFN, GM-CSF, G-CSF, M-CSF, TNF-alpha, TGF-beta).	3.0
f. Compare and contrast the individual effect of several cytokines (including IL- 1, TNF-alpha, IL-6 and interferons) on innate immunity.	2.0
g. Recall the lipopolysaccharide (LPS)-induced cytokine cascade.	2.0
h. List the cytokines involved in the acute phase response.	2.0
i. Recall the role of several cytokines (including IFNs, Lymphotoxin, IL-5, IL-12) as regulators of	

immune-mediated inflammation.	2.0
j. Recall the role of cytokines in regulation of Ig class switch recombination.	2.0
k. Discuss the role of several cytokines and cytokine receptors (including IL-2, IL-4, IL-6, TGF-beta) in the activation, growth and differentiation of lymphocytes.	2.0
l. Discuss the role of cytokines in T helper cell differentiation into Th1, Th2 or Th17 cells and the production of cytokines by these distinct T helper cell subsets.	2.0
m. Recall the role of chemokines and chemokine receptors in regulation of immune cell trafficking and localization within immune organs.	2.0
I. CELL MEDIATED IMMUNITY	
1. Recall the different populations of effector T cells and their activation requirements.	3.0
2. Discuss the process whereby effector CTLs are generated from CTL precursors.	1.0
3. Recall the process by which effector CTLs recognize target cells.	3.0
4. Discuss the role of Fas and Fas ligand in CTL-mediated lysis of target cells.	3.0
5. Diagram the process of CTL-mediated cell lysis, e.g. role of perforin.	3.0
6. Discuss the role of NK cells in mediating lysis of virally infected target cells.	3.0
7. Recall the mechanism by which NK cell activation is controlled (i.e. activating receptors versus inhibitory receptors).	2.0
8. Describe the mechanisms used by NK cells to lyse target cells.	2.0
9. Recall what antibody dependent cell-mediated cytotoxicity (ADCC) is.	2.0
10. Recall cell-mediated immune responses induced by: NK responses, ADCC, LAK, DTH and give some clinical examples (e.g. contact sensitivity, intracellular infections, granulomas).	2.0
DIVISION II: THE IMMUNE SYSTEM AND DISEASE	
A. HYPERSENSITIVITIES, ALLERGY AND ASTHMA	
1. List the Gell and Coomb's classification of hypersensitivity.	3.0
2. Describe the pathophysiologic mechanisms associated with Type I (IgE)-mediated injury.	3.0
3. Diagram the process of mast cell degranulation.	2.0
4. List the primary effector mediators released by mast cells.	3.0
5. Describe the pathologic changes in tissues during anaphylactic reactions – compare and contrast the acute phase reaction with the late phase reaction.	3.0
6. Explain the modulator role of eosinophils in allergic and anaphylactic reactions.	2.0
7. Correlate the effect of mediators on target organs with clinical expression of allergic reactions.	3.0
8. Discuss therapeutic modulation of type I hypersensitivity.	3.0
9. Describe the clinical expression of anaphylactic reactions and diagnosis via skin tests, RAST, immunoassays, etc.	3.0
10. Describe allergic asthma.	3.0
11. List the bronchial wall changes that occur in asthma.	3.0
12. Recall the treatment considerations of various forms of asthma.	3.0
13. Define type II and type III reactions.	3.0
14. Compare complement mediated cell lysis and antibody dependent cell cytotoxicity.	2.0
15. Compare immunopathology of Goodpasture's syndrome and Lupus.	1.0
16. Diagram the effects of antibodies on cell surface receptors.	1.0
17. Recall drug-induced type I and II hypersensitivity.	2.0
18. Recall erythroblastosis fetalis.	3.0
19. Recall mechanism and histopathology of Arthus reaction.	3.0
20. Describe type IV cell mediated hypersensitivities.	3.0
21. Recall the basis for and examples of contact hypersensitivity.	3.0
22. Discuss the tuberculin reaction.	3.0
23. Describe the granulomatous reaction.	2.0
B. AUTOIMMUNITY	
1. List autoimmune diseases associated with specific organs.	3.0
2. List autoimmune diseases that are systemic in nature.	3.0

3. List several examples of autoimmune diseases mediated by autoantibodies, e.g. myasthenia gravis, Graves' disease, Lupus, etc.	3.0
4. List several examples of autoimmune diseases mediated by T cells, e.g. EAE.	2.0
5. Discuss the role that gender, genetics, environment and infectious disease play in the development of autoimmunity.	2.0
6. Describe several mechanisms that help to explain anti-self responses (e.g. immunological cross-reaction or molecular mimicry).	2.0
7. Describe the role of MHC genes in autoimmunity.	2.0
8. Provide several hypotheses to account for the association of autoimmune diseases with MHC genes, e.g. "molecular mimicry", etc.	1.0
9. Describe the basic types of therapeutic intervention used to treat autoimmune disease.	3.0
C. TRANSPLANTATION IMMUNOLOGY	
1. Discuss the immunologic basis of graft rejection.	3.0
2. Recall the principle of first set and second set rejection.	3.0
3. Understand the terms autograft, isograft, allograft and xenograft.	3.0
4. Discuss the role of CD4 and CD8 T cells in graft rejection.	3.0
5. Recall that the major molecular targets in graft rejection are the non-self MHC molecules.	3.0
6. Describe the difference between major and minor MHC molecules.	1.0
7. Recall the overall molecular structure of the MHC Class I and II molecules.	2.0
8. Recall the overall genetic organization of the HLA complex.	2.0
9. Recall the laws of transplantation.	1.0
10. Describe hyperacute, acute and chronic rejection.	3.0
11. Compare and contrast the immunological reactions occurring in the above types of host response to a foreign graft.	3.0
12. Give examples of tests used to measure tissue histocompatibility.	3.0
13. Recall areas of clinical organ transplantation.	3.0
14. List several approaches to prolonging graft survival (e.g. immunosuppressive drugs, mAbs, immune modulators).	3.0
15. Recall the mechanism of inhibition of T cell activation used by several drugs, e.g. cyclosporin A.	3.0
16. Describe the special immunological complexities that can be associated with bone marrow transplantation (GVHD, etc.).	3.0
D. IMMUNODEFICIENCIES – CONGENITAL AND ACQUIRED	
1. Define congenital versus acquired immunodeficiency.	3.0
2. Recall the basic classification of congenital immunodeficiencies.	1.0
3. Discuss the presentation and pathophysiology associated with severe combined immunodeficiencies: list specific examples of SCID.	3.0
4. Describe the condition associated with DiGeorge Syndrome.	3.0
5. Be familiar with B cell defects, including X-linked agammaglobulinemia, Hyper-IgM Syndrome, Common variable immunodeficiency and selective IgA deficiency.	3.0
6. Be familiar with phagocytic defects, including chronic granulomatous disease, leukocyte adhesion deficiencies and Chediak-Higashi syndrome.	2.0
7. Recall miscellaneous immunodeficiencies, including Wiscott-Aldrich syndrome, Ataxia-telangiectasia and IFN-gamma/IL-12 receptor deficiencies.	1.0
8. Explain the effects of specific complement deficiencies on patients.	2.0
9. Recall basic therapeutic approaches for treatment of SCID, B cell deficiencies and phagocytic cell deficiencies.	2.0
10. List examples of acquired immunodeficiencies and their causes (e.g. AIDS, drug induced, radiation induced).	3.0
11. Recall the immunological abnormalities associated with HIV infection.	3.0

