

**The Graduate College
Rush University**

Integrated Biomedical Sciences MS Program



**Program Manual
2018-2019**

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IBS MS Program Director**

MS in Integrated Biomedical Sciences
 Graduate College, Rush University
 A Manual for Students and Faculty
 Academic Year 2018-2019

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Rush University MS in Integrated Biomedical Sciences

Program Description:

The Master of Science in Integrated Biomedical Sciences program is designed to educate science professionals to have productive careers in research and academic positions, as well as for career advancement opportunities within their specialized fields. Graduates of this program will go on to perform high quality, impactful biomedical research at colleges and universities, government agencies, hospitals, non-profit agencies and industry. Students in the program will work with faculty and scientists to generate new knowledge in the fields of biomedicine through the application of sophisticated research methods. This degree is intended to offer students an intermediate step in a career path, provide research experience to supplement their primary professional path or provide supplementary training for other reasons.

The MS in Integrated Biomedical Sciences is designed to be completed within two years and requires completion of **37 semester hours (SH)** of credit, which is distributed as core courses (20SH), track specific cognates (7SH), electives (4SH), and thesis research (6SH), creation of a thesis containing original research and defense of the thesis. The core curriculum, which will be common to all students, builds knowledge and skills in research theories and methodology, data analysis and statistics, laboratory applications and skills, and the molecular and cellular sciences basic to health and disease. These courses will provide systematic exposure to the contemporary process of scientific discovery and will serve as the basis for the remainder of the curriculum. In addition, students will be required, in conjunction with their advisors, to select from concentration focused cognates in one of five tracks: Translational Cancer Research; Cardiovascular and Respiratory Biology; Immunity, Infection & Inflammation Research; Function & Disorders of the Musculoskeletal System; and Function & Disorders of the Nervous System. All students will be required to participate in track specific Advanced Topic Seminars in terms 4 and 5. All students will also be required to complete a minimum of 6SH dedicated to completion of their thesis research project. Thesis research hours are consistent across all of the tracks within the Integrated Biomedical Sciences MS program and encompass laboratory research time required for completion of the thesis including: analyzing published data, developing a research proposal, learning and applying advanced methodologies and statistical data analyses, developing skills to write a project proposal, practicing presentation skills to disseminate own research findings, and developing, writing, presenting and defending a thesis project.

Program Goals:

- (1) Develop individuals who can formulate appropriate research questions, organize and test hypotheses, interpret the findings in a robust manner, and apply research results to biomedical sciences.
- (2) Prepare competent Biomedical Science professionals at the Masters level with interdisciplinary knowledge and experiences who can successfully function within inter-professional research teams in areas from basic science to healthcare delivery.
- (3) Provide specialized training in professional skills development enabling the graduates to function as competent and capable researchers, educators and managers in their specialized field.
- (4) Enable students to find satisfying careers in the biomedical sciences. Students will create Individual Development Plans (IDP) to better define their areas of interest including teaching, administration, research in industry, or academic research during

the first year and will continue to actively explore career opportunities and create career development goals throughout the rest of their graduate career. This will include use of the AAAS IDP website, attendance at special seminars and events hosted by the graduate program at Rush and other nearby Institutions, and active pursuit of opportunities that will provide exposure to experiences related to career goals. Upon graduation, the graduate will use the IDP to refine their career interests and will have benefited from mentorship and training opportunities in their chosen career paths.

Student progress will be evaluated and monitored by the faculty advisor and an interdisciplinary Thesis Committee that is comprised of Rush Graduate College faculty members with expertise in the selected area of research. The completion and defense of a research thesis is required for degree conferral. Recipients of the MS degree in Integrated Biomedical Sciences will demonstrate the capability to conduct rigorous, impactful research that contributes to and expands knowledge in the biomedical sciences.

Student Learning Outcomes:

Graduates of the Master of Science in Integrated Biomedical Sciences will be able to:

- (1) Contribute to the body of knowledge in the biomedical sciences through critical inquiry, scientific reasoning and scholarly pursuits
- (2) Conduct research that adheres to ethical principles and professional standards
- (3) Collaborate with multidisciplinary teams in the design, conduct of experiments and contribute to the dissemination of biomedical research

Student achievement will be assessed through a variety of direct and indirect performance measures that include but are not limited to: examinations; participation in class discussions and seminars; written assignments; journal club presentations; outside scientific presentation; laboratory skill demonstrations; student, alumni and employer surveys; research thesis completion; and roles and contributions within the academic and scholarly community. Student progress will be assessed based on their meeting specified milestones and myIDP professional development goals throughout the course of the program.

Registration:

Initial registration can occur in the months preceding the start of classes.

Subsequent registrations: Registration times for continuing students are specified in the University timetable (available from the Registrar's website). The student will also be notified when registration is due by email and is required to register within the indicated timeframe. This is usually 2-3 months before the start of classes. **There is a \$50 fee for late registration.** There is an additional late fee of \$50 if the students register after classes start. **Do not delay registration for consultations, as you will incur late fees. Students may change their registration after consultation using Drop/Add privileges, which will not incur a fee.**

In the first year, students should register for the courses listed in the year 1 curriculum. Questions should be addressed to the Assistant Program Director. Students with advance standing should meet with the Assistant Program Director prior to registration in order to determine individual course schedules.

Students who have progressed to the point where they are eligible to select a track are required to discuss course selection with their Track Director prior to registration.

Immediately following online registration, all students must submit a paper copy of their registration to the Graduate College Office.

Expectations:

Students are expected to become independent critical thinkers who are able to effectively present their research in oral and written formats.

The Graduate College requires that all students remain in good academic standing by maintaining at least a “B” (3.0) average. Failure to do so will result in the student being placed on probation and, if the student does not regain a B average following one semester of probation, they may not be permitted to register for subsequent courses or semesters without the approval of the Committee on Progress and Promotions. For more information see the Academic Standing section of the University Catalog in the appendix.

All outside employment is strongly discouraged and requires express approval by the Graduate College. This excludes activities that would be in line with IDP goals like tutoring, teaching and proctoring.

Students are expected to conduct themselves in a professional manner. This includes respecting the rights of others and being kind and courteous to students, faculty and patients. Intimidation of other students and staff will not be tolerated and is grounds for dismissal. All students are expected to sign and abide by the University Honor Code. Actions that violate the University Policy on Academic Honesty and Student Conduct (see appendix) should be reported to the Honor Code committee. The Honor Code is also listed in the appendix along with the bylaws for the Honor Code Committee. The Graduate College Honor Code Committee bylaws contain information on reporting infractions and how such reports are handled.

Sexual harassment as well as harassment related to race, color, religion, sexual orientation, national origin, ancestry, age, marital or parental status, or disability is prohibited. The University Catalog details the policies regarding inclusion of minorities and those with disabilities as well as the policies and procedures for reporting harassment. All issues regarding sexual harassment as well as harassment related to race, color, religion, sexual orientation, national origin, ancestry, age, marital or parental status, or disability are covered under University Policy and are referred directly to the Office for Equal Opportunity (See University Harassment policy in the appendix).

Students working with laboratory animals must follow IACUC guidelines and will be subject to disciplinary action in the case of abuse.

Evaluation:

The Graduate College is actively evaluating the program. Please respond to surveys and to course evaluations in a thoughtful and constructive manner. Such good faith participation is needed to help us improve the educational experience and outcomes for both current and future students. In order for such changes to be beneficial, we need honest and professional feedback from you.

Changes to the Program:

You may be affected by changes to the program during your studies due to changes in policy or course offerings. Changes in policy may be necessary to bring the program in line with changes in Graduate College and or University policies. You will be notified in writing or by email of any program changes, and such notification will override the contents of this manual.

Changes to the curriculum may include revision of courses or the addition or deletion of particular courses. Students will be notified in writing or by email of the changes and may be required to take the new or replacement courses. However, curriculum changes will not be made retroactively. For example, changes in the first year curriculum will not affect second year students. Likewise, students who have already taken a course will not be required to take the replacement for that course unless that course has changed substantially.

Organization of the Integrated Biomedical Sciences MS Program

Program Directors:

The IBS Program Director chairs the IBS Education Committee, oversees the Track Directors and is ultimately responsible for the overall execution of both the PhD and MS programs. The MS Program Director is primarily responsible for the MS program. The MS Program Director will create and update an individual course plan with each student and approve your registration during the first year. (The Program Director fulfills the above roles for the PhD students.) The MS Program Director works with the IBS education committee to clarify and update policies and procedures, monitor the progress of first year MS students, and interactively works with the Track Directors to help MS students find rotation and thesis mentors, track placement and appropriate track-specific cognate courses to fulfill their requirements. Both the Program Director and MS Program Director are very interested in feedback concerning curriculum and student life issues. Your input helps us do our jobs better!

As the student moves into the second year, the Track Directors and the Research Advisor are responsible for advice and consultation regarding course registration and programs of study. However, the Program Director and MS Program Director remain available for student consultation and may be contacted directly.

Associate Program Director:

The Associate Program Director is concerned with the implementation and evaluation of the program. If the Program Director is not available the Associate Program Director will temporarily assume the duties of the Program Director.

Track Directors:

The Tracks are: Translational Cancer Research (CAN); Cardiovascular and Respiratory Biology (CVR); Immunity, Infection & Inflammation Research (III); Function & Disorders of the Musculoskeletal System (MSK); and Function & Disorders of the Nervous System (NEU).

The Track Director will help oversee the students once they have chosen their track to assure there is timely progression towards degree completion within 2 years. This includes ensuring that the student follows a proper plan of study fulfilling track requirements, cognates and electives; assuring that the student has an IDP and that the selection of courses meets their objectives; and helping the student in the formation of a thesis committee that meets as required by the Graduate College. The Track Director will work in close communication with the Research Advisor to assess student progression by participation in meetings of the thesis committee, specifically the proposal and thesis presentation. If deemed necessary, additional meetings between the student, Advisor and Track Director may be held to assess student progress and ensure completion of program requirements.

Integrated Biomedical Science Education Committee:

The IBS Education Committee is composed of the Program Director, the Associate Program Director, the MS Program Director, the Track Directors, a student representative, and two faculty representatives. The IBS Education Committee is chaired by the Program Director. In the event of the absence of the Program Director the Associate Program Director should chair the meeting. This Committee appoints an Admission Committee that makes recommendations for admission to the Dean of the Graduate College. The Committee evaluates student feedback and recommends changes to the curriculum. The IBS Education Committee also reviews and approves rotation, track, and mentor requests, updates the program policies and procedures, evaluates the results of the qualifying exams for the PhD students, hears appeals on the remediation of grades and complaints concerning academic dishonesty, non-professional behavior and student misconduct that are not covered in the University Harassment Policy. All issues regarding harassment are referred directly to the Office of Equal Opportunity of the University.

Graduate College Council

The Graduate College Council is the senior representative body for The Graduate College. The Council is comprised of all Program Directors, an elected representative from each program/division, and three elected student representatives. Only elected members are allowed to vote. The Graduate College Council considers issues and topics such as the Graduate College Curriculum that affect all graduate programs. The Graduate College Council meets monthly on the first Thursday and is chaired by a Graduate College faculty member.

Graduate College Student Council (GCSC)

President: Matt Russo, Matthew_L_Russo@rush.edu

Vice President: Diptaman (Neal) Chatterjee, Diptaman_Chatterjee@rush.edu

Faculty Advisor: Dr. Marcus Wimmer, Markus_A_Wimmer@rush.edu

The Graduate College Student Council is an open forum, whereby any student member of the Graduate College (PhD or MS) can attend meetings held by the elected graduate student representatives. These meetings are for the students to discuss concerns related to the graduate college, including the curriculum, insurance, academic matters, fund raising, degree requirements, professional development opportunities, as well as community engagement. All issues raised at these meetings will be brought to the attention of the Graduate College Council. Also, the GCSC coordinates awards given to the faculty by the graduate students (Mentor of the Year and Excellence in Teaching Award). We encourage all students to participate because your input is essential. Students serving on the council are provided the opportunity to take an active role in college governance; serving as liaisons who interact between the student body, the Graduate College, and the University.

Also, as part of a community of science professionals, the Graduate College Student Council should partake in university or community outreach programs such as fund-raisers, elementary school science demonstrations, and philanthropic work deemed appropriate by the members of the council that year. In more recent years, the GCSC have instituted the *Emerald Event*, their largest fund-raiser for the students of the

Graduate College to attend scientific conferences and present their research. Since its beginning, the GCSC has raised money to pay for well over 50 travel awards. The GCSC has also helped in various charitable endeavors which raise money for research; as well as university-sponsored fund-raisers including golf-outings, wine and cheese art auctions, and smaller projects throughout the year. The GCSC also organizes several social outings from BBQs to whirly ball competitions, to intra-departmental sporting competitions.

Contacts and Communication:

Integrated Biomedical Science Program Administration

IBS Program Director:	Animesh Barua, PhD (312) 942-6666	Animash_Barua@rush.edu Cohn 410
Associate Program Director:	Ed Barker, PhD (312) 942-3136	Edward_Barker@rush.edu Cohn 620
IBS Master's Program Director:	Amanda Marzo, PhD (312) 942-7268	Amanda_marzo@rush.edu Cohn 514
Track Director (TCR):	Jeffrey A. Borgia, PhD (312) 942-7837	Jeffrey_A_Borgia@rush.edu Jelke 1414
Track Director (CVR):	Kathrin Banach, PhD (312) 563-3553	Kathrin_Banach@rush.edu Jelke 1577
Track Director (III):	Amanda Marzo, PhD (312) 942-7268	Amanda_marzo@rush.edu Cohn 514
Track Director (MSK):	Tom Schmid, PhD (312) 942-3051	Tom_Schmid@rush.edu Cohn 524
Track Director (DNS):	Dan Nicholson, PhD (312) 942-5418	Daniel_Nicholson@rush.edu Jelke 1474

Graduate College Administration

Dean:	Andrew Bean (312) 942-3588	Andrew_Bean@rush.edu AAC 438
Assoc. Dean:	Marenda Wilson-Pham	
Assoc. Dean:	Gabriella Cs-Szabo, PhD (312) 942-2255	Gabriella_Cs-Szabo@rush.edu AAC 438

Students will be given a Rush email account, which will be used for communication from Rush University, the Graduate College and others involved in the program. **This is our official way of contacting students and it is important to monitor this account.** An email sent to this account may notify students of program, scheduling or registration changes and constitutes **official notification**. It is essential that students make sure Rush accounts are working and check it regularly.

Curriculum for 2018-2019 IBS MS Students

Course number	Course Title	Semester hours
Year 1: Fall 2018-19		
BTN 525	Experimental Design and Models of Disease	2
GCC 501	Molecular Biology and Human Genetics	3
GCC 502	Cellular Biochemistry: Proteins, Transport and Signaling	2
GCC 503	Functional Cell Biology	2
GCC 511*	Advanced Readings in Molecular Biology	1
GCC 512*	Advanced Readings in Cell Biochemistry and Cell Biology	1
GCC 505	Techniques in Biomedical Sciences	2
GCC 530	Laboratory Rotations I	1
Spring 2018-19		
GCC 504	Functional Tissue Biology	3
GCC 506	Research Ethics	1
GCC 507	Biomedical Statistics	2
GCC 508	Writing Practicum	2
GCC 533	Laboratory Rotations II	1
	**Track Specific Cognates (1-4)	v
Summer 2018-19		
GCC 599	Thesis Research	2
Year 2: Fall 2019-20		
GCC 694-698	Advanced Topic Seminar (track specific)	1
GCC 599	Thesis Research	2
	* Electives (1)	v
	**Track Specific Cognates (2-4)	v
Spring 2019-20		
GCC CC 694-698	Advanced Topic Seminar (track specific)	1
GCC 599	Thesis Research	2
	** Track Specific Cognates (2-4)	v
	* Electives (0-1)	v

GCC = Graduate Core Curriculum – These classes are taken by MS and PhD students from a variety of different graduate programs. These provide a basic understanding in the Biomedical Sciences and acquaint the students with the biomedical literature. Classes marked with a # can count towards core or electives.

BTN = Biotechnology courses- Taken with the Biotechnology students. Course descriptions can be found in the Graduate Catalog.

* **Electives:** GCC 511 and 512 are reading courses associated with GCC 501, 502, and 503.

**** Track Specific Cognates** - actual course numbers will vary with elective or cognate required by the track. Consult with your Track Director and Research Advisor.

Note: The Integrated Biomedical Sciences graduate program may revise courses and the student may be required to take the replacement courses. Such a requirement does not apply to students who have already taken a course.

MS Program Progression:

Year 1 Classes

The goal of the coursework in the first year is to expose the students to the biomedical sciences in a logical progression and to provide tools for approaching their future research experience. The reading courses provide a critical understanding of the literature and existing base of knowledge. They will also show the student how new knowledge in these areas can help us understand diseases and use this information to identify new therapeutics. This broad-based approach to disease is the core of the Integrated Biomedical Sciences program.

Year 1: Research Experience, Advisor and Research Track Selection

During the first year the student will typically have 2 lab rotations in different laboratories. The laboratory rotations will expose the student to diverse research environments and allow them to assess their 'fit' in a particular laboratory/mentor situation. Students are expected to learn techniques and attend all scheduled experiments, lab meetings, mentor-student discussions, etc. Based on these rotations, students will submit the name of a potential advisor and their track choice to the Integrated Biomedical Sciences Education Committee. The Integrated Biomedical Sciences Education Committee, in consultation with the potential advisor, will approve advisor-student matches. Specific research projects will be determined by the thesis advisor after advisor-student discussions. If a student cannot choose a thesis advisor based on their first two laboratory rotations, a third rotation may be taken (Spring or Summer). The selection of a research advisor and project will determine the student's selection of a research track.

Year 2: Classes, Research Experience, Thesis Committee, Thesis Proposal and Thesis Presentation

Any classes will be dictated by the track cognates and electives available that academic year. Course selection should complement research activity and the interests of the student and should help prepare him or her for the career choice identified through the use of the Individual Development Plan (IDP) website and additional resources provided by the Graduate College. When the student is not in class or studying, the student should be working on his or her research project.

The student's assessment at this time relates to the following student outcomes:

- The graduate is able to acquire research skills, collect and analyze data, and interpret results in order to address an original research question.

In addition, this step begins the continuing assessment of the following outcomes:

- A graduating student is capable of independent critical thinking and writing as well as proposing, performing and effectively presenting his or her research.
- The graduate is able to work collaboratively with other scientists, physicians and health care professionals to give and obtain feedback concerning the approach to research problems, data analysis and implications of research.

The student creates an Individual Development Plan (IDP) to better define his or her areas of interest including teaching, administration and research in industrial or academic environments or further professional education. Upon graduation, the graduate will have used the IDP, mentorship and training opportunities to refine his or her career path.

In consultation with their advisor, the student chooses a Thesis Committee consisting of the advisor, and two additional Graduate College faculty members. Committee members should be familiar with either the research area or crucial technical aspects of the student's project. Each student will write a succinct research project proposal, which will be presented, to the committee for approval. The proposal serves to keep the student focused on achieving the aims of their project and allows the committee to track student progress based on the stated aims. Students should view the committee members as a resource for didactic and technical assistance in the conduct of their project.

The student is expected to write a Thesis and present their research to the Rush research community (Thesis Presentation). The Thesis Committee will then meet with the student to address any questions or issues related to the data or format of the Thesis document. The student may be asked to make revisions before final Thesis approval by the committee.

Minimal Credit Hours Required for the Integrated Biomedical Sciences MS Degree

The program is designed to be completed in five consecutive semesters and requires completion of at least **37 semester hours** including:

20 credit hours of core courses,

7 credit hours of track-specific cognates,

4 credit hours of elective courses, and

6 hours of thesis research credit.

The core curriculum focuses on developing knowledge and skills in research theories and methodology, data analysis and statistics, laboratory applications and skills, and the molecular and cellular sciences basic to health and disease. In addition, students will work with faculty advisors to select one area from five available tracks: Translational Cancer Research (TCR), Cardiovascular and Respiratory Biology (CVR), Immunity, Infection and Inflammation (III), Function and Disorders of the Musculoskeletal System (MSK) and Function and Disorders of the Nervous System (DNS). Students in this program will have a research project and will write a thesis and give a thesis presentation at the completion of their project.

The core curriculum, which will be common to all MS students, builds knowledge and skills in research theory and methodology, data analysis and statistics, laboratory applications and skills, and the molecular and cellular sciences basic to health and disease. These courses will provide systematic exposure to the contemporary process of scientific discovery and will serve as the basis for the remainder of the curriculum.

Students will be required, in conjunction with their advisors, to select 7 credit hours of courses from concentration-focused cognates in their chosen track and a minimum of 4 elective credits from the Graduate College courses offered. Finally, students will be required to accrue a minimum of 6 credit hours of Thesis Research. MS students will be encouraged to participate in track-specific Advanced Topic Seminars in their second year. These will count as cognate credits.

Integrated Biomedical Sciences: Academic Policies - MS Program

Academic Advisor/Principal Advisor

The IBS MS Program Director functions as the academic advisor to the students during the first year of matriculation in the program. After 2 rotations, the student will identify the track that he or she wishes to enter and will begin working in the laboratory of his or her research advisor. At this time, the Track Director along with the research advisor will serve as mentors for the student.

Research Advisor Selection

During the first year, the student, in consultation with the MS Program Director and Track Directors in areas related to the student's interests, will select 2 laboratories for research rotations. Based on these rotations, the student will identify the track he or she is interested in and submit the name of a potential advisor to the IBS education committee. The IBS education committee will match students with advisors.

Guidelines for Rotation Reports:

Format: 3 pages maximum

Your report should start with the following information:

- Student
- Rotation Advisor
- Other mentors and their roles in the project
- Dates of rotation
- Number of hours spent in the rotation per week

Body of Report should have the following sections:

- Hypothesis
- Aims
- Introduction/Background
- Methods
- Results
- Discussion/Conclusions

Were you satisfied with the rotation experience?

Rubric for Evaluating Student Performance during Laboratory Rotations – Rotation Advisors should fill out 1-5 and add any comments they wish to add.

Evaluators should use the following scale to rate student performance for items 1-6

A = 4; B = 3; C = 2; F = 1

1. Professionalism:
Student was present at appropriate times and spent an adequate amount of time on their research project _____
 2. Preparedness/participation:
Student showed enthusiasm for learning lab techniques/procedures and prepared by previewing relevant information _____
 3. Literature:
Student reviewed pertinent literature for their project and was able to summarize/discuss with mentor and/or lab personnel _____
 4. Lab Notebooks:
Student was diligent and followed GLP procedures with lab notebook _____
 5. Interpersonal Interactions:
Student interacted/cooperated with mentor and other lab personnel in a positive and constructive manner _____
 6. Final Report: (evaluated by Track Director)
Student submitted final report of rotation experience (no later than 2 weeks post-rotation) and included the required components _____
- Final Rotation Grade** (avg. must be 3.0 or greater to pass) _____

The final rotation grade is given by the Program Director taking the rotation advisor's evaluation, the research report and other related information into consideration.

Integrated Biomedical Sciences: Track/Research Opportunities

The research tracks for the Integrated Biomedical Sciences MS program are Translational Cancer Research; Cardiovascular and Respiratory Biology; Immunity, Inflammation, and Infection; Functions and Disorders of the Musculoskeletal System; and Functions and Disorders of the Nervous System. The tracks include qualified faculty from Rush University Medical Center who have an interest in research in these tracks. They come from academic departments as well as clinical departments, which enables the students to select a variety of individuals with basic and clinical expertise to serve on their advisory committees and guide them through their projects.

Master's Thesis Research Committee

After the student selects a research advisor and begins to collect preliminary data, the student and advisor will select a research committee in consultation with the track director. This committee will advise the student and evaluate his or her proposal and thesis documents. The thesis committee will consist of the advisor and two additional Graduate College faculty members. Committee members should be familiar with either the research area or crucial technical aspects of the student's project. Committee members are intended to be a resource for the student and their advisor to enhance didactic and technical knowledge towards the completion of the student's project. The Track Director (or their designated representative) will serve as an *ex officio* non-signing member of the thesis committee to oversee the procedural aspects of the committee meetings and student progression through the program. The thesis committee will strive for consensus in all its actions; however, a majority vote of the committee's membership is sufficient for all activities and approvals.

Research Proposal

Each student will write a succinct research project proposal (~ 3-10 pages.), which will be presented to the committee for approval. The proposal serves to keep the student focused on achieving the aims of their project and allows the committee to track student progress based on the stated aims. Ideally, proposals should be modeled after the NIH R21 Grant Application; students should consult with their advisors regarding formatting of their proposal. The target date for proposal presentations is within the first 45 days of the start of the Fall semester of year 2; it is also acceptable for the proposal presentation to be held in the summer term between year 1 and 2. The thesis committee evaluates the feasibility and scope of the project and recommends alterations as needed to ensure adequate student progress through the MS IBS program in a timely fashion.

Thesis Document, Presentation and Approval

The student is expected to write a Thesis document (approved by the director of the Library of Rush University Medical Center) and present the work in a public lecture attended by the thesis committee, faculty and students of the University. The thesis committee will then meet with the student in closed session to address any additional questions and to deliberate on approval of the thesis. Typically the meeting immediately follows the public lecture. The student may be asked to make revisions before final thesis approval by the committee.