E. TUMOR IMMUNOLOGY	
1. Describe the concept of immunosurveillance.	2.0
2. Recall several examples of tumor antigens, e.g. TSTAs, oncogenic vial antigens, etc.	1.0
3. Describe the roles of antibody, T cells, NK cells, macrophages, etc. in tumor immunity.	2.0
4. Describe the involvement of MHC molecules in tumor immunity, e.g. the effect of virally induced low MHC expression.	2.0
5. List several ways in which tumors evade immune recognition, e.g. antigen modulation.	2.0
6. Describe several approaches to tumor immunotherapy, e.g. antibody-toxin conjugates, IL-2, etc.	1.0
7. Recall the causes of lymphoproliferative disorders.	2.0
8. Recall the different tumors of the immune system.	3.0
F. IMMUNITY TO MICROBES AND VACCINES	
1. Recall the functional differences between the innate versus adaptive immune response.	3.0
2. Discuss the regulation and function of Natural Killer cells in innate immunity.	2.0
3. Describe the role of CD4+ and CD8+ T cells in the adaptive immune response to viral infection.	3.0
4. Recall the role of CD4+ T cells in activation of macrophages.	3.0
5. Discuss the activation and differentiation of CD8+ T cells into cytolytic T cells.	2.0
6. Diagram the process of CTL-mediated cell lysis, e.g. role of perforin.	3.0
7. Describe the immune response to extracellular bacterial infections.	3.0
8. Discuss the immune response to intracellular bacterial infections.	3.0
9. Describe delayed type hypersensitivity as it relates to host responses against intracellular bacteria.	3.0
10. Recall the host immune response to parasitic infection.	2.0
11. Discuss mechanisms of immune evasion.	3.0
12. Recall different types of vaccines (inactivated, attenuated, recombinant vaccines, DNA vaccines).	3.0
13. Recall active vs. passive immunity to microbes.	3.0
14. Recall primary versus secondary immune responses to vaccines and microbes.	3.0
15. Explain the mode of action of adjuvants and recall some examples of adjuvant materials and give examples.	3.0
DIVISION III: APPLIED IMMUNOLOGY	
A. IMMUNOTHERAPEUTICS	
1. Recall the use of monoclonal antibodies to modulate immune cell function or to remove specific immune cells from the body (e.g. anti-CD20 to delete B cells in lymphoma/leukemia or in certain autoimmune diseases).	**
2. Discuss the use of immunosuppressive drugs for the treatment of autoimmune disease or to prevent transplant rejection.	
3. Discuss the use of bone marrow transplantation in the treatment of congenital immunodeficiencies, or cancer.	
4. Recall the use of IVIG in the treatment of autoimmune disease and congenital immunodeficiencies.	
5. Discuss the potential therapeutic roles of cytokines or antibodies specific for cytokines and/or their receptors in: Sepsis, Inflammatory Bowel Disease, Rheumatoid Arthritis and Graft-versus-Host Disease.	
B. IMMUNODIAGNOSTICS	
1. Describe a range of tests used in studying human disease that are based on the specificity of antibodies, e.g. ELISA, Western blotting, flow cytometry, immunofluorescence staining, RIA, etc.	**
2. Describe in overall terms the general principles of each test, e.g. ELISA is based on bound antibody being detected by an enzyme-dependent color change reaction, etc.	
3. Give examples of how different tests are utilized, e.g. immunofluorescence microscopy to detect antigen in tissue sections.	

*Numbers represent a value of *'3'* for essential knowledge, *'2'* for important knowledge and *'1'* for information that was found to be trivial and not an absolute requirement for the curriculum; in this table numbers were rounded to the nearest whole integer.

**Data not available

Table 3. Pathogenesis/Infectious Diseases Core Knowledge Objectives

DIVISION I: ESSENTIAL CONCEPTS IN INFECTIOUS PATHOGENESIS	
A. ENCOUNTER WITH PATHOGEN	
1. Basic Principles	*fn
a. Define, in detail, endogenous (i.e. normal flora) versus exogenous sources of infection.	2.8
b. Explain how normal flora on skin or mucosal membranes can cause disease when introduced into deeper tissues.	3.0
c. Explain how exogenous infections are a result of encounters with organisms in the environment. (e.g. food, water, air, inanimate objects, insect bites, other humans, animals, etc.).	3.0
d. List and discuss the following common mechanisms of microbial transmission.	
(1) Direct skin or mucosal contact	3.0
(2) Inhalation	3.0
(3) Ingestion	3.0
(4) Vertical transmission (congenital; mother to baby)	3.0
(5) Vector –born transmission	3.0
e. Discuss how anatomical sites exposed to the environment serve as portals of microbial entry. (i.e. nose, mouth, respiratory tract, alimentary tract, female genital tract, urinary tract and anus).	3.0
f. Discuss how entry may or may not involve the crossing of epithelial barrier (e.g. inhalation vs. the carrying of microorganisms into deeper tissues by macrophages, or insect bites).	3.0
B. INVASION AND DISSEMINATION	
1. Virulence Factors: Adhesins/Colonization Factors	
a. Explain the significance of microbial adhesion as a component of the establishment of an infection.	3.0
b. Explain which microbial surface structures can function as adhesins	3.0
c. Differentiate between bacterial fimbrial and afimbrial adhesins	1.8
d. Describe what structures act as adhesins for enveloped versus nonenveloped viruses.	3.0
e. Identify the host cell surface components that can act as receptors.	1.7
f. Discuss the function of neutralising antibodies in preventing microbial attachment.	3.0
g. Clarify how attachment helps microorganisms to remain at a particular location/evade innate defense mechanisms.	2.8
h. List some antimicrobial compounds and the targets that are used to interfere with attachment.	1.2
2. Invasins	
a. Define the action of invasins.	3.0
b. Describe the factors responsible for invasiveness of Shigella.	1.4
c. Describe the role of secreted enzymes in invasiveness of bacteria.	3.0
3. Antiphagocytic Mechanisms	3.0
a. Describe the advantage of encapsulation for bacteria.	3.0
b. Name several organisms with anti-phagocytic capsules.	3.0
4. Hemolysins, Cytolysins	
a. Define hemolysin and cytolysin and give an example of a toxin and its producing microorganism for each.	3.0
b. Explain the mechanisms of action for the pore-forming and phospholipase cytolysins.	3.0
c. Discuss the streptococcal hemolysins in terms of their mechanisms of action.	1.7
d. Explain how hemolysis patterns on blood agar can help with species differentiation and disease diagnosis.	1.3
e. List some hemolysins/cytolysins and their functions in terms of the damage seen with a particular infection.	2.3
5. Intracellular vs. Extracellular Multiplication	
a. Discuss the advantages of intracellular growth from a microbial perspective.	3.0
b. Contrast mechanisms of bacterial entry into a phagocytic versus a non-phagocytic cell.	3.0
c. Identify bacteria that rearrange actin to enable their entry and identify the basic steps in the process.	2.5
d. Make a list of (a) Obligate intracellular bacteria and (b) Facultative intracellular bacteria	2.3
e. Fully characterize each of the following intracellular survival mechanisms, giving specific microbial	3.0