The registrar's office must be notified of impending completion of the degree by submission of an Intent-to-Graduate form at the beginning of the final semester. As the thesis is reaching final form, the student should consult with the University librarian to assure that the document is formatted correctly. Once the thesis is approved, the student will complete the final checklist to assure that all necessary approvals have been

obtained. Students will be required to have an exit interview and provide feedback concerning their experience at Rush University.

Student Grievance Procedure

A student who is having difficulty with a course should contact his or her course director to discuss the situation and possible avenues of action. A student that anticipates being absent from class or faces an emergency that will impact his or her attendance or performance should contact their course director(s). A student with a problem in laboratory rotations or thesis research should attempt to resolve the issue through direct communication with the laboratory advisor or mentor. A good faith attempt to plan/resolve any issues directly with the course director, laboratory advisor or mentor should always be the first course of action. If this fails, the student should bring the concern to the program director if the student is a first-year student or to their track director if the student has already identified a track. The program director or track director will work with the student and faculty member(s) to resolve the issue. If it is a serious issue, the program director, track director or faculty member may involve the Education Committee. The student may also appeal directly to the Education Committee by requesting in writing that it meet to discuss the issue. The Education Committee will meet within 10 days to render a judgment to best address the interests of the student within the guidelines of the Integrated Biomedical Sciences program.

The Education Committee will also hear complaints concerning academic dishonesty, nonprofessional behavior and student misconduct. The Education Committee may become involved through a direct request from a student or faculty member or by a referral from the Honor Code Committee. If the Honor Code Committee has not been involved, the Education Committee may refer the initial request to the Honor Code Committee. The Education Committee will hear testimony from any involved faculty and/or students. They may recommend remediation or disciplinary measures. Recommendations for dismissal or suspension are made to the office of the Dean of the Graduate College. Appeals will be heard by the Graduate College Council (GCC) or a subcommittee of the GCC that will be organized solely for the purpose of hearing the appeal and making a recommendation to the Dean. The ultimate decision regarding student expulsion or suspension rests in the office of the Dean of the Graduate College.

Graduate College/Rush University Academic Policies

Students must maintain a B average in the first year. If they drop below a B average, they should discuss the possibility of remediation with the director of the course they had difficulty with. The course director may issue an incomplete grade for a limited time in accordance with university policy while agreed-upon remediation takes place. However, once a failing grade (No Pass or letter grade less than B) has been given, the Education Committee must approve a remediation plan. Until a grade is remedied or the average is improved in some other way, the student is on probation. A student who remains on probation for two semesters will be dismissed.

Students who have entered a track must receive at least a B grade in any courses deemed required by their tract director. Failure to remediate a grade of less than B in a required course or a no-pass grade in a pass/no-pass course or the receipt of another such grade while on probation will result in dismissal.

Transferring Credits:

If a student has previously taken courses required for this program, they may either be transferred or requirements may be waved. If a course is transferred, then the student will be given credit for that course. If a requirement is waved, the student may have to take other courses to fulfill the number of required hours of core, cognate or elective hours.

Graduate level transfer credit is subject to approval by the Education Committee based on an evaluation of quality and equivalence by the course director. For graduate level programs, no more than one-third of the total number of required credits with a letter grade may be granted to a student as transfer credit for work done at another graduate institution. Grades may only be transferred if a letter grade of B or better was received.

Transfer credits can only be applied to satisfy the degree requirements of one program. Once applied, they cannot be used a second time for a new degree program. Previously earned program credits at Rush University may only be used to satisfy the requirements of another program if they are at the graduate level and if they meet the current curricular standards.

The number of credits granted for a given course cannot exceed the number awarded for the course on the transcript of the school where the course was taken or the number earned for the corresponding course at Rush University. Credits earned on the quarter system will be converted into semester credits where applicable. A quarter credit is equal to two-thirds of a semester credit (e.g. three quarter-system credits equal two semester credits). Course information (including grades) from transferred courses is not recorded on the student's transcript; only the number of credits is recorded and added to the cumulative number of credits.

In situations where students have advance standing in professional schools including Medical and Dental school, exceptions will be considered on a case-by-case basis.

Dropping Courses:

In year 1, a course may be dropped only with permission from the Program Director. After a student has entered a track, a course may be dropped only with the permission of the Track Director, followed by the approval of the IBS Program Director.

Appeal of Grades:

A student having difficulty with a course who anticipates being absent from class or faces an emergency that will impact their attendance or performance, should contact their course directors. A student who experiences a problem in their laboratory rotations or research should attempt to resolve the issue through direct communication with the Research Advisor. A good faith attempt to plan/resolve any issues directly with the course director or Research Advisor should always be the first course of action. If this fails, the student should bring their concern to the Program Director if they are a first year student or to their Track Director if they have already identified a track. The Program Director or Track Director will work with the student and faculty member(s) to resolve the issue. If it is a serious issue, the Program Director, Track Director or faculty member may involve the Integrated Biomedical Science (IBS) Education Committee. The student may also appeal directly to the IBS Education Committee by requesting in writing that it meet to discuss the issue. The IBS Education Committee will meet within

10 days to render a judgment to best address the interests of the student within the guidelines of the Integrated Biomedical Sciences MS program.

Students must maintain a B average. If a student's cumulative GPA drops below a B average, they will be on academic probation. They should meet with the Educational Committee to determine an appropriate course of action to enable the student to regain a B average. They should also discuss the possibility of remediation with the course director of the course(s) they had difficulty with. The course director may issue an incomplete grade for a limited time in accordance with university policy while agreed upon remediation takes place. If a student's GPA is less than 3.0 for 2 semesters, the Education Committee, who will determine whether or not the student should be dismissed, must review the student's progress. Students who have entered a track must receive at least a B grade in any courses deemed required by their Track. Failure to remediate a grade of less than B in a required course will be grounds for dismissal.

University Appeal Process:

The University has an appeal policy that allows for an appeal beyond the Graduate College. The University Appeal Policy is limited to the following issues: 1) a final course grade, 2) failure on a preliminary or comprehensive examination, 3) failure of the thesis/dissertation that results in his/her academic probation or dismissal from the University or an unreasonable delay in his/her graduation from the University.

The University appeal policy is covered in the "Appeals Procedure" in the University Catalog (included in the appendix).

Dismissal of students:

Grounds for student dismissal include, but are not limited to, the following:

1. Academic deficiencies
2. Inability to find a Research Advisor
3. Insufficient research progress
4. Exceeding the 2-year limitation for the MS degree without permission from the Graduate College
5. Unprofessional conduct or, if in the view of the IBS Education Committee, of if the student is unsuitable for a scientific career.
6. Failure to live up to conditions specified in the letter of acceptance into the Graduate College.

Course Descriptions

Integrated Biomedical Sciences Courses

BTN 525 Experimental Design & Models in Disease

This course will study the role of the experimental model in research. The various aspects of experimental models, computer (in silico) to animal models, will be discussed, building on principles of experimental design. This course requires the student to critically evaluate published work and develop their model for a given disease. Research problems posed by faculty will be understood, developed and solved by students in a cooperative interactive application of computer and library resources. FA [2]

GCC 501 Molecular Biology: Genome to Proteome

DNA structure, replication, recombination, cloning, sequencing and related topics will be covered. This course will continue with organization of the human genome, the cell cycle, genetic mapping and relationships between genes and diseases. Transcriptional and translational regulations will be included. FA [3]

GCC 502 Cellular Biochemistry: Proteins, Transport and Signaling

Concepts of cellular biochemistry, which underlie the structure, organization, and communication of cells will be presented. Protein, carbohydrate and lipid structure and function in cellular organization will be covered. Special emphasis will be placed on the roles of enzymes, signaling systems, receptors and membrane transport systems in cell function. This section will also overview neurons, synapses and neurotransmitters. FA [2]

GCC 503 Functional Cell Biology

The major concepts of cell structure and function will be covered. Topics include tissue origin and organization, extracellular matrix, cytoskeleton, cell-cell adhesion, organelles and compartments, endocytosis, exocytosis, metabolic requirements for signal transduction, cell motility, and regulation of cell proliferation. FA [2]

GCC 504 Functional Tissue Biology

The biochemical and cellular basis for tissue structure and function will be covered. Topics include systems histology and anatomy, immunity, tissue injury and repair/regeneration, regulation of cell-cell adhesion, apoptosis, endocrinology, pharmacology, and toxicology. SP [3]

GCC 505 Techniques in Biomedical Science

This laboratory course will provide a didactic overview and a demonstration of certain laboratory techniques. Topics include electrophoresis, genomics, PCR, tissue culture, cell-sorting techniques, ELISA, chromatography/LC mass spectrometry, imaging techniques, histochemistry and microscopy. Additionally, students will be required to take the relevant Rush University Medical Center safety training modules on LEAP (the on-line training platform) before they can commence any laboratory rotation experience. A series of faculty presentations will be included to help students choose specific laboratories/mentors for their rotations. FA [2]

GCC 506 Biomedical Ethics

The major issues of honesty and fairness as practiced in the scholarly pursuit of new knowledge will be reviewed. Topics include equal opportunity and non-discrimination, abusive relationships, student-faculty relationships, responsibilities of students, faculty, chairpersons and administrators, honesty in writing, authorship, and ownership of data. SP [1]

GCC 507 Biomedical Statistics

This is an introduction to study design and hypothesis testing. Topics include data definition, study design, probability theory, confidence intervals, hypothesis testing, and the techniques used in modern biostatistics. SP [2]

GCC 508 Writing Practicum

This is a hands-on writing course, which focuses on the requirements for abstract, manuscript and grant application writing. Topics include abstract writing, manuscript writing and grant writing. Each topic is covered in several sub-components. SP [2]

GCC 511 Readings in Molecular Biology

Journal club that covers topics related to GCC 501. FA [1]

GCC 512 Readings in Cellular Biochemistry and Cell Biology

Journal club that covers topics related to GCC 502 and GCC 503. FA [1]

GCC 530 and 533 Lab Rotations

Students will gain hands-on experience in a laboratory to provide the student with an understanding of laboratory interests and learn research protocols. The course is repeatable for exposure in different labs. FA [1], SP [1]

FA = Fall and SP = Spring

Where to find more information:

The copy of this manual specifies required courses and the expectations of the program. Keep this manual for future reference. The Integrated Biomedical Science MS program abides by the policies and procedures of Rush University and the Graduate College of Rush University. Policies not detailed here are found in both the Rush University and Graduate College sections of the University Catalog. The University Catalog can be accessed online at the Registrar's site; highly relevant sections are reprinted in Appendix 1 of this document.

Disclaimer:

While we strive for consistency, there may be conflicting information between this document, the University Catalog and the website. Nothing in this manual can override Graduate College or University policies. If you notice discrepancies or have any questions concerning these issues please alert the Program Director. The policies/curriculum in the manual you receive at matriculation (this document) govern your studies. If future changes in policy or curriculum apply to you or your course of study, you will be notified of the changes by email or in writing.

Appendix 1: Excerpts from the Rush University Catalog

The University Catalog specifies the rules that govern the Graduate College and its Programs. Each Program may have additional policies and procedures providing that they do not conflict with those specified in the Catalog.

As a service to students and faculty, the Academic Policies, Academic Standing and Appeal sections of the Catalog have been reprinted in this Appendix. Please Note: Since the University Catalog for the current year is not available until the start of classes, the excerpts given here are from last years catalog. Please check the registrars website for the current catalog.

You are governed by the policies in effect at the time you entered Rush Univ. A copy of the catalog for each academic year is kept on line in a PDF file. A change in the policies can be made provided you are notified in writing or by email.

[The Graduate College: Academic Policies:](#)

The Graduate College adopts college-wide policies and procedures and reviews division regulations. Students follow the college and division policies in effect at the time of initial matriculation in The Graduate College. However, The Graduate College reserves the right to make substantive changes in its programs after the student's matriculation. Students will be informed in writing by the division director of any changes made during their tenure in the program. Students re-entering the college after an absence will be guided by policies and procedures in effect at the time of re-entry.

Examination Policy:

The examination policy is the responsibility of the individual course director who will inform students of examination requirements for that particular course. A period at the end of the quarter is provided for examinations. This period may be used as the course director chooses.

Pass/No Pass Grades:

Each division identifies all courses required of its students. Required courses are usually taken for grade and not under the pass/no pass (P/N) option. Research hours are generally graded using the P/N option. However, a division may opt to provide a letter grade for research classes (under 600) for master's students. The grading policy for post-candidacy research hours (over 600) for doctoral students is P/N.

[Good Academic Standing](#)

To remain in good academic standing, students must maintain a cumulative grade point average of 3.0 and meet the requirements of his/her division. A student must be in good academic standing to be admitted to candidacy and to graduate. Students failing to maintain a GPA of 3.0 will be notified by the Dean in writing that their student status has been changed to "on probation." Students who fail to remediate their deficiencies within one academic year or are placed on probationary status a third time, are subject to dismissal by The Graduate College.

Academic Difficulty

Each division/program has policies and procedures regarding students who fail to maintain good academic standing. While the responsibilities of informing students of their academic problems and of establishing conditions for regaining good academic standing reside within the divisions/programs, the Graduate College Council monitors

the progress and promotion of all students and gives final approval to award students' degrees.

Dismissal

Each division establishes grounds for dismissal beyond the minimal criteria established by The Graduate College. Should a division recommend the dismissal of a student, the director will forward such recommendation to The Graduate College Council for final action. Letters of dismissal come from the Dean. Appeal of a dismissal action begins within the appropriate division.

Full-time Enrollment

Full-time enrollment is required of all Graduate College students with the exception of the Clinical Research students and students within the divisions of Nursing and Health Sciences. Full-time students must register for at least 12 semester hours for the Fall and Spring Semesters and for 9 credit hours for the Summer term of the first year; 10 semester hours thereafter per term are required for full-time enrollment. Students must obtain written permission from the division director for exceptions to this policy. Students receiving a master's degree from The Graduate College as a full-time student must be enrolled for a minimum of two semesters and the summer term; part-time students earning a master's degree must be enrolled a minimum of two semesters per academic year. The minimum requirement for graduation from the college is 53 hours. At the time of graduation, the student must be enrolled in the College. The maximum time allowed for enrollment for a full-time master's degree is four years starting the first semester of official enrollment and for the MS degree is five years.

Residency:

Doctor of Philosophy (MS) candidates are expected to meet all requirements for graduation within five enrolled academic years in the Graduate College (excluding leaves of absence (see below)). This period begins the quarter in which the student formally matriculates. A student exceeding that time limitation must submit to the Graduate Council, in writing, a request to extend their candidacy beyond that time period. This request must identify the reasons for the extension and provide a written plan with reasonable deadlines for completion. This document will be co-signed by the student's advisor and division director. The council will then vote whether to accept the extension or not (passed by simple majority). The student's advisor will then provide an update on the student's progress after six months. One year after the extension is granted, the student is expected to complete all requirements. A second request may be made by the student's advisor and division chair, but only will be accepted through a two-thirds majority of the voting members present at a formal hearing of the Graduate College Council. Within one year of that second request, the student must complete all requirements for the MS degree or face dismissal. Alternatively, the student may be awarded a MS degree upon the recommendation of the student's graduate division.

Readmission:

Any student who has withdrawn from the University or any dismissed student may apply for readmission by submitting an application for this purpose to the Graduate College admission office. An interview may be required. A re-entering student must meet the conditions for re-enrollment stated in his/her dismissal or re-entry acceptance letter and all policies, requirements and course sequence in effect at the time of re-entry. The student will pay tuition and fees at the rates in effect at the time of re-enrollment. Application deadlines may vary by division.

Academic Progression: The graduate division in concert with the rules of the College and Rush University develop specific regulations governing the process that results in final awarding of the degree. While such regulations differ slightly from one division to another, The Graduate College Council reviews each division's program and regulations for approval. In all cases, graduate divisions are required to be explicit and clear about regulations that will affect the candidate. This must be stringently observed in divisional regulations concerning selection of principal advisors, advisory committees, and a plan of study. Similarly, divisions will be explicit and clear concerning academic policies and procedures surrounding qualifying, preliminary, and final examinations when they are required. The divisions are also responsible for providing the candidate with the support needed to plan and conduct the dissertation research. At the same time, a major responsibility of the student is to become familiar with the regulations and expectations of his/her chosen division. These regulations and expectations are included in this Catalog within the sections devoted to each divisional program and are also included within program publications. The student is responsible for understanding the regulations, and monitoring changes that may occur during their tenure in the program.

Student Academic Appeals Policy:

Any student of The Graduate College may appeal a final course grade, failure on a preliminary or comprehensive examination, or failure of the thesis/dissertation that results in his/her academic probation or dismissal from the University. A student may also appeal an unreasonable delay in his/her graduation from the University. No other issues may be appealed through this process. The process for filing an appeal is maintained by each division. The student may request a copy of the Division Appeal Process from the Division Director. This process will be completed within one quarter. If a resolution cannot be achieved at the Division level, the following procedure must be followed. At any step in the process, the student may withdraw the appeal by written notification to the program director with a copy to the Dean. In the event of a dismissal decision, a student may continue to enroll until the appeal process is completed or the student withdraws the appeal.

Step 1: If the student wishes to appeal the decision beyond the Division, within two weeks of receiving a decision from the Division, the student will submit a written statement to the Dean requesting consideration of his/her case by an advisory panel. The student must provide the following in the written statement.

- Course number and grade being appealed or other cause for probation or dismissal, i.e., failure of preliminary/comprehensive examination or thesis/dissertation
- Action being requested
- Justification for the request
- An outline of the efforts and actions already taken to obtain consideration of the request.

The student will send copies of this communication to the Division Director and the Department Chairperson. In addition, if a course grade is being appealed, the student will send a copy to the course director. If the evaluation of a thesis or dissertation is being appealed, the student will send a copy to the chairperson of the thesis/dissertation committee. The Advisory Panel will be The Graduate College Council. Its Chairperson will be appointed by the Dean from among the members. The Division Director of the student's division and any other member who is evaluating the student's academic status will not vote.

Step 2: Within two weeks after notification to the Dean, the Chairperson of the Advisory Panel will arrange a meeting of the advisory panel. It will submit a written recommendation to the Dean.

Step 3: Within two weeks following receipt of the advisory panel's recommendation and upon discussion with the student and with others as appropriate, the Dean shall reach a final decision and notify each party of the decision. The decision reached by the Dean is final.

The issues discussed and the outcomes of all meetings in this appeal process are documented. This record-keeping is the responsibility of a faculty member who is to be designated at each meeting. Copies of the documentation should be distributed to the individuals present at a meeting, to the Division Director, the Dean and to the student's academic file.

Rush University Academic Policies

The Academic Resources and Policies section of this catalog contains additional Rush University academic policies.

[Rush University Statement on Academic Honesty and Student Conduct](#)

Rush University students and faculty belong to an academic community with high scholarly standards. As essential as academic honesty is to the relationship of trust fundamental to the educational process, academic dishonesty violates one of the most basic ethical principles of an academic community, and will result in sanctions imposed under the University's disciplinary system.

Examples of conduct that would subject a student to disciplinary action include but are not limited to:

- All forms of academic dishonesty including but not limited to: cheating; plagiarism; collusion; gaining or seeking unfair advantage in relation to any work submitted; helping others to gain an unfair advantage; removing examination materials from a secure examination area; the unauthorized downloading or copying of examinations that are given on-line; fabricating assigned academic work, including clinical assessments, and presenting them as authentic; facilitating academic dishonesty; unauthorized examination behavior.
- Obstruction or disruption of teaching, research, administration, clinical practice and community outreach or other University/Medical Center activities
- Falsification of student records, transcripts or financial aid forms or applications
- Theft of or damage to University/Medical Center property or the property of a member of the University/Medical Center community
- Threatened or physical abuse of any person or action that threatens or endangers the safety of others
- Misrepresentation, falsification, alteration, or misuse of the University/Medical Center documents, records or identification, or research data
- Unauthorized use or entry of University/Medical Center facilities
- Conviction of a crime deemed serious enough to render the student unfit to pursue his or her profession
- Conduct that is inconsistent with the ethical code of the profession the student is preparing to enter
- Unlawful use or possession of controlled substances on the Medical Center campus

- Unauthorized possession or concealment of firearms or other weapons on medical center premises at any time
- Attempting to gain access to another's e-mail or computer account, username or password
- Knowingly setting off false fire, safety or security alarms
- An accusation of student and/or faculty academic dishonesty or misconduct made in bad faith

Diversity, Equal Opportunity, Affirmative Action

For over three decades, the Rush approach to equal opportunity and diversity has not wavered. It is that equal opportunity and diversity in employment, education, and the delivery of health care are essential and must be furthered. This is a continuation of a policy that emanated from the Hospital Charters of 1865 and 1883 and the documents governing the establishment of Rush University in 1972.

In certain instances, the implementation of these policies requires the use of affirmative action initiatives. At Rush these are focused on strong recruitment and programming efforts, not on the use of quotas - and these recruitment and programming efforts will be continued, consistent with federal, state, and municipal guidelines.

Rush University is committed to attracting students who will enable the student body to achieve the educational benefits of diversity, and to providing services to all students, faculty, and other employees on a nondiscriminatory, equitable basis.

Discrimination or harassment against any member of the Rush University Medical Center community because of age, ancestry, color, disability as defined by Section 504 of the Rehabilitation Act of 1973 and the Americans with Disabilities Act, gender, gender identity and/or expression, marital or parental status, national origin, pregnancy, race, religion, sexual orientation, veteran's status, or any other category protected by federal or state law is prohibited and will not be tolerated, nor will any person for those reasons be excluded from the participation in or denied the benefits of any program or activity within Rush University.

Shanon Shumpert, Director, Employee Relations and Equal Employment Opportunity Officer, has been designated to oversee the implementation of this policy for Rush University. Ms. Shumpert can be contacted by telephone at (312) 942-5239 or via email at Shanon_Shumpert@rush.edu

Additional resources may be found in Human Resources along with the following university individuals/offices:

Susan Chubinskaya, MS

Associate Provost, Academic Affairs Armour Academic Center 441A (312) 942-6306
Susanna_Chubinskaya@rush.edu

Paula J. Brown, MBA

Manager, Equal Opportunity Programs Rush University Medical Center
 128 Professional Office Building (312) 942-7094 Paula_J_Brown@rush.edu

Harassment: Policies and Procedures

The Policies and Procedures on Sexual and Other Harassment for the University and nonacademic sectors of the institution are intended to increase the awareness of Rush's long-standing commitment to preventing harassment and to focus on the internal resolution of any complaints. Under these policies and procedures, the more familiar category of sexual harassment as well as harassment related to age, ancestry, color,

disability as defined by Section 504 of the Rehabilitation Act of 1973 and the Americans with Disabilities Act, gender, gender identity and/ or expression, marital or parental status, national origin, pregnancy, race, religion, sexual orientation, veteran's status, or any other category protected by federal or state law is prohibited. The provisions include protections for and prohibit retaliation against an individual making a complaint or supplying information about a complaint. They also incorporate protections for a person who considers himself or herself accused in bad faith. While all administrators and supervisors have responsibility under this document, certain people have been specifically designated to deal with concerns and complaints that might come forward.

Inquiries or complaints of harassment from students, residents, or faculty members will be handled through the Office for Equal Opportunity by contacting Paula J. Brown, Manager, Equal Opportunity Programs, at (312) 942-7094, by mail (128 Professional Office Building), or via email at Paula_J_Brown@rush.edu. Copies of the Policies and Procedures are available from the Office for Equal Opportunity and are on the Rush Intranet.

Disability Rights

Rush University provides reasonable accommodations to all students on a nondiscriminatory basis consistent with legal requirements as outlined in the Americans with Disabilities Act (ADA) of 1990 and the Rehabilitation Act of 1973 and applicable implementing regulations of these statutes. A reasonable accommodation is a modification or adjustment to an instructional activity, facility, program or service that enables a qualified student with a disability to have an equal opportunity to participate in all Rush University student activities.

To be eligible for accommodations, a student must have a documented disability as defined by the ADA and Section 504 of the Rehabilitation Act of 1973. Both the ADA and Section 504 define disability as (a) a physical or mental impairment that substantially limits one or more major life activities of such individual; (b) a record of such impairment; or (c) being regarded as having such a condition. For information to request accommodation(s), please contact your college representative listed below. Please do not make requests for accommodation(s) to individual faculty members, lectures or course directors.

In keeping with its goal to promote diversity among its student population, Rush University is committed to attracting and educating students who will help to make the population of health care professionals representative of the national population, including students with disabilities. In addition, Rush University wishes to insure that access to its facilities, programs and services are available to students with disabilities. The University provides reasonable accommodations to all students on a nondiscriminatory basis consistent with legal requirements as outlined in the Americans with Disabilities Act of 1990, the American Disabilities Amendment Act of 2008, and the Rehabilitation Act of 1973.