examples:	
(1) Escape from phagolysosome	2.2
(2) Prevention of phagosome-lysosome fusion	2.0
(3) Evasion/neutralization of lysosomal contents	2.0
(4) Alteration of phagolysosomal environment	2.0
f. Describe the challenges facing an extracellular bacterium	3.0
g. List the adaptations/virulence factors utilized by extracellular bacteria to evade the host's antimicrobial defenses.	2.7
h. Assess the significance of intracellular growth when selecting an appropriate antimicrobial agent.	3.0
i. Describe the significance of intracellular growth when selecting an appropriate antimicrobial agent.	3.0
6. Tissue Tropism	
a. Explain the significance of tissue tropism in helping to understand microbial pathogenesis	3.0
b. List some bacterial, viral and fungal examples of microorganisms that are tropic for a particular tissue/cell type.	2.2
c. Identify the other factors, both host and microbial, that influence the colonization of a particular site by a microorganism.	2.2
d. Describe how the adhesin-receptor interaction determines the tissue tropism of a microorganism.	2.0
e. Hypothesize as to the significance of a microorganism being able to use more than one receptor-ligand combination.	1.0
C. OUTCOMES OF INFECTION	
1. Colonization vs. Disease	
a. Define symbiosis, commensalism and parasitism.	3.0
b. Describe the benefits to the host of colonization by microorganisms.	2.0
c. Describe several sources of exogenous infection.	3.0
d. Name several factors that predispose to the development of disease when host encounters a microorganism.	3.0
e. Mechanisms of host cell damage	3.0
f. Direct damage from the organism	3.0
2. Toxins	
a. Explain the genetic control of bacterial toxin production.	2.3
b. Explain the difference between exotoxins and endotoxins.	3.0
c. What determines the cell to which an exotoxin binds?	3.0
d. Name several enterotoxins and describe their mechanism of action.	3.0
e. Name two clostridial neurotoxins and describe their mechanism of action.	3.0
f. Name two cytotoxins that exert their effects via inhibition of protein synthesis.	2.8
g. Describe the effects of 3 types of toxins produced by Staphylococcus aureus.	2.5
h. Describe the effect of streptococcal pyrogenic exotoxin.	2.0
i. Explain the mechanism of action of pertussis toxin.	2.0
j. Explain the mechanism of action of tracheal cytotoxin in whooping cough	2.0
k. Name the organisms that produce Shiga toxin and explain its damaging effects.	3.0
l. Describe the differences between apoptosis and necrosis.	2.0
m. Define the source of endotoxins.	2.8
n. Explain the pathogenesis of septic shock produced by endotoxins.	3.0
o. Explain the infectious pathogenesis of disseminated intravascular coagulation.	2.8
3. Invasion	
a. What is the major mechanism of tissue damage of fungi?	2.4
b. What are the two morphologic growth patterns of fungi and which of them is advantageous for the organisms' invasion of host tissue?	3.0
c. What host cell surface molecule is a receptor for several bacteria and viruses?	2.2
d. Explain the process that occurs with bacterial invasion into host cells with the example of Shigella	2.3
e. What process of cell death may be triggered on bacterial invasion of host cells?	2.2
f. Describe the mechanism of amebic enteric disease	2.0
4. Viral Cytopathic Effect	

a. Describe the process by which a virus enters the host cell and brings out cell death in a lytic infection.	3.0
b. Describe the changes in the host cell seen as a result of viral infection.	3.0
c. Explain occurrences in a virally infected cell that result in persistent or latent infection.	3.0
d. Describe the changes in a cell that is transformed by viral infection.	2.7
5. Damage from the Inflammatory Response	3.0
a. Which bacterial components are active in eliciting a host immune response?	3.0
b. Describe the elicitation of the cytokine response to microbial infection of the host.	3.0
c. Which type of immune response is involved in the development of lesions characteristic of *Mycobacterium tuberculosis*?	2.3
d. Differentiate between the immune responses in tuberculoid and lepromatous leprosy.	2.3
e. Describe the mechanism of damage to the host that may occur from virus-antibody immune complexes.	1.3
f. Describe the mechanism of damage to the host that may occur from the cell-mediated response to a virus.	3.0
g. Describe the relationship of the cellular immune response and leishmaniasis.	3.0
h. Explain the damage that may occur with autoimmune sequelae of an infection.	2.0
i. Name the four types of hypersensitivity reactions and give examples of their involvement in host damage in infections.	2.3
6. Mechanisms of Evasion of the Host Defenses	2.3
a. For each of the bacterial virulence factors listed, describe how the factor facilitates evasion of the host immune response (innate and/or adaptive):	
(1) Polysaccharide capsule	3.0
(2) Pili/fimbriae	3.0
(3) IgA protease	2.3
(4) Leucocidin	2.3
(5) Coagulase	1.5
(6) Protein A	2.3
(7) M protein	2.5
(8) Lipoteichoic acid	1.8
b. For each of the bacterial virulence factors listed, give specific examples of medically-important bacteria that possess the factor:	
(1) Polysaccharide capsule	3.0
(2) Pili/fimbriae	3.0
(3) IgA protease	2.0
(4) Leucocidin	2.0
(5) Coagulase	3.0
(6) Protein A	2.8
(7) M proteín	2.8
(8) Lipoteichoic acid	2.8
c. List several medically-important bacteria that are able to survive intracellularly and extracellularly, and explain how their ability to invade and survive inside cells helps them evade the host immune response.	3.0
d. Describe 3 different mechanisms used by some bacteria to evade the degradative enzymes inside phagocytic cells (polymorphonuclear cells, macrophages, or monocytes) and survive intracellularly.	2.8
e. Explain how bacteria in a biofilm are often more resistant to host immune responses.	2.5
f. Explain how antigenic variation facilitates evasion of the host immune response by microbial pathogens, and how this affects host and therapeutic/prophylactic mechanisms to prevent re-infection.	2.8
g. Describe how antigenic variation in the microbial structures listed below contribute to the pathogenesis of the organism:	
(1) *Streptococcus pyogenes* M protein	2.5
(2) *Neisseria gonorrhoeae* pilin protein	2.5
(3) *Streptococcus pneumoniae* capsule	2.5
(4) *Neisseria meningitidis* capsule	2.0

(5) *Salmonella* O and H antigens	2.0
(6) Influenza virus hemagglutinin and neuraminidase	2.8
(7) Rhinovirus capsid protein	2.5
(8) HIV envelope proteins	3.0
(9) HCV envelope proteins	2.0
h. Explain how cytokine "decoy" receptors (or cytokine decoys) produced by some viruses enhance their virulence.	2.0
i. List several viruses that produce cytokine decoys and the host cytokines that are targeted.	1.0
j. Explain what "virokines" are and how they enhance the ability of some viruses to evade the host immune response.	1.0
k. Describe several mechanisms used by viruses to evade the anti-viral interferon response.	2.0
l. Explain how HIV- and CMV-mediated downregulation of MHC class I expression enhances their ability to evade the host immune response.	2.0
m. List several viruses that produce syncytia and how this mechanism of cell-to-cell spread enhances their ability to evade the host immune response.	3.0
n. Explain how HIV infection of T cells affects the host immune response to this virus and other infectious agents.	3.0
o. Explain what is meant by "immune privileged" sites in the body and list several viruses that exhibit a tropism for these sites.	1.0
p. Describe several mechanisms used by viruses to produce persistent infections.	2.0
q. Describe the mechanism by which herpesviruses produce a latent infection in their host and how this contributes to their ability to evade the host immune response.	3.0
r. Describe how "immune tolerance" is developed in neonates infected with hepatitis B virus, rubella virus, or CMV and the effects on the infant.	2.0
s. Compare and contrast the mechanisms of persistence for HBV and HIV.	1.0
t. Compare and contrast the mechanisms of persistence for HBV and HCV.	1.0
u. Explain why prion diseases do not induce a host immune response.	1.0
v. Explain how antigenic shift and antigenic drift contribute to the ability of influenza virus to evade the host immune response.	3.0
w. Explain how viral "quasispecies" are generated and how this contributes to the ability of some viruses to evade the host immune response.	3.0
7. Transmissability	
a. Define/Describe and give examples of the following modes of transmission of infectious agents:	
(1) Person-to-person	2.7
(2) Nosocomial/Hospital-acquired	2.7
(3) Endogenous infection	2.7
(4) Percutaneous/blood-associated	2.7
(5) Fomites	2.7
(6) Soil	2.3
(7) Vertical transmission	2.7
(8) Horizontal transmission	2.7
(9) Aerosols	2.7
(10) Food, water	2.7
(11) Zoonotic	2.7
(12) Sexual contact	2.7
(13) Fecal-oral	2.7
b. Describe structural features of viruses that often affect their stability in the environment and mode of transmission.	3.0
c. List the major sites of entry for infectious agents into the body and the barriers they must overcome at these sites to survive.	3.0
d. Describe conditions that enhance the transmission of infectious agents from person-to-person via non-sexual modes.	3.0
e. Define "reservoir" and "vector" in the context of zoonoses.	3.0
f. Define self-limited vs. resolution of infection vs. chronic infection.	3.0
g. Describe the steps that occur in an acute, self-limiting infection with respect to the pathogen,	