Additional information can be found at:

<http://www.rushu.rush.edu/catalog/aboutrush/disabilityrights.html>

For disability-related assistance, questions, or concerns, contact:

Paula J. Brown, Manager

Equal Opportunity Programs Office for Equal Opportunity

Rush University Medical Center 1725 W. Harrison Street, Suite 128 Chicago, IL 60612

Tel. 312-942-7094, Fax. 312-942-4283, Email. Paula_J_Brown@rush.edu

Student Honor Code:



Rush University Honor Code

I pledge that my academic, research, and/or clinical work will be of the highest integrity. I shall neither give nor receive unauthorized aid; I shall not represent the work of others as my own; I shall not engage in scientific misconduct; and I shall treat all persons with the greatest respect and dignity, just as the ethical codes of Rush University Medical Center and my future profession demand.

I recognize that behaviors that impede learning or undermine academic, research, and clinical evaluation, including but not limited to falsification, fabrication, and plagiarism, are inconsistent with Rush University values and must be reported.

Implementation of the Honor Code

This Honor Code (hereafter referred to as the Code) sets the standards for expected professional behavior within the University and the Medical Center. Commitment to this Code is a shared responsibility of all faculty, staff, and students within the Rush University community to ensure the highest standards of behavior, whether in the classroom, the laboratory, or in the clinical setting, and to ensure that education obtained at Rush provides a sound foundation for each student's future success as an academic, scientific, or healthcare professional.

Code Enforcement

Any violations of this Code or suspicion of student or academic misconduct should be reported to the student's college for further review in accordance with the procedures specified by that college. Each college will be expected to set standards for addressing Honor Code violations and cases of misconduct in a fair and consistent manner that best fits their respective student population. Students refusing to sign must submit a letter to their dean's office explaining why, and adherence to the Code is required for matriculation, whether or not the document has been signed. The Code may also be enforced for off-campus actions when the student is representing themselves as a member of the University.

Commitment

By signing below, I affirm my commitment to this Code and pledge to act with integrity and adhere to the Rush University values of Innovation, Collaboration, Accountability, Respect, and Excellence. I understand that this signed document becomes part of my permanent record, and I must uphold the letter and spirit of this Code throughout my Rush education.

Student Signature

Date

Printed Name

College

HONOR CODE COMMITTEE

THE GRADUATE COLLEGE RUSH UNIVERSITY

The Honor Code Committee (hereafter referred to as the Committee) acts as a third party working to investigate potential infractions of the Graduate College Student Honor Code. The Committee is made up of three voting faculty members (an Assistant Professor, an Associate Professor and a Professor) and three voting graduate students reflecting the diversity of the College. These members are appointed annually by the Dean of the Graduate College. A Chairperson is elected annually at the beginning of the academic year from among the voting members of the Committee. The Associate Dean of the Graduate College is a non-voting member of the committee.

Disciplinary Procedure

Students or Faculty reporting a potential infraction of the Graduate Student Honor Code may do so by contacting any member of the Committee or bringing the matter to the attention of the Associate Dean of the Graduate College (the liaison to the Honor Code Committee). The individual reporting the infraction should complete the Misconduct Complaint form and submit it to the Associate Dean of the college in which the accused student is enrolled before any further action is taken. However, during the process following this report, the individual reporting the infraction retains the option to remain anonymous. The following actions will ensue:

1. A written statement defining what infraction was committed (where, when and by whom) shall be presented to the Chairperson of the Committee within five working days of the incident.
2. Upon receiving the written statement, the Chairperson will meet with the individual who made the statement no later than one week after receiving the written statement. The Chairperson will inform the accused of the information upon which the allegation is based and give him/her an opportunity to respond. The individual who made the original written statement and brought it to the attention of the Committee may at all times remain anonymous.
3. The Chairperson of the Committee determines whether the accusation needs to be further investigated by the whole Committee.
4. If the Chairperson of the Committee concludes that the complaint is warranted, a hearing between the accused party, the accuser and the entire Committee should be scheduled within fifteen days but no later than 30 days after reception of the written report.
5. The accused party may be accompanied by an advisor of her/his choice who is not a party to the violation. The advisor may be any person of the accused party's choosing. The role of the person accompanying the student is that of an advisor, not a presenter of the case. The student must inform the Committee 24 hours before the hearing of his/her intention to bring an advisor. The advisor may not speak on the accused party's behalf during the hearing.
6. The accused student may not cancel a hearing within 24 hours of the hearing. The hearing will continue in the accused student's absence unless proof of extenuating circumstances is provided.
7. At the scheduled hearing/meeting of the Committee, the accuser will present the details of the incident to the voting members. He/she may choose not to face the accused. In that case, the accuser and accused parties will be heard separately, so that anonymity is preserved.

8. Following the presentation of the accusation, the accused party will be given the chance to challenge/refute the accusation by introducing orally, or in a written form, evidence in support of his/her defense.
9. After presentations from the accuser and the accused party, the Committee will be given the chance to question both parties in regards to the accusation. Should the accuser wish to remain anonymous, the Committee will not be allowed to question the accuser at the hearing and will make their decision based on the initial written report and the presentation made by the accused party.
10. The Honor Code Committee will meet in a private session following the hearing to render a decision. The votes of the members of the Committee on motions made and seconded by Committee members will remain anonymous. The result of the vote(s) will be submitted to the Associate Dean who will be responsible for informing the parties involved in the case, including the Division Director of the accused student.

Disciplinary Actions

1. The Honor Code Committee's first response should, when feasible, be educational/remedial.
2. Upon repeated offenses, the Honor Code Committee shall bring the incidences to the attention of the Dean of the Graduate College and the Graduate College Council. This may trigger the College Policy on Dismissal.
3. The Honor Code Committee may also make recommendations for changes to the Course Director, Division, or the Graduate College. Such recommendations may include changes to the course or examination structure, clarification of policy regarding student conduct, and suggestions to better implement such a policy.

<https://www.rushu.rush.edu/graduate-college/faculty-research-projects>

Track for Cardiovascular/Pulmonary Biology

Ai, Xun M.D		Associate Professor		Xun_Ai@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>My research focus on understanding the mechanisms of cardiac arrhythmias in pathologically altered hearts (e.g. heart failure, aging, alcohol abuse). Atrial fibrillation (AF) is the most common arrhythmia, and dramatically increases risk of mortality and morbidity due to stroke and heart failure. Age is a non-specific and unavoidable AF risk factor. Yet, the mechanisms that couple aging and AF propensity remain unclear, making targeted therapeutic interventions unattainable. Our intriguing data show AF risk in aged atria correlates with enhancement of abnormal sarcoplasmic reticulum (SR) calcium (Ca) activities. Why this happens is unknown. The stress response c-Jun N-terminal kinase (JNK) is known to contribute to various cardiovascular diseases (heart failure, hypertrophy, myocardial infarction, atherosclerosis etc.). We discovered that JNK2-driven CaMKII activation is a novel mode of kinase crosstalk and a causal link between aging and AF initiation. The long-term goal of my lab is to explore new therapeutic targets for AF prevention and treatment.</p> <p>My laboratory has established several state-of-the-art electrophysiological techniques in intact hearts and isolated myocytes including dual channel optical mapping, confocal Ca imaging, and <i>in vivo</i> arrhythmia induction. In addition, my laboratory has updated molecular biochemical techniques such as flow cytometry, and enzyme activity assays, qPCR, immunoblotting, immunostaining, ChIP assay, gene cloning.</p>					

Banach, Kathrin Ph.D.		Associate Prof./ Track Director Cardiovascular/Pulmonary		Kathrin_Banach@rush.edu	
Department of Internal Medicine/Cardiology					
MS/Ph.D. Students for Laboratory Rotations	YES	Master Thesis Students	YES	PhD Dissertation Students	YES
<p>Dr. Banach's laboratory focuses on the cellular mechanism of atrial fibrillation (AF). AF is the most common cardiac arrhythmia (>2.3 million Americans) with older age, obesity, diabetes, and hypertension increasing its likelihood. AF significantly increases the risk of heart failure and stroke (> 25% strokes preceded by AF). The mechanisms that induce AF however are not entirely understood and no effective pharmacotherapy is available. Specific projects are:</p> <p>1. Loss of Pak1 increases the propensity of AF. Goal of the project is to determine the mechanism by which Pak1 is regulated in patients with AF; how reduced Pak1 increases the propensity of AF and how restoration of Pak1 activity can be used as a pharmacological treatment for AF. 2. Cellular alternans is long known to increase the propensity for ventricular arrhythmia but new clinical evidence shows episodes of alternans preceding AF. In collaboration with Dr. Blatter (see below) we determine the</p>					

cellular mechanism of alternans and the tissue parameter that facilitate the transition from cellular alternans into tissue alternans and AF.

3. While pre-menopausal women have a lower prevalence to develop AF, post-menopause their propensity for AF picks up significantly and their overall disease burden is increased. We aim to understand gender dependent differences in atrial function with the goal to exploit gender specific cardio-protective signaling mechanism to attenuate AF progression. The lab uses state of the art electrophysiological, biochemical, and imaging techniques to characterize cellular and tissue function (e.g. patch clamp, multi electrode mapping, ECG, Echo, confocal imaging techniques, stem cells, genetic mouse models, large animal models of AF and chemotherapy, qPCR etc.).

<https://www.rushu.rush.edu/research/departamental-research/internal-medicine-research/cardiology-research/laboratory-kathrin-banach-phd>

Blatter, Lothar A., M.D.		Professor		Lothar_Blatter@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	No	PhD Dissertation Students	No
<p>Dr. Lothar A. Blatter's research group investigates control of intracellular calcium and its role in the regulation of excitation-contraction and excitation-transcription coupling in cells of the cardiovascular system. The research includes fundamental characterization of ion transporters and channels, electrophysiology, excitation-contraction coupling, mitochondrial Ca signaling and energetics, excitation-transcription coupling and changes in Ca signaling in cardiac disease (arrhythmia, hypertrophy and heart failure). Our research efforts have resulted in seminal contributions to the understanding of the spatio-temporal organization of cardiac Ca signals ranging from global Ca transients, arrhythmogenic Ca waves and alternans to the elementary Ca release events from ryanodine receptor (Ca sparks and blinks) and IP₃ receptor (Ca blips and puffs) Ca release channels. The laboratory has extensive experience in high-resolution state-of-the-art fluorescence microscopy, including laser scanning confocal microscopy using single-photon and two-photon excitation, TIRF microscopy and video imaging in conjunction with cellular electrophysiology and molecular techniques.</p> <p>Website: https://www.rushu.rush.edu/faculty/lothar-blatter-md-dr-med</p> <p>Publications: http://www.ncbi.nlm.nih.gov/pubmed/?term=Blatter+LA</p>					

Fill, Michael Ph.D.		Professor, Chair		Fill_Michael@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr. Fill: Each time the heart beats a large intracellular calcium signal drives the contraction. Dr. Fill studies the Ca channel which mediate that Ca signal. Normally, those Ca channels (called ryanodine receptors) stay closed between heart beats. Abnormal ryanodine receptor opening (between beats) contributes to heart failure and can cause life-threatening arrhythmias. Dr. Fill's research explores ryanodine receptor local control in healthy hearts and how that control is distorted by disease. Recently, Dr. Fill has begun developing new drugs that normalize those distortions with the goal of developing anti-arrhythmic drugs that can be used to prevent and/or treat dangerous cardiac arrhythmias. Outside the heart, ryanodine receptor malfunction is also known to contribute to various skeletal muscle, renal and neurological diseases (including Alzheimer's disease). Thus, a longer term goal is to develop new ryanodine receptor targeted agents that may help prevent and treat those diseases as well.</p>					

Gillespie, Dirk Ph.D.		Associate Professor		Gillespie_Dirk@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr. Dirk Gillespie. The goal of my lab is to use modeling, simulations, and theory to understand biological processes. The lab focuses mainly on calcium movement during heart muscle contraction (excitation-contraction coupling) to understand the normal process as well as what leads to cardiac arrhythmias and how certain antiarrhythmic drugs work (and possibly design better ones). The release of calcium through the ryanodine receptor (RyR) ion channel out of the sarcoplasmic reticulum (SR) is a fundamental part of muscle contraction. We study this on several different levels: on the single channel level to understand how RyR selects for and conducts calcium over the other ions that are present; on the multi-RyR level to understand the process and termination of calcium-induced calcium release, a positive feedback mechanism where the calcium released by one RyR can open neighboring RyRs; and on the organelle level to understand how ions of all kinds move in and out of the SR during a heartbeat. For all of these studies, we collaborate very closely with experimentalists in the Department of Physiology.</p>					