pathogenesis, and host immune response.	2.0
h. List several infectious agents that cause acute, self-limiting infections in healthy, immunocompetent hosts.	3.0
i. List several infectious agents that cause acute, self-limiting infections in healthy, immunocompetent hosts, but can cause persistent infections in immunocompromised/immuno-immature hosts.	3.0
j. Compare and contrast the major characteristics of a chronic viral infection vs. a latent viral infection.	3.0
k. List several infectious agents that can produce chronic infections.	3.0
l. Describe the roles of humoral vs. cell-mediated immune responses in mediating clearance of different types of viruses.	3.0
m. Explain what is meant by the term "chronic carrier" and list examples of infectious agents that can induce this state in human hosts.	3.0
n. List an example of a slow virus and explain how slow virus infections are defined.	2.5
D. TREATMENT AND PREVENTION	
1. Pharmacotherapy – Antibacterial Agents	
a. Differentiate bacteriostatic and bactericidal anti-bacterial agents and give examples of each.	3.0
b. Describe major classes of antibacterial agents based on their mechanisms of action.	3.0
c. What is the most important structural feature of penicillins and cephalosporins?	1.7
d. What is their mechanism of action?	3.0
e. Describe the spectrum of activity of penicillin.	2.3
f. What is the source of bacterial resistance to penicillin?	3.0
g. Name several penicillins that have been developed to overcome this resistance and describe how that has been done.	3.0
h. Name several penicillins (beta-lactam antibiotics) that have been developed to overcome this resistance	3.0
i. Describe how that has been done.	1.3
j. What has been done to develop extended spectrum penicillins?	1.7
k. What is the major side effect of penicillins and why is their toxicity generally limited?	3.0
l. Describe the spectrum of activity of the various generations of cephalosporins.	1.3
m. What is the mechanism of action of vancomycin?	3.0
(1) Against what organisms is it the antibiotic of choice?	2.3
(2) Describe the toxicity of vancomycin.	2.0
n. Describe the mechanism of action and spectrum of activity of daptomycin.	1.7
o. Name antibacterial agents whose activity depends on inhibition of nucleotide synthesis.	3.0
p. Describe the toxicities seen with sulfonamides.	2.0
q. Explain the usage of the sulfamethoxazole-trimethoprim combination.	3.0
r. Name the group of antibacterial agents active through inhibition of DNA synthesis.	3.0
s. What is the spectrum of activity and toxicities of the fluoroquinolones?	2.5
t. Describe the mechanism of action, spectrum of activity, and toxicity of metronidazole.	2.5
u. Name an antibacterial agent that acts through inhibition of RNA synthesis.	3.0
v. In which infections is rifampin used?	2.0
w. Name an antibacterial agent that acts in the early translation steps of bacterial protein synthesis.	2.5
x. Describe the mechanism of action, spectrum of activity, and toxicities of aminoglycosides.	2.5
y. Which antibacterial agent is especially useful against intracellular organisms?	3.0
z. Describe the mechanism of action, spectrum of activity, and toxicity of the tetracyclines.	3.0
aa. What are the adverse effects that have led to decreased usage of chloramphenicol?	3.0
bb. Name the macrolide antibiotics and their specific usages.	3.0
cc At what step in bacterial protein synthesis are they active?	2.0
dd. What is the reason for their numerous drug interactions?	2.5
ee. What is the major adverse effect of clindamycin?	2.5
2. Pharmacotherapy – Antiviral Agents	
a. What steps in the process of viral pathogenesis are targets of antiviral agents?	3.0
b. Name a group of natural antiviral compounds.	3.0
c. Describe the clinical uses and antiviral effects of the Type I and Type II interferons.	3.0

d. Describe the mechanism of action and clinical usage and amantidine and rimantidine.	2.0
e. How does Pleconaril exert its antiviral effect on enteroviruses?	3.0
f. Name antiviral agents that act through inhibition of DNA polymerase.	3.0
g. Why are acyclovir and its related drugs relatively nontoxic?	3.0
h. Describe the mechanism of action of the reverse transcriptase inhibitors, their usages and side effects.	3.0
i. What is the role of protease inhibitors in antiretroviral therapy?	2.0
j. Name two neuraminidase blockers in current usage and show how they are useful.	3.0
k. Describe the mechanism of action of ribavirin and the areas of its usage.	3.0
l. How does foscarnet exert its antiviral effects?	2.0
m. Describe the mechanisms and usage of adefovir, tenofovir, and cidofivir.	1.0
3. Pharmacotherapy – Antifungal Agents	
a. Name the targets of attack for current antifungal agents.	3.0
b. What antifungal drug has been used in most severe life-threatening fungal infections? Why is its usage currently decreasing?	3.0
c. What related drug is clinically useful only in Candida albicans infections of skin, mucous membranes, and GI infections?	3.0
d. Describe the mechanism of action, clinical uses, and side effects of the azoles and allylamines.	2.0
e. Name the class of antifungal agents that attacks fungi at the cell wall. How are they used.	2.0
f. Name the antifungal agent that is now used almost exclusively for dermatophyte infections of the hair and explain its mechanism of action.	3.0
4. Pharmacotherapy – Antiparasitic Agents	
a. Explain why the choice of antiparasitic drugs is limited.	1.0
b. Describe the mechanism of action and usage of metronidazole.	3.0
c. What drug is used for asymptomatic amoebiasis?	2.0
d. Explain the usage of nifurtimox and allopurinol for trypanosomiasis.	1.0
e. How are the pentavalent antimonials effective in leishmaniasis?	1.0
f. Name the current recommended drugs for malaria and explain their mechanisms.	2.0
g. Explain the mechanism of sulfamethoxazole/trimethoprim in parasitic diseases.	2.0
h. Explain the mode of action of antibacterial agents in parasitic diseases.	2.0
i. Explain the usage of the benzimidazoles and ivermectin in helminth infections.	2.0
5. Vaccines	
a. Explain the origin of the term “vaccination”.	1.5
b. Describe the types of vaccines and explain their differences in effectiveness.	3.0
c. Name 2 inactivated viral vaccines currently in use.	3.0
d. What attenuated bacterial vaccine is recommended throughout the world with the exception of the US and the Netherlands?	2.5
e. Name several live attenuated viral vaccines in current use.	3.0
f. Describe the advantages and disadvantages of the oral polio vaccine.	3.0
g. Name two bacterial polysaccharide vaccines and explain their disadvantages.	3.0
h. How has the immunogenicity of *Hemophilus influenza* vaccine been enhanced?	2.5
i. Name two bacterial toxoid vaccines.	3.0
j. What type of vaccine is the current pertussis vaccine?	2.5
k. Give an example of a viral component vaccine in current use.	3.0
l. Explain the concept of recombinant vaccines.	2.0
DIVISION II: SYSTEMS-BASED DISEASES	
A. UPPER RESPIRATORY TRACT INFECTIONS	
1. Rhinitis	
a. Define rhinitis	3.0
b. Name the two types of viruses that cause most cases of rhinitis	2.0
c. Identify the characteristics of each virus	2.0
d. Describe the attachment mechanisms of each virus	3.0
e. Describe the means by which the viruses are spread	1.0

f. Identify the major host defenses preventing infection by these viruses	1.0
g. Identify treatment recommended for rhinitis	3.0
h. What are important causes of rhinitis (rhinoviruses, coronaviruses)?	3.0
2. Pharyngitis	
a. Define pharyngitis	3.0
b. Name the viruses that cause pharyngitis	2.0
c. Identify the characteristics of each of these viruses	2.0
d. Describe the means by which the viruses are spread	3.0
e. Identify sites other than the pharynx that may be associated with pharyngitis caused by some of these viruses	3.0
f. Describe treatment for viral pharyngitis	1.0
g. Name the most common cause of bacterial pharyngitis	3.0
h. Identify the virulence factors of this species	3.0
i. Describe the method of diagnosing bacterial pharyngitis	3.0
j. Describe the normal reservoir of this species	3.0
k. Identify the complications of infection by this species	3.0
l. Describe the events that lead to the complications	2.5
m. Identify the antibiotic(s) used to treat bacterial pharyngitis	3.0
n. List important causes of pharyngitis	3.0
1) Viral	
a) Rhinoviruses	
b) Adenoviruses	
c) Coronaviruses	
d) Epstein Barr virus	
2) Bacterial	
a) *Streptococcus pyogenes*	
b) *Corynebacterium diphtheria*	3.0
c) *Neisseria gonorrhoeae*	1.0
3. Sinusitis	2.0
a. Define sinusitis	
b. Name the three major bacterial causes of sinusitis	2.5
c. Identify the characteristics of each of these bacteria	2.5
d. Identify the virulence factors of these bacteria	2.5
e. Describe the normal reservoir of the bacteria	3.0
f. Identify the major host defenses that protect against infection by these bacteria	2.0
g. Identify factors that predispose a patient to sinusitis	1.5
h. Identify the major complication of sinusitis	2.0
i. Identify the treatment recommended for sinusitis	2.0
j. Important causes of sinusitis	
1) *Streptococcus pneumonia*	3.0
2) *Haemophilus influenzae*	3.0
3) *Moraxella catarrhalis*	2.0
4. Otitis media	
a. Define otitis media	3.0
b. Name the three major bacterial causes of otitis media	2.5
c. Identify the characteristics of each of these bacteria	2.5
d. Identify the virulence factors of these bacteria	2.5
e. Describe the normal reservoir of the bacteria	3.0
f. Identify the major host defenses that protect against infection by these bacteria	1.5
g. Identify factors that predispose a patient to otitis media	2.0
h. Identify the major complication of otitis media	2.5
i. Identify the treatment recommended for otitis media	2.0
j. Important causes of otitis media	
1) *Streptococcus pneumonia*	3.0
2) *Haemophilus influenza*	3.0

3) *Moraxella catarrhalis*	2.0
B. LOWER RESPIRATORY TRACT INFECTIONS	
1. Bronchitis	
a. Define bronchitis	3.0
b. List the types of infectious agents that are involved in most cases of bronchitis	3.0
c. Identify the clinical presentation associated with each infectious agent	1.0
d. Identify the characteristics of each etiologic agent	2.0
e. Describe the attachment mechanisms of each etiologic agent	1.0
f. Describe the major virulence factors and mechanism of pathogenesis of each infectious agent	1.0
g. Describe the means by which the etiologic agents are spread	3.0
h. Identify the major host defenses preventing infection by these agents	2.0
i. Identify treatment recommended for bronchitis	1.0
j. Important causes of bronchitis	
1) Bacterial	
a) *Bordetella pertussis*	
b) *Mycoplasma pneumoniae*	
c) *Chlamydophlia pneumoniae*	
2) Viral	
a) Influenza virus	
b) Adenovirus	
c) Respiratory syncytial virus (RSV)	
2. Bronchiolitis	
a. Define bronchiolitis	3.0
b. Name the viruses that cause bronchiolitis	3.0
c. Identify the characteristics of each of these viruses	2.0
d. Describe the major virulence factor(s) and mechanism(s) of pathogenesis of each virus	1.0
e. Describe the means by which the viruses are spread	3.0
f. Describe treatment for viral pharyngitis	1.0
g. Name the most common cause of bacterial bronchiolitis	2.0
h. Identify the clinical presentation associated with each bacterium	1.0
i. Describe the method of diagnosing bacterial bronchiolitis	2.0
j. Describe the major virulence factors and mechanism of pathogenesis of each infectious agent	1.0
k. Describe the normal reservoir of this species	3.0
l. Identify the complications of infection by this species	2.0
m. Describe the events that lead to the complications	2.0
n. Identify the antibiotic(s) used to treat bacterial bronchiolitis	1.0
o. Important causes of bronchiolitis	
1) Bacterial	
a) *Mycoplasma pneumoniae*	
b) *Bordetella pertussis*	
2) Viral	
a) Respiratory syncytial virus	
3. Pneumonia	
a. Define pneumonia	3.0
b. Differentiate between chronic and acute pneumonia	1.5
c. Name the major etiologic agents of pneumonia	3.0
d. Describe the normal reservoir of these etiologic agents	3.0
e. Identify the clinical presentation associated with each infectious agent	2.5
f. List pneumonia agents suggested by environmental history	3.0
g. Discuss the differential diagnosis of cavitary lesion on chest radiograph	2.0
h. Identify the characteristics of each etiologic agent	2.5
i. Describe the attachment mechanisms of each etiologic agent	1.5
j. Describe the major virulence factors and mechanism of pathogenesis of each infectious agent	2.5