Gupta, Vineet		Professor of Medicine, Vice Chair for Research and Innovation		Vineet_Gupta@rush.edu	
Department of Internal Medicine/ Immunology/Microbiology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>A major goal in the Gupta laboratory is to fully understand integrin activation at a molecular and cellular level to develop novel integrin agonists as therapeutics. We are initially targeting beta1 and beta2 family of integrins. The laboratory specializes in Drug Discovery approaches using high-content screening (HCS). Two specific research areas are:</p> <ol style="list-style-type: none"> 1. Targeting innate immune cells for treating Inflammation: Several years ago, we made a paradigm-changing observation that integrin activation is a novel mechanism for targeting innate immune cells. Subsequently, we identified novel small molecules to target integrin CD11b/CD18 (Mac-1) on neutrophils and macrophages and used it to show that such agents are highly efficacious in reducing cardiovascular inflammation and injury. Current studies are focused on unraveling its molecular and cellular signaling mechanism in models of autoimmune diseases (lupus and lupus nephritis). We recently showed that CD11b activation suppresses TLR signaling, to reduce pro-inflammatory pathways in cells. We have also made a surprising finding that these agents target tumor associated macrophages (TAMs), modify tumor microenvironment and reduce tumor growth, thus also have a significant effort in Cancer Biology. We are currently completing studies to be able to initiate a new Phase I clinical trial with these agents. 2. Podocyte targeted therapeutics: Podocytes are essential for kidney function and are one of the first cells to be damaged during injury. These cells use integrin beta1 to adhere to basement membrane and function. We recently developed a first-of-its-kind high content screening assay to identify novel beta1 integrin agonists that protect podocytes from injury. Current studies in the laboratory are focused on understanding the molecular and cellular signaling mechanism of our novel hits as well as their efficacy in models of diabetic kidney disease and FSGS. <p>The laboratory currently has three post-docs, two graduate students, two medical students and several undergraduate interns.</p>					

Predescu, Sanda Ph.D.		Associate Professor		Predescu_Sanda@rush.edu	
Department of Internal Medicine/ Pulmonary, Critical Care, and Sleep Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	No	PhD Dissertation Students	Yes
<p>Our research is focused on understanding the molecular mechanisms of endothelial cell functions in normal and pathological conditions and how intersectin proteins regulate these functions. In particular, we are interested in endothelial cell hyperproliferation, abnormal neovascularization and vascular malfunctions, which are hallmarks of acute respiratory distress syndrome (ARDS) and pulmonary arterial hypertension (PAH). Integrated understanding of the mechanisms regulating the properties and function of endothelial cells in these pathological settings may result in the development of a number of exciting approaches for the treatment of these severe human diseases. We use cell culture systems, murine models and specimens of ARDS and PAH patients in conjunction with molecular, cell biology, morphological and functional approaches to investigate:</p> <p>i) the molecular mechanism triggered by circulatory microparticles leading to endothelial cells proliferation and lung microvascular remodeling in the repair phase of ARDS. We will define the proliferative pathways upregulated by disruption of Smad2/3-Erk1/2 signaling of TGFβ/Alk5 as induced by intersectin-1s deficiency and test the hypothesis that Runx1 transcription factor is responsible for ARDS-associated neo-vascularization and tissue repair.</p> <p>ii) the signaling events triggered by the NH2-terminal granzyme B-cleavage product of intersectin-1s during pulmonary inflammation associated with PAH leading to endothelial cell proliferation, formation of plexiform lesions and vascular remodeling. We explore whether the proliferative potential of this cleavage product and the vascular remodeling triggered can be ameliorated via an inhibitory, cell permeable peptide and the implications of granzyme B cleavage products of intersectin-1s on the genetic instability of endothelial cell phenotype in PAH.</p>					

Ramos-Franco, Josefina M.D/Ph.D.		Assistant Professor		jrranco@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	No	PhD Dissertation Students	Yes
<p>My research involves the phenomenon of intracellular Ca²⁺ release in excitable cells. The inositol trisphosphate receptor (IP₃R) and the ryanodine receptor (RyR) are homologous ion channels that mediated Ca²⁺ release from intracellular storage organelles (endoplasmic reticulum; ER). For the signals to be effective, Ca²⁺ release must start, terminate, stop as sharply as it starts. Dysregulation of this process has severe ramifications in nearly all types of cells. Recent emerging evidence suggests that IP₃R-mediated Ca²⁺ signaling is important in both the development and pathophysiology of diseases like ataxia, neuronal degeneration, familial Alzheimer's disease, exocrine secretion deficit and heart disease.</p> <p>Specifically, my research interest is, understand Ca²⁺ release mediated by IP₃R and RyR</p>					

channels and its mechanism(s) of local Ca^{2+} control in cells. My research uses a multidisciplinary approach from the cellular to the molecular levels. Such approach is based on the use of site-directed mutagenesis, laser-scanning confocal microscopy, and ion channel reconstitution into planar lipid bilayers.

Reiser, Jochen M.D./Ph.D.		Professor, Chair		Jochen_Reiser@rush.edu	
Department of Internal Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Title: Soluble urokinase receptor and cardio-renal diseases</p> <p>Soluble urokinase receptor (suPAR) is typically a three domain signaling protein that can be released into the circulation by the cleavage of membrane bound uPAR. It is considered a broad marker of immune activation and was found to be a direct pathogen in podocytopathies like Focal and Segmental Glomerulosclerosis (FSGS). In FSGS, elevated plasma suPAR binds to and activates $\alpha\beta 3$ integrin on podocytes causing their structural rearrangement and proteinuria. suPAR can cooperate with CD40 auto-antibodies and is implicated in FSGS recurrence. More recent data unravels that suPAR is predictive of incident chronic kidney disease (CKD) and associates with future decline in already existing mild to moderate CKD. suPAR is also associated with future cardiovascular events in CKD patients further supporting a central role for this molecule in the pathogenesis of kidney disease and its complications. Novel studies pinpoint the source of suPAR excess to myeloid cells in the bone marrow and decipher important new interactions with genetic kidney disease risk factors such as APOL1.</p> <p>The laboratory would like to study mechanisms of suPAR mediated kidney and heart disease using in silico, cell and animal systems as well as human samples.</p>					

Rios, Eduardo Ph.D.		Professor, Section Head		Eduardo_Rios@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	No	PhD Dissertation Students	Yes
<p>The laboratory of Eduardo Rios works on the mechanisms of control of rapid calcium signals in excitable cells. While crucial to many cells, we focus on their workings in striated muscle, skeletal and cardiac, where they support movement, locomotion, and the cardiac beat. Currently we explore the roles of a protein, calsequestrin, which constitutes the main device for storage of the calcium ions that convey these signals. A number of proteins, including calsequestrin, link together mechanically in a supra-molecular device, named the couplon, which controls the calcium signals. Mutations of the couplon proteins cause a group of diseases, with both diverse and similar features, named "couplonopathies". Through studies in cells of patients, we are trying to understand the alterations in normal mechanisms that underlie these couplonopathies. Of specific interest is a group of patients (which we named the "HH"), recently identified by hypersensitivity to a surgical anesthetic. In research funded by the National Institutes of Health we are currently defining the cellular mechanisms of their disease, with the long term goal of contributing to their treatment.</p>					

Shannon, Thomas Ph.D.		Associate Professor		Thomas_Shannon@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>Dr. Thomas Shannon is a member of the Section of Cellular Signaling in the Department of Molecular Biophysics and Physiology. He is interested in ionic channels, voltage gated ionic channels, fluorescence signal detection and electrophysiology, particularly as they relate to excitation-contraction coupling in striated muscle. Dr. Shannon uses multiple biochemical, biophysical and molecular approaches to study the control of the concentration of calcium ([Ca]) in the storage organelle (the sarcoplasmic reticulum) of normal and abnormal cells of the heart. Quantitative determination of the release of Ca will allow him to understand the mutual interactions of SR [Ca] and Ca release, and thus the control of contractile force, an important determinant of cardiac ejection (blood flow) in health and disease.</p> <p>For instance, Dr. Shannon has also demonstrated that the SR [Ca] is reduced during heart failure, perhaps because the “leak” of Ca during diastole out of the SR is increased. His research suggests that this reduction may be a critical factor in causing reduced cardiac contraction in this condition.</p> <p>In addition to reducing the SR [Ca], this SR Ca leak may cause an increased tendency towards cardiac arrhythmias, especially during adrenergic stimulation (i.e. during stress). Ongoing experiments are aimed at determining what causes this increase SR Ca “leak”, especially during adrenergic stimulation in heart failure. The goal is to identify drug targets which may lead to reduction of this leak for the treatment of heart failure.</p>					

Wasse, Monnie MD,MPH		Professor		Monnie_Wasse@rush.edu	
Department of Internal Medicine/Nephrology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr. Wasse’s research focuses on the predictors, consequences and treatment of dialysis arteriovenous access dysfunction. She seeks to characterize the key mechanisms, pathways and genes involved in dialysis access dysfunction, particularly in high-risk populations and to establish a comprehensive vascular access database and biologic specimen biobank. Her previous work, as principal investigator of studies funded by the National Institutes of Health, the Robert Wood Johnson Foundation paves the way for these efforts.</p>					

Shannon, Thomas Ph.D.		Associate Professor		Thomas_Shannon@rush.edu	
Department of Molecular Biophysics & Physiology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>Dr. Thomas Shannon is a member of the Section of Cellular Signaling in the Department of Molecular Biophysics and Physiology. He is interested in ionic channels, voltage gated ionic channels, fluorescence signal detection and electrophysiology, particularly as they relate to excitation-contraction coupling in striated muscle. Dr. Shannon uses multiple biochemical, biophysical and molecular approaches to study the control of the concentration of calcium ([Ca]) in the storage organelle (the sarcoplasmic reticulum) of normal and abnormal cells of the heart. Quantitative determination of the release of Ca will allow him to understand the mutual interactions of SR [Ca] and Ca release, and thus the control of contractile force, an important determinant of cardiac ejection (blood flow) in health and disease.</p> <p>For instance, Dr. Shannon has also demonstrated that the SR [Ca] is reduced during heart failure, perhaps because the “leak” of Ca during diastole out of the SR is increased. His research suggests that this reduction may be a critical factor in causing reduced cardiac contraction in this condition.</p> <p>In addition to reducing the SR [Ca], this SR Ca leak may cause an increased tendency towards cardiac arrhythmias, especially during adrenergic stimulation (i.e. during stress). Ongoing experiments are aimed at determining what causes this increase SR Ca “leak”, especially during adrenergic stimulation in heart failure. The goal is to identify drug targets which may lead to reduction of this leak for the treatment of heart failure.</p>					

Track for Immunity, Infection and Inflammation

Lena Al-Harhi Ph.D.		Professor, Interim Chair		Lena_Al-Harhi@rush.edu	
Department of Microbial Pathogens and Immunity					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Lena Al-Harhi is a Professor and Interim Chair in the Dept. of Microbial Pathogens and Immunity at Rush University Medical Center in Chicago, IL. She is also the Program Director of Rush Initiative to Maximize Student Development (RUSH-IMSD), an NIH funded PhD training grant for underrepresented minority scientists enrolled in the Integrated Biomedical Sciences (IBS) program at Rush graduate college. Dr. Al-Harhi's research over the past 18 years has focused on HIV/host interactions, with a special emphasis on bridging basic and clinical science in the HIV/AIDS field. Because of her experience in HIV molecular biology, immunology, and for the past ten years in neuroAIDS, she has been able to probe mechanistic questions that are clinically relevant to HIV/AIDS. She has over 70 peer-reviewed publications and invited reviews/book chapters. Her research focus is on understanding the dynamic cross talk between Wnt/β-catenin signaling, inflammatory mediators, and HIV as they regulate HIV transcriptional activity and pathogenesis in the CNS. The Wnt/β-catenin pathway is vital for proper CNS development and homeostasis. Her group has identified the β-catenin signaling pathway as an important regulator of HIV replication in multiple compartments, including the central nervous system, and in playing key roles in HIV neuropathogenesis. Her lab is also evaluating the role of host factors (e.g. inflammatory mediators), HIV and co-variants (e.g. drugs of abuse) in modulating β-catenin which will in turn impacts overall CNS homeostasis. Further, she is investigating the role of astrocytes in HIV latency. Lastly, she has an interest in neuroimmunology, as it relates to anti-HIV responses in the CNS. Her lab uses cutting-edge methodologies and tools to address these important topics.</p>					

Aloman, Costica MD		Associate Professor		costica_aloman@rush.edu	
Department of Internal Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Costica Aloman, MD. is the Medical Director of Liver Transplant at Rush University Medical Center and Associate Professor of Medicine. Our work was initially centered by evaluating how alcohol affects functions of a highly specialized population of mononuclear phagocytes, dendritic cells. As a result we identified the critical role of dendritic cell defects in the impaired cellular immune response against HCV proteins induced by alcohol. Working in close collaboration with the well renowned group of Dr. Miriam Merad in the field of mononuclear phagocytes, we further developed expertise in characterization of leukocyte populations in the liver in the steady-state and inflammatory conditions and we uncovered an unexpected role of dendritic cells in fibrosis regression, an effect independent of hepatic natural killer cells. Our expertise in the field of cellular immunology is reflected by multiple publications resulting from collaborations with well known research groups investigating hepatic injury and repair.</p>					

Currently, our group's research is focused on: 1. Investigation of plasmacytoid dendritic cells in the immune abnormalities after chronic exposed to alcohol; 2. Effect of IL15 signaling on liver fibrosis; 3. The role of plasmacytoid DC in liver cancer development; 4. How circadian disruption modulates ethanol induced liver and brain injury (in collaboration with Dr. Ali Keshavarzian group); 5. Characterization of how NK cells control hepatic immune responses. Other key personnel include Holger Fey, PhD (Post-doctoral Fellow) and Alyx Vogle (Research Assistant).