k. Describe the means by which the etiologic agents are spread	3.0
l. Identify the major host defenses preventing infection by these agents	2.0
m. Identify factors that predispose a patient to pneumonia	3.0
n. Identify treatment recommended for pneumonia	3.0
o. Important causes of pneumonia	
1) Bacterial	
(a) *Streptococcus pneumoniae*	3.0
(b) *Legionella pneumoniae*	3.0
(c) *Mycoplasma pneumoniae*	3.0
(d) *Mycobacterium tuberculosis*	3.0
(e) *Bacillus anthracis*	2.0
(f) *Chlamydia psittaci*	2.0
(g) *Rickettsia*	1.5
(h) *Coxiella burnetti*	1.5
(i) *Klebsiella*	2.5
(j) *Pseudomonas* (COPD, cystic fibrosis)	3.0
2) Fungal	
(a) *Histoplasma capsulatum*	2.0
(b) *Coccidioides immitis*	2.0
(c) Pneumocystis jiroveci	3.0
(d) *Blastomyces dermatitidis*	2.0
3) Viral	
(a) Respiratory syncytial virus	3.0
(b) Influenza virus	3.0
(c) Severe Acute Respiratory Syndrome Coronavirus	1.5
(d) Human metapneumovirus	1.5
(e) Bunyaviridae (Hantavirus pulmonary syndrome)	1.5
a. Adenovirus	2.5
C. CARDIAC INFECTIONS	
1. Endocarditis	
a. Name the organisms that commonly cause endocarditis.	3.0
b. Explain the epidemiologic factors (exposure, portal of entry) underlying specific etiologies in particular patients (i.e., Strep or Staph are common causes due to repeated transient exposure from the normal flora of the patient, for instance transient viridans Strep viremia associated with brushing teeth or dental work; Candida and other infectious agents associated with prosthetic valves or injection drug users; etc..)	3.0
c. Describe the "vegetative" lesions associated with endocarditis and explain how such lesions contribute to the diagnosis (persistently positive blood cultures, mass on valves by echocardiogram) and affect therapeutic options (choice of bacteriostatic versus bactericidal antibiotic therapy, etc.)	2.5
d. Explain how laboratory procedures could distinguish between these various organisms.	3.0
e. What clinical sample would be used, what lab procedures, which selective & differential media, and which biochemical assays would be necessary to distinguish between these pathogens?	3.0
f. What are important virulence factors for these pathogens? How do these factors contribute to the virulence of the organisms?	3.0
g. Important causes of endocarditis	
1) Streptococci	3.0
2) Pneumococci	2.5
3) Enterococci	3.0
4) Staphylococci	3.0
5) Gram (-) bacilli	2.0
6) Candida	1.5
7) Other microbes	1.5
2. Myocarditis	

a. Name the most common infectious cause of myocarditis.	2.5
b. Describe the epidemiology and pathogenesis of coxsackievirus infections and explain why most coxsackievirus infections are subclinical.	2.0
c. What is the protective acquired immune response that prevents disease in most people infected with this virus and how does the timing of this immune response correlate with symptomatic versus nonsymptomatic infection?	1.0
d. Important causes of myocarditis	
1) Coxsackieviruses	
2) Many other infectious agents	
D. GASTROINTESTINAL INFECTIONS	
1. Gastroenteritis	
a. Define diarrhea.	3.0
b. Differentiate gastroenteritis and enterocolitis.	3.0
c. Name the most common cause of diarrhea in infants.	3.0
d. Describe the clinical findings in acute gastroenteritis.	3.0
e. Differentiate an invasive infection vs. a toxin-mediated illness based on clinical findings.	3.0
f. Describe the two main modes for transmitting infectious agents that cause gastroenteritis and diarrhea.	3.0 3.0
g. Describe the pathogenesis of bacterial diarrhea.	2.5
h. Explain the mechanisms of damage from enterotoxins, cytotoxins, and invasive organisms.	2.5
i. Differentiate bacterial and viral causes of gastroenteritis based on clinical findings.	3.0
j. Describe the diagnostic techniques used to identify organisms causing gastroenteritis.	3.0
k. Describe the recommended treatment for gastroenteritis.	
l. Important causes of gastroenteritis	
1) Bacteria	3.0
a) *E. coli*	3.0
b) *Shigella sp.*	3.0
c) *V. cholerae*	2.0
d) *V. parahemolyticus*	3.0
e) *C. difficile*	3.0
f) *Salmonella sp.*	1.0
g) *Yersinia sp.*	3.0
h) *C. perfringens*	
2) Viruses	3.0
a) Norovirus	3.0
b) Rotavirus	
3) Parasites	2.0
a) *Entamoeba histolytica*	2.5
b) *Giardia lamblia*	2.5
c) *Cryptosporidium*	
2. Hepatitis	
a. Define hepatitis.	3.0
b. Define jaundice.	3.0
c. Describe the symptoms and laboratory findings in hepatitis.	3.0
d. Describe the mechanism of liver damage in hepatitis.	3.0
e. Name the potential long-term sequelae of hepatitis.	3.0
f. Name several external factors that greatly accelerate microbe-induced liver damage.	2.0
g. What is the fatality rate of fulminant hepatitis?	1.0
h. For the following hepatotropic viruses describe the basic viral properties, principal routes of infection, global prevalence, potential to establish chronic infections, clinical symptoms, means of diagnosis including serologic markers, treatment options, and availability of vaccines:	3.0
1) Hepatitis A Virus (HAV; Picornavirus)	3.0
2) Hepatitis B Virus (HBV; Hepadnavirus, Pararetrovirus)	3.0

3) Hepatitis C Virus (HCV; Flavivirus)	3.0
4) Hepatitis D Virus (HDV; Unclassified defective virus, needs HBV helper)	3.0
5) Hepatitis E Virus (HEV; Unclassified – Calicivirus-like)	3.0
6) Yellow Fever Virus (YFV; Flavivirus)	2.0
i. Name several additional viruses that may target the liver.	2.0
j. Name 2 spirochetes that may target the liver.	2.0
k. Name 2 parasites that may target the liver	
3. Other food/water-borne diseases	3.0
a. Typhoid fever	3.0
b. *Campylobacter jejuni* infection	2.5
c. Botulism	2.5
d. Infant botulism	3.0
e. *Staphylococcus aureus* infection	
4. Oral/oral diseases	3.0
a. *Helicobacter*	
E. GENITOURINARY INFECTIONS	
1. Urinary Tract: Cystitis; Pyelonephritis	
a. Define cystitis and pyelonephritis	3.0
b. Distinguish acute from chronic pyelonephritis	1.5
c. List the most common causes of community-acquired v. nosocomial urinary tract infections (UTIs)	3.0
d. Explain the routes of transmission of agents of UTIs	3.0
e. Describe the primary virulence factors of bacterial agents of UTIs	2.0
f. Identify the major host defenses that protect against infection by these bacteria	2.0
g. Identify factors that predispose patients to UTIs	3.0
h. Explain the prevalence of bacterial UTIs in females	3.0
i. Describe diagnostic methods for bacterial UTIs	3.0
j. Identify the treatment recommended for bacterial UTIs	3.0
k. List viral and parasitic agents of UTIs	1.5
2. Common causes of urinary tract infections:	
a. Aerobic gram-negative rods, esp.	3.0
(1) Uropathogenic *Escherichia coli*	3.0
(2) *Pseudomonas aeruginosa*	3.0
(3) *Klebsiella*	3.0
(4) *Proteus*	3.0
(5) *Staphylococcus* sp., esp. *S. saprophyticus*	3.0
(6) *Enterococcus* sp.	2.0
3. Less common causes of urinary tract infections	
a. Adenovirus-hemorrhagic cystitis	2.0
b. *Schistosoma haematobium*-schistosomiasis (blood in urine, associated with rural Africa)	1.0
F. GENITAL TRACT	
1. Syphilis	
a. Describe structural and cultural characteristics of *Treponema pallidum*	3.0
b. Describe the epidemiology and pathogenesis of syphilis, including primary, secondary and tertiary manifestations of the disease	3.0
c. Define congenital syphilis and describe its manifestations and prevention	3.0
d. Define neurosyphilis and describe its manifestations	3.0
e. Describe the mode of transmission of the disease	2.5
f. Describe methods for the diagnosis of syphilis	3.0
g. Explain the difference between non-specific and specific serological tests for syphilis and the pattern of the immune response vis-à-vis these tests in treated and untreated cases	3.0
h. Identify antibiotics of choice in treating syphilis	3.0
2. Gonorrhea	

a. Describe structural and cultural characteristics of *Neisseria gonorrhoeae*	3.0
b. List the virulence factors associated with Neisseria gonorrhoeae	3.0
c. Describe modes of transmission of gonorrhea	3.0
d. Describe the diagnosis and treatment of gonorrhea	3.0
e. Distinguish between a diagnosis of gonococcal and non-gonococcal urethritis	2.5
f. Describe disseminated gonococcal infections and distinguish them from gonococcal infections of the eyes and throat.	2.5
g. Describe the mechanisms of acquired penicillin resistance and alternative drugs for treating resistant strains	2.5
h. Explain the importance of phase and antigenic variation in pathogenesis of *Neisseria gonorrhoeae*	2.5
i. Appreciate that *Neisseria gonorrhoeae* infections can lead to pelvic inflammatory disease in women	3.0
3. Non-gonococcal urethritis	
a. List the causative agents of non-gonococcal urethritis	3.0
b. Distinguish between a diagnosis of gonococcal and non-gonococcal urethritis	2.5
c. Describe the life cycle and unique properties of *Chlamydia trachomatis*	2.0
d. Describe structural and cultural characteristics of *Ureaplasma urealyticum*	1.0
e. Describe structural and cultural characteristics of *Mycoplasma genitalium*	1.0
f. Describe the diagnosis and treatment of non-gonococcal urethritis	2.5
g. Appreciate that these bacteria can also cause pelvic inflammatory disease in women	2.5
h. Describe the characteristics of l*ymphogranuloma venereum*	1.0
i. Describe the causative agent of lymphogranuloma venereum (LGV) and *Chlamydia trachomatis*	1.0
j. Describe the clinical progress and symptoms of LGV	1.0
k. Explain the recent increase in LGV cases among travelers to Asia	1.0
l. Describe the diagnosis and treatment of LGV	1.0
m. Granuloma inguinale	1.0
n. Describe structural and cultural characteristics of *Klebsiella* (*Calymmatobacterium*) *granulomatis*	1.0
o. Describe the pathogenesis and symptoms of granuloma inguinale (GI)	1.0
p. Describe the diagnosis and treatment of GI	1.0
q. What is chancroid (soft chancre)	2.0
r. Describe structural and cultural characteristics of *Hemophilus ducreyi*	1.0
s. Describe the pathogenesis and symptoms of chancroid	1.0
t. Describe the diagnosis and treatment of chancroid	1.0
u. Appreciate how symptoms of chancroid can be confused with those of primary syphilis, LGV, GI, or genital herpes	2.0
4. Trichomoniasis	
a. Describe characteristics of the protozoan *Trichomonas vaginalis*	3.0
b. Describe symptoms associated with trichomoniasis	3.0
c. Describe the diagnosis and treatment of trichomoniasis	3.0
5. Bacterial vaginosis	
a. List the four signs associated with non-specific vaginitis	2.5
b. Describe the organisms associated with bacterial vaginosis (BV)	1.5
c. Describe the diagnosis and treatment of BV	2.5
6. Vulvovaginal candidiasis	
a. Describe the structural and cultural characteristics of *Candida albicans*	3.0
b. Explain how candida can cause disease as a member of normal human flora	3.0
c. Describe the diagnosis and treatment of vulvovaginal candidiasis	3.0
7. Genital herpes	
a. Describe the virion and genome structure of herpes simplex type 2 (HSV-2)	2.0
b. Describe the transmission and pathogenesis of HSV-2 infections	3.0
c. Discuss the concept of viral latency/reactivity and its significance with respect to genital herpes infections	3.0
d. Describe the current strategies for preventing and treating HSV-2 infections	2.0
8. Genital warts	
a. Describe the virion and genome structure of human papillomavirus (HPV)	3.0
b. Describe the transmission and pathogenesis of HPV	3.0

c. Appreciate the association of cervical cancer with certain types of HPV infections	3.0
d. Describe methods for detection and treatment of HPV infections	3.0
9. Cytomegalic inclusion disease	
a. Describe the virion and structure of human cytomegalovirus (HCMV)	2.0
b. Describe the epidemiology and pathogenesis of HCMV	3.0
c. Appreciate that primary HCMV infection in a healthy individual is clinically inapparent but in adults can lead to a Mononucleosis syndrome	2.0
d. Appreciate that HCMV causes the most common intrauterine viral infection and that cytomegalic inclusion disease in pregnant women can cause fetal death or damage to liver, spleen, blood-forming organs and nervous system	3.0
10. Other sexually transmitted diseases	
a. Appreciate that other organisms can be sexually transmitted without causing disease in the genital tract	2.0
b. Describe the genome, pathogenesis and transmission of hepatitis b virus	3.0
c. Describe the transmission and life cycle of the human immunodeficiency virus (HIV)	3.0
G. MUSCULOSKELETAL INFECTIONS	
1. Myositis	
a. Name the most common infectious cause of myositis.	1.0
b. How does coagulase help *Staphylococcus aureus* evade host immunity?	1.0
c. What are other important virulence factors for this pathogen?	1.0
d. How do these factors contribute to the virulence of the organism?	1.0
e. Name an important cause of myositis is *Staphylococcus aureus*	1.0
2. Necrotizing Fasciitis	
a. Name the infectious causes of necrotizing fasciitis.	3.0
b. Explain the pathogenesis of necrotizing fasciitis.	3.0
c. How do the pathogens' virulence factors contribute to the pathogenesis?	3.0
d. Why is surgery, even amputation, often necessary in the treatment of necrotizing fasciitis and antibiotic therapy alone inadequate?	3.0
e. Name an important cause of necrotizing fasciitis is *Streptococcus pyogenes* (Group A Strep)	3.0
3. Osteomyelitis	
a. Name the most important infectious causes of osteomyelitis.	2.5
b. What are important virulence factors for these pathogens?	2.5
c. How do these factors contribute to the virulence of the organisms?	2.5
d. Describe the routes by which various microbes gain access to bone (hematogenous spread, trauma-wound, prosthetic device, etc..) and be aware that lesions are often polymicrobial.	3.0
e. Explain why surgical debridement and prolonged bactericidal antibiotic therapy are needed in chronic osteomyelitis.	2.5
f. Explain how laboratory procedures could distinguish between these various bacteria.	2.5
g. What procedures, media & biochemical assays would be necessary to distinguish between these pathogens?	2.5
h. Important causes of osteomyelitis are:	
(1) *Staphylococcus aureus* (common)	3.0
(2) Group B streptococcus (common)	2.5
(3) *Escherichia coli* (common)	2.5
(4) *Pseudomonas aeruginosa*	1.5
(5) *Enterococcus* sp.	1.0
(6) *Salmonella* sp. (in Sickle Cell disease patients)	3.0
4. Gas Gangrene	
a. Name the most important infectious cause of gas gangrene.	3.0
b. How is *Clostridium perfringens* identified from gangrenous tissue and how is the disease diagnosed and treated?	3.0
c. What are important virulence factors for c. Perfringens? How do these factors contribute to the virulence of the organism?	3.0

d. Reconcile why gas gangrene is so infrequent despite the presence of relatively large amounts of the organism in human intestines and pervasive presence in soil. 1.0
e. Explain why wounds are important in the pathogenesis of gas gangrene. 3.0
f. Name an important cause of gas gangrene (*Clostridium perfringens*) 3.0

5. Tetanus

a. Explain why wounds are typically necessary for tetanus. 3.0
b. How can tetanus be prevented? 3.0
c. Why is honey dangerous for infants? 1.5
d. What are important virulence factors for *Clostridium tetani*? 3.0
e. How do these factors contribute to the pathogenesis of tetanus? 3.0

6. Skin and Soft Tissue Infections

a. Define abscess, boil, carbuncle, furuncle, folliculitis, pyoderma (impetigo), erysipelas, cellulitis. 3.0
b. Define macule, papule, plaque, pustule, vesicle, bulla. 3.0
c. For the paired diseases and pathogens in the chart below:
(1) Describe the clinical case setting in which the disease would be found. 3.0
(2) Describe the microbial pathogens known to cause the disease. 2.0
(3) Describe the pertinent microbial structures related to virulence (virulence factors, including toxins). 3.0 2.0
(4) Describe the pertinent biochemical pathways related to pathogen virulence. 3.0
(5) Describe the epidemiology of the disease. 3.0
(6) Describe the etiology/ pathogenesis of disease. 2.0
(7) Describe clinical aspects of the disease. 2.0
(8) Describe the immune response to pathogens causing the disease. 3.0
(9) Describe methods of diagnosis of the disease. 3.0
(10) Describe current therapy (and antibiotic resistance) for the disease. 3.0
(11) Describe methods of prevention of the disease.