Barker, Edward Ph.D.		Professor		Edward_Barker@rush.edu	
Department of Microbial Pathogens and Immunity					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Edward Barker: Combination anti-retroviral therapy (cART) has dramatically improved the prognosis of HIV-infected individuals. <i>However, patients on cART are still at increased risk for non-AIDS related conditions, particularly those associated with sustained increases in systemic inflammation (e.g., heart disease, type II diabetes).</i> This implies a slow, yet significant uptick in systemic inflammation secondary to HIV infection that has long-term (years to decades) pathological consequences. Our <u>overarching goal</u> is to fill the emerging gap in addressing the needs of HIV-infected patients as they age. The <u>central objective</u> of the proposed work is to elucidate the cellular and molecular mechanisms underlying the longer-term effects of HIV-associated inflammation, with the broader goal of informing novel treatment strategies. We propose that a sustained systemic inflammatory response is likely part of a pathological cascade that disrupts the intestinal epithelial barrier, allowing microbial translocation (MT) from the gut lumen. <i>our central hypothesis that HIV infection triggers the release of the pro-inflammatory cytokine IL-18 in response to microbial products in the mucosa. This in turn triggers the subsequent release of IFN/TNF by mucosal lymphocytes leading to tight junction dysfunction, MT, and the persistent mucosal and systemic inflammation characteristic of HIV infection.</i> We also are investigating the impact of the inflammatory response to microbes in the gut mucosa on epithelial cell function.</p>					

Barua, Animesh Ph.D.		Assistant Professor		Animesh_Barua@rush.edu	
Laboratory for Translational Research on Ovarian Cancer, Departments of Cell and Molecular Medicine. Conjoint appointments in Pathology, Obstetrics and Gynecology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Our main focus is the translational research on gynecological malignancies including ovarian, leiomyosarcoma (a form of uterine cancer) and cervical cancers.</p> <p>Project-1: Development of Immunotherapies against OVCA: Cancer chemotherapeutics are toxic to normal tissues and affects the quality of life especially in postmenopausal women with OVCA. Immunotherapies are safe and offer effective alternatives to chemotherapeutics. In this project, we are testing herbal products to enhance tumoricidal functions of NK cells.</p>					

Project-2: Effects of Metformin on the prevention of tumor induced immune suppression: Tumor induce immunosuppression is a hallmark of tumor progression. Metformin, an FDA approved medication for patients with diabetes. Emerging information shows that Metformin may prevent tumor progression in patients with diabetes. The goal of this project is to examine the effects of metformin on the immune modulation in patients with ovarian cancer.

Project-3: Anti-tumor autoantibodies as marker for early detection of ovarian cancer. Malignant transformation is associated with the expression of tumor antigens against which immune system produces anti-tumor autoantibodies. Thus, production of anti-tumor autoantibodies are the harbinger of malignant transformation and offers a potential marker for early detection of ovarian cancer. The goal of this project is to examine and detect the prevalence of anti-tumor autoantibodies for early detection of ovarian cancer.

Cohen, Frederic Ph.D.		Professor		Fredric_Cohen@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	No	Master Thesis Students	No	PhD Dissertation Students	YES
<p>Frederic Cohen: We are investigating the molecular mechanisms by which viruses deposit their genetic material into cells. When a virus fuses its coating membrane (known as an envelope) with a cell membrane, a fusion pore is created. This pore connects the interior of the virus to that of the cell. The viral genome passes through this pore and enters the cell's cytosol. Proteins within the viral envelope, called fusion proteins, drive fusion, but many cellular factors regulate the process. Our goal is to discover and identify the mechanisms and cellular controls for viral fusion, and to formulate general principles that can be applied to prevent infection. Over the years, we have studied fusion induced by the proteins of HIV, influenza, Leukemia viruses, and Ebola, and have identified common mechanisms among them as well as specifics that apply to only a particular virus. We have also identified ways that cellular proteins and physical properties of membranes regulate fusion, for example, via electrical voltages across them.</p>					

Forsyth, Christopher B. Ph.D.		Associate Professor		Christopher_Forsyth@rush.edu	
Department of Internal Medicine/Gastroenterology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Christopher B. Forsyth, PhD. Director of the Digestive Diseases Basic Research Lab. The rationale for our approach is that: Chronic diseases are primarily inflammatory disorders and <i>gut-derived inflammation is a plausible source/trigger for inflammatory cascades</i>. Gut-derived inflammation could be a consequence of changes in intestinal barrier permeability (i.e., "leaky gut") and/or changes in intestinal microbiome</p>					

composition/function (i.e., dysbiosis) both of which would lead to exposure of the immune system to pro-inflammatory microbial factors like endotoxins. Chronic diseases are more common in Western societies (and societies with Western life style) thus environmental factors associated with the Western life style are promising factors to study. Examples of Western life style-associated risk factors we study include: alcohol consumption, dietary factors including fiber and fat content, stress, as well as circadian rhythm disruption. Our group studies the impact of these factors on numerous pathological and disease states including: intestinal barrier function and microbiome composition in: aging, obesity/metabolic syndrome, HIV/AIDS, Parkinson's and Alzheimer's disease, multiple system atrophy (MSA), post traumatic stress disorder (PTSD), epilepsy, alcoholic liver disease, non-alcoholic fatty liver disease (NAFLD), allergy, inflammatory bowel disease (ulcerative colitis, Crohn's disease), irritable bowel syndrome (IBS), colon and breast cancer, neonatal development, and osteoarthritis. Our laboratory combines data from clinical studies with data from mouse models, and *in vitro* and intestinal organoid molecular methods including genomics, transcriptomics, and metabolomics, as well as tissue protein measurements and staining. Other key personnel include Ali Keshavarzian, MD; Faraz Bishehsari, MD/PhD; Garth Swanson, MD/MS; and Robin M. Voigt-Zuwala, PhD.

Glant, Tibor T. MD/Ph.D.		Professor		Tibor_Glant@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Tibor Glant, MD/PhD: The ongoing research projects are the genetics and autoimmunity of inflammatory joint diseases; more specifically the pathogenesis of rheumatoid arthritis and ankylosing spondylitis. These studies include (i) the screening, identification and localization of "disease susceptible" gene(s) that control autoimmune processes and inflammatory cell migration into rheumatoid synovium, (ii) the autoimmune mechanisms of ankylosing spondylitis including the screening, identification and localization of "disease susceptible" gene(s) in a corresponding animal model, (iii) the immunology/immunopathology and genetics of extracellular matrix components (specifically cartilage macromolecules), (iv) the mechanisms of antigen presentation in normal and pathological (autoimmune) conditions, and tolerization of autoimmune-prone individuals against the disease, (v) potential gene- and immunotherapeutic interventions in autoimmune/inflammatory diseases. (vi) Recently, we have identified a gene, and partially characterized its disease-associated function, which gene (if defected by mutation or inherited by parents) is responsible for at least two rare skin diseases such as pyoderma gangrenosum and a severe myeloproliferative disease leading to Sweet's syndrome. All these studies were initiated by animal models and then supported and/or confirmed by information of patients with the corresponding human disease. A "hold on" project is the cellular and molecular (signaling) mechanisms of pathological bone resorption in failed joint replacements. All these major directions involve studies employing state-of-the-art strategies and techniques in basic molecular biology, biochemistry, genetics, cell biology and immunology.</p>					

Gupta, Vineet		Vice Chair for Research and Innovation		Vineet_Gupta@rush.edu	
Department of Internal Medicine/ Immunology/Microbiology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Vineet Gupta, PhD is a member of the Departments of Medicine and Microbiology/Immunology. We are applying cross-disciplinary approaches to develop novel therapeutics in two research areas:</p> <ol style="list-style-type: none"> <u>Modulating innate immune response in Inflammation and Cancer:</u> Several years ago, we made a paradigm-changing observation that integrin activation is a novel mechanism for targeting innate immune cells. Subsequently, we identified novel small molecules to target integrin CD11b/CD18 (Mac-1) on neutrophils and macrophages and used it to show that such agents are highly efficacious in reducing cardiovascular inflammation and injury. Current studies are focused on unraveling its molecular and cellular signaling mechanism in models of autoimmune diseases (lupus and lupus nephritis). We have also made a surprising finding that these agents target tumor associated macrophages (TAMs), modify tumor microenvironment and reduce tumor growth, thus also have a significant effort in Cancer Biology. We are currently completing studies to be able to initiate a new Phase I clinical trial with these agents. <u>Protecting podocytes from damage in Chronic Kidney Disease:</u> Podocytes form the filtration barrier in the kidneys and their damage is a central hallmark of all chronic kidney diseases. Therefore, we recently developed a first-of-its-kind high content screening assay to identify compounds to protect podocytes from injury. Current studies in the laboratory are focused on understanding the molecular and cellular signaling mechanism of our novel hits as well as their efficacy in models of diabetic kidney disease and FSGS. <p>The laboratory currently has one junior research faculty member, three post-docs and three graduate students, in addition to several undergraduate interns. For further information please go to: http://guptalab.com</p>					

Hu, Xiu-Ti, MD/Ph.D.		Associate Professor		Xiu-Ti_Hu @rush.edu	
Department of Microbial Pathogens and Immunity					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Xiu-TI, Hu, MD/PhD. Research in my laboratory focuses on revealing the brain dysfunction induced by the neuropathogenesis of HIV and drugs of abuse, especially during aging. Comorbid neuroHIV and cocaine profoundly alter vulnerable brain regions, including, but not limited to, the prefrontal cortex (PFC), striatum, and hippocampus, the regulators of cognition. Such changes likely contribute to the progression of HIV-associated neurocognitive disorders (HAND) and cocaine or methamphetamine (a.k.a. psychostimulants) addiction. Moreover, the side effect of anti-retroviral drugs and aging may exacerbate the comorbid dysfunction induced by HIV and drugs of abuse. Defining the mechanism(s) underlying the functional and anatomical alterations in the medial PFC and ventral/dorsal striatum in the mesocorticolimbic and corticostriatal systems, and</p>					

between neurons and glial cells in these brain regions, could help the development of novel therapeutic strategies to treat HAND and comorbid drug addiction during aging. Integrative electrophysiological, biochemical/molecular and behavioral approaches, associated with animal models of drug abuse and neuroHIV of humans at young and old ages, are utilized in our research. Collaboration between our research team and others are actively ongoing within and out of Rush University. My research is currently supported by the National Institute of Neuronal Disorders and Stroke (NINDS), and by the National Institute on Drug Abuse (NIDA).

Keshavarzian, Ali MD		Professor		Ali_Keshavarzian@rush.ed	
Department of Internal Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Ali Keshavarzian, MD: Studies the involvement of the intestinal tract in human diseases. The lab emphasizes translational research utilizing human clinical research, animal models of disease (primarily mice), as well as <i>ex vivo</i> and <i>in vitro</i> studies to investigate disease mechanisms. Major themes of research include mechanisms related to inflammation, cancer, circadian rhythms disruption promotion of disease, alcohol-related diseases, and intestinal microbiota-host interactions. The role of intestinal barrier integrity (i.e., leaky gut) promoting disease is also a major theme of the lab. Specific diseases include studies of: (1) inflammatory bowel disease (IBD), (2) alcoholic liver disease, (3) colon cancer, and (4) Parkinson's disease. Other active projects are exploring the role of the intestinal tract in (5) HIV pathogenesis, (6) mind body medicine impacting the brain-gut axis, (7) food allergy, (8) irritable bowel syndrome (IBS), (9) pancreatic cancer, (10) metabolic syndrome-obesity, (11) epilepsy, (12) osteoarthritis, and (13) the role of socioeconomic status in disease processes. In all of the diseases mentioned the focus of our research is to mechanistically understand the role of inflammation and specific immune cells, especially T cell subsets, innate lymphoid cells, and cytokines on disease progression. In addition, the lab is interested mechanistically in understanding how changes in the intestinal microbiota are influencing immune function especially during circadian rhythm disruption, alcohol consumption, and leaky gut. Other key personnel include Faraz Bishehsari, MD/PhD, Garth Swanson, MD, Robin M. Voigt-Zuwala, PhD and Christopher B. Forsyth, PhD.</p>					

Marzo, Amanda L. Ph.D.		Assistant Professor		Amanda_Marzo@rush.edu	
Department Internal Medicine/ Hematology, Oncology & Cell Therapy					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Amanda Marzo PhD: Our main focus is to understand the mechanisms underlying the induction and maintenance of viral memory CD8 T cells, the factors responsible for sustaining effector function and the mechanisms for their persistence. For the most part, efficacious vaccines that rely on T cells directed against mucosa-acquired infections have been ineffective. This shortcoming may result from insufficient numbers of functional memory CD8 T cells being generated and/or maintained at the site of initial</p>					

transmission. Recently, the mammalian target of rapamycin (mTOR) has been implicated in regulating the intrinsic transcriptional programs that determine the fates of effector and memory CD8 T cells. This project will focus on understanding how mTOR controls the generation of viral effector and memory CD8 T cells and how mTOR influences trafficking of mucosal memory CD8 T cells. The majority of viral infections in the female reproductive tract (FRT) are established through transmission across mucosal surfaces that line the genital tract. Successful control of infections that occur in the FRT, such as HSV-2, are thought to be associated with the presence of sustained tissue resident memory CD8 T cells that reside in the draining lymph nodes of the genital tract and the vaginal mucosal tissues. This project will determine 1) if we can increase HSV-2 specific effector memory CD8 T cells in the FRT by regulating the level of mTOR signaling and 2) if HSV-2-specific CD8 T cells in the FRT can be enhanced by IL-15 therapy. Results from this project will provide mechanisms and signals involved in generating HSV-2-specific effector memory CD8 T cells in the FRT and strategies to enhance cellular immunity in the FRT that would ultimately protect women against HSV-2.

Mikecz, Katalin MD/Ph.D.		Professor		Katalin_Mikecz@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes

Dr. Mikecz studies autoimmune responses to cartilage components in arthritis-susceptible mice and in rheumatoid arthritis (RA) patients. RA is an autoimmune disease affecting nearly 1% of the human population and causing painful inflammatory destruction of the joints. Immune recognition of self antigens, particularly those in the joint is a hallmark of RA. One of the major components of joint (articular) cartilage is a large molecule called proteoglycan (PG) or aggrecan. With age, some amino acids in the protein backbone of the PG molecule undergo chemical modifications. One of the most common post-translational modifications is the enzymatic conversion of the amino acid arginine to citrulline. While this modification (citrullination) apparently does not cause any problem in healthy individuals, in RA patients the cells of the adaptive immune system mistakenly recognize citrullinated proteins as foreign molecules and mount an inflammatory attack to eliminate them from the body. During this process, B cells produce antibodies that can bind to molecules containing citrulline, thereby targeting these citrullinated proteins for engulfment and destruction by other immune cells. Most patients with RA produce anti-citrullinated protein antibodies (ACPA). We have recently found that ACPA bind to human cartilage PG, a macromolecule that induces both arthritis and ACPA production when injected into mice. With regard to RA, citrullinated self PG can be either an inducer or a target of ACPA. In this project we characterize citrullinated PG-specific T- and B-cell responses in RA patients and identify ACPA bound to cartilage in their joints. We will also investigate the mechanisms that connect citrullinated PG-specific immune responses to the development of joint inflammation in ACPA-producing mice with citrullinated PG-induced autoimmune arthritis. We believe that these studies, by providing insights into the role of citrullinated PG in provoking immune attacks against the joints in both RA and a mouse model of the human disease, will facilitate the development of new treatments for RA.

Rauch, Tibor A. MD/Ph.D.		Professor, Director, Section of Molecular Medicine		Tibor_Rauch@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Tibor Raunch, MD/PhD: Epigenetic factors form bridges between the environment and the genetic information-harboring DNA by interpreting external stimuli and regulating gene expression. Alterations in epigenetic mechanisms influence immune system activity and contribute to the pathogenesis of various inflammatory diseases including rheumatoid arthritis (RA). RA is a degenerative autoimmune disease and it is well established that both genetic risk factors (mutations) and epigenetic alterations (epimutations) can be implicated in the etiology of RA. Our research is mainly focused on (i) exploring epigenetic factors involved in RA, (ii) understanding their contribution to the initiation and the progression of the disease and (iii) testing new drugs that target differentially expressed epigenome modifiers. We employ genome-wide DNA methylation profiling techniques, gene expression microarrays, and a wide variety of recombinant DNA technologies for achieving the above-delineated goals. We investigate arthritis-associated epigenetic events in cell cultures and a mouse model of RA. Getting involved in one of these projects can allow you to (i) contribute to the better understanding of pathomechanisms of RA and (ii) delineate new therapeutic options for arthritis.</p>					

Shafikhani, Sasha. Ph.D.		Associate Professor		<u>sasha_shafikhani@rush.edu</u>	
Department of Internal Medicine/Hematology, Oncology & Cell Therapy					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Sasha Shafikhani:Our lab is highly multidisciplinary in nature. We conduct projects that involve bacterial pathogenesis, cancer biology, and chronic wound healing. We are funded by NIH RO1 and philanthropy grants to conduct research in cancer and diabetic wound infections. Since 2015, our lab has published 8 manuscripts in high impact journals (such as Dev Cell, JBC, and PLoS Pathogens) and we currently have two more manuscript under review. Of major accomplishments of the lab, we recently discovered that cancer cells (under therapy) release specialized micro vesicles before they die that stimulate other cancer cells to proliferate. The ability of dying cancer cells to stimulate proliferation in other cancer cells no doubt reduces the effectiveness of current cancer drugs to eliminate cancer. Importantly, we have found that a bacterial toxin (ExoT) is able to induce potent cell death in a variety of cancer cells but also prevents them from releasing these vesicles, which gives us hope that we can enhance the effectiveness of current cancer therapeutics. Another important accomplishment of our lab is the discovery that normal tissues produce natural antibiotics that can target and kill bad pathogenic bacteria amongst normal good bacterial flora. We have found that diabetic tissues lack this ability and are thus vulnerable to bacterial infection. We are hoping to develop antibiotics that only kill pathogenic bacteria not commensals. We use state-of-the-art technologies and my students have all been incredibly successful. Currently, we</p>					

have 2 postdocs, one PhD student, and a clinician volunteer researcher who help train new students.

Williams, David. Ph.D.		Associate Professor		<u>David_williams@rush.edu</u>	
Department of Microbial Pathogens and Immunity					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>David Williams: Dr. Williams' lab focuses on translational research on schistosomiasis. More than 200 million people have schistosomiasis, a neglected tropical disease, resulting in more than 200,000 deaths annually. We investigate schistosome worm biochemistry to identify targets useful for developing new drugs for schistosomiasis treatment. Specifically, we investigate a variety of detoxification pathways in the worm that we have found to be unique or to differ significantly from human pathways. We have found that the schistosome redox network is distinctly different from humans. Most importantly, schistosomes lack both authentic glutathione reductase and thioredoxin reductase with both activities provided by a single protein, thioredoxin-glutathione reductase. Using both reverse genetic and pharmacological approaches we have generated compelling evidence that thioredoxin glutathione reductase is an essential protein and druggable target. We have completed large compound library screens and identified promising hits specifically targeting this protein. We are also interested in mechanisms of detoxification of heavy metals, drugs and environmental toxins (xenobiotics). While humans have 57 cytochrome P450 genes for xenobiotic metabolism, schistosomes have a single CYP450 gene. We have used reverse genetic and pharmacological approaches to validate <i>S. mansoni</i> CYP450 as an essential and druggable protein. Our research has identified a novel mechanism for heavy metal detoxification in schistosomes based on phytochelatin synthase, a worm-specific process.</p>					

Track for Translational Cancer Research

Barua, Animesh Ph.D.		Associate Professor		Animesh_Barua@rush.edu	
Department of Pharmacology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Our main focus is the translational research on gynecological malignancies including ovarian, leiomyosarcoma (a form of uterine cancer) and cervical cancers.</p> <p>Project-1: Molecular markers for Early detection of Ovarian Cancer (OVCA): OVCA is a fatal malignancy of women. In most cases it is detected at late stages with <30% 5-year survival rates of patients. Survival rate increases remarkably (90%) when OVCA is detected at early stage. This project examines early changes in DNA damage repair mechanism associated with OVCA development to establish a non-invasive early detection test for OVCA.</p> <p>Project-2: Development of targeted imaging agents and enhancement of resolution of Ultrasound imaging for OVCA detection: Ultrasound imaging is the currently available non-invasive imaging method for the detection of ovarian abnormalities including OVCA. However, due to its limited resolution, ultrasound imaging cannot detect OVCA at early stage. In this project we are developing targeted contrast agents for enhancing the resolution of ultrasound signals for early detection of OVCA.</p> <p>Project-3: Development of Immunotherapies against OVCA: Cancer chemotherapeutics are toxic to normal tissues and affects the quality of life especially in postmenopausal women with OVCA. Immunotherapies are safe and offer effective alternatives to chemotherapeutics. In this project, we are testing herbal products to enhance tumoricidal functions of NK cells.</p> <p>Project-4: Transition of Leiomyoma (fibroids) to Leiomyosarcoma: Uterine fibroids are common benign tumors of the uterus affecting around 70% of women of reproductive age. However, emerging information shows that a subset of fibroids transforms to malignant leiomyosarcoma. In this project we are examining factors associated with the transition of fibroids into leiomyosarcoma.</p> <p>Project-5: Development of pain medication alternative to opioids for patients with endometriosis. Endometriosis is a benign disease of women where a type of uterine cells (endometrial) are translocated to distant organs including the ovaries and fallopian tubes as well as several other tissues. Endometriosis is associated with extreme pain and affects fertility. This is a pilot study to understand the molecular feasibility of non-opioid alternative pain medication for patients with endometriosis.</p>					

Asadi, Farrokh DVM, MPVM, PhD, SI		Associate Professor		<u>farrokh_asadi@rush.edu</u>	
Department of Adult Health and Gerontological Nursing					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>During early stages of prostate cancer, tumor cells are dependent on androgen as a growth factor and withdrawal of androgen results in apoptotic cell death and clinical remissions. However, tumor recurrences after therapeutic interventions are commonly associated with increasing androgen independence of the tumor cells and increasing resistance to pro-apoptotic and chemotherapeutic agents. Overexpression of interleukin-6 (IL-6) and parathyroid hormone-related peptide (PTHrP) have been implicated in prostate cancer progression and bone metastases. The interactions between prostate cancer cells and bone marrow derived mesenchymal stem cells (BM-MSCs) also appear to be critical in the development of androgen-independence and bone metastases. IL-6 induces the expression of PTHrP in human osteoblastic cells, a signature of androgen-independence and osteogenic differentiation of tumor cells. However, no studies have directly tested the IL-6 mediated PTHrP effects in prostate cancer progression. Previously, we have shown that the expression of PTHrP is enhanced in prostate cancer as compared with benign prostatic hyperplasia and is greater in poorly differentiated carcinoma as compared with the well-differentiated tumors. We have also reported that PTHrP is highly expressed in androgen-independent prostate cancer cell lines and the expression of PTHrP may contribute to the apoptosis-resistant phenotype in prostate cancer cells. Additionally, we demonstrated that the adenovirus E1A oncoprotein represses PTHrP promoter and mRNA expression in androgen-independent prostate cancer cell line. Furthermore, we have reported that E1A repression of PTHrP expression increases the caspase-3 activation and sensitivity of androgen-independent prostate cancer cell line to apoptosis triggered by tumor necrosis alpha. We hypothesize that: 1) IL-6 induction of PTHrP is crucial in tumor progression and androgen-independent growth of prostate cancer; 2) IL-6 inhibitors will repress PTHrP expression and provoke apoptosis in androgen-independent prostate cancer cells. Strategies to measure increased co-expression of IL-6 and PTHrP in prostate cancer patients may inform the development of clinical assays to reliably predict the prostate cancer recurrence</p>					

Borgia, Jeffrey Ph.D.		Associate Professor, Director - University Cancer Center Biorepository; - Biomarker Development Core; Translational Cancer Research Track		Jeffrey_A_Borgia@rush.edu	
Department of Biochemistry					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>My research is focused on the use of high-throughput proteomic and genomic methodologies to better understand the biological mechanisms that underlie disease progression in patients with lung cancer. It is our ultimate goal to use these insights to develop