PATHOGEN	DISEASE	
Staphylococcus aureus	Scalded skin syndrome, carbuncle, furuncle, folliculitis, impetigo, wound infection, toxic shock syndrome	3.0
Streptococcus pyogenes (Group a strep)	Impetigo, erysipelas, cellulitis, necrotizing fasciitis, gas gangrene, scarlet fever, toxic shock syndrome	3.0
Clostridium perfringens	Gas gangrene	3.0
Clostridium tetani	Tetanus (local infection but systemic toxin)	3.0
Propionibacterium acnes	Acne	2.0
Mycobacterium leprae	Leprosy	1.0
Treponema pallidum	Syphilis	3.0
Treponema sp.	Yaws, pinta	1.0
Borrelia burgdorferi	Lyme disease rash	3.0
Rickettsia rickettsii	Rocky mountains spotted fever rash	3.0
Rickettsia prowazekii	Epidemic typhus	1.0
Rickettsia typhi	Endemic typhus	1.0
Erysipelothrix rhusiopathiae	Erysipeloid	1.0
Nocardia	Cutaneous nocardiosis	2.0
Superficial and cutaneous mycoses		
Malassezia furfur	Tinea versicolor	3.0
Microsporum, trichophyton, epidermophyton	Tinea corporis, tinea pedis, tinea cruris, tinea nigra, onychomycosis	3.0
SUBCUTANEOUS MYCOSES		

(INTRODUCED BY TRAUMA)		
Sporothrix schenckii (3.0)	Sporotrichosis	3.0
Phialophora and *Cladosporum*	Chromomycosis	1.0
Petriellidium and *Madurella*	Mycetoma	1.0
SYSTEMIC MYCOSES WITH SKIN MANIFESTATIONS		
Coccidioides immitis	Coccidioidomycosis	3.0
Cryptococcus neoformans	Cryptococcosis	3.0
Blastomyces dermatiditis	Blastomycosis	2.0
PARASITES		
Leishmania tropica	Cutaneous leishmaniasis	3.0
Leishmania braziliensis	Mucocutaneous leishmaniasis	1.0
Hookworms *(Ancylostoma and Necator)*	Cutaneous larva migrans	2.0
Onchocerca volvulus	Onchocerciasis	1.0
VIRUSES/DISEASE	**PRESENTATION**	
Papilloma viruses	Warts	3.0
Poxvirus – molluscum contagiosum	Fleshy papules	2.0
Herpes simplex, coxsackievirus	Vesicles	3.0
Measles, rubella, dengue, parvovirus B19	Maculopapular rash	3.0

H. INFECTIONS OF THE NERVOUS SYSTEM

1. Principles	
a. Differentiate meningitis from encephalitis.	3.0
b. Name the 2 organisms that cause 80% of cases of bacterial meningitis beyond the neonatal period.	3.0
c. Name the common causes of bacterial meningitis in infants less than 1 month of age.	3.0
d. Describe host factors that may increase the risk for bacterial meningitis.	2.0
e. Describe the methods of acquisition of the organisms causing bacterial meningitis.	2.5
f. Define aseptic meningitis.	2.5
g. Describe the clinical signs and symptoms, pathogenesis, diagnostic techniques, and treatment options for bacterial, viral, and fungal meningitis and encephalitis.	3.0
2. Describe the Bacterial Causes of Meningitis/Encephalitis	
a. *Streptococcus pneumoniae*	3.0
b. *Neisseria meningitidis*	3.0
c. *Hemophilus influenzae*	2.0
d. Group b strep	3.0
e. *Escherichia coli*	2.0
f. *Klebsiella* sp.	2.0
g. *Listeria monocytogenes*	2.5
h. *Mycobacterium tuberculosis*	2.0
i. *Treponema pallidum*	2.0
3. Describe the Viral Causes of Meningitis/Encephalitis	
a. Enteroviruses	3.0
b. Mumps	1.0
c. Arboviruses	3.0
d. Lymphocytic choriomeningitis	1.0
e. Herpes viruses	3.0

f. Influenza viruses	1.0
g. HIV	2.0
h. CMV	1.0
i. Rubella	1.0
4. Describe the Fungal Causes of Meningitis/Encephalitis	
a. *Candida albicans*	1.0
b. *Cryptococcus neoformans*	2.0
c. *Coccidioides immitis*	1.0
d. *Histoplasma capsulatum*	2.0
5. Parasitic Causes of Meningitis/Encephalitis	
a. *Toxoplasma gondii*	2.0
b. *Plasmodium falciparum*	1.0
c. *Acanthamoeba* sp.	1.0
d. *Naegleria fowlerii*	1.0
I. DEGENERATIVE BRAIN DISEASES	
1. Principles	
a. Describe the pathophysiology of subacute sclerosing panencephalitis and progressive multifocal leukoencephalopathy.	2.0
b. Define prion disease.	2.5
(1) Describe the course of Creutzfeldt-Jakob disease.	2.0
(2) Describe the relationship of bovine spongiform encephalopathy to its associated human disease.	2.0
J. ZOONOTIC DISEASES	
1. Describe the Etiologic Agents	
a. *Yersinia pestis* (PLAGUE)	2.5
b. *Pasteurella multocida* cellulitis (cat/dog bite)	2.0
c. *Francisella tularensis* (tularemia)	2.5
d. *Bartonella* (cat scratch)	2.5
e. *Brucella* sp.	1.5
f. *Leptospira*	2.0
g. *Ehrlichia*	2.0
h. Anaplasmosis	1.5
i. *Borrelia hermsii* (relapsing fever western U.S.)	2.0
j. Rabies	3.0
k. Viral hemorrhagic fever	1.0
K. OPPORTUNISTIC INFECTIONS	
1. Describe the Etiologic Agents	
a. *Enterobacter*	1.5
b. *Vibrio vulnificus*	2.0
c. *Aeromonas* sp.	1.5
d. *Hemophilus influenzae* (non-typeable)	2.0
e. *Eikenella corrodens*	1.5
f. *Pseudomonas aeruginosa*	3.0
g. *Actinomyces*	2.0
h. *Bacteroides*	2.5
i. *Fusobacterium*	2.0
j. *Prevotella*	2.0
k. *Porphyromonas*	2.0
l. *Peptostreptococcus*	2.0

*Represents values of *'3'* for information that is essential knowledge to be included, *'2'* for information that is important knowledge to be included if there is time in the curriculum and *'1'* for information that is trivial knowledge not required in a curriculum on Pathogenesis/Infectious Diseases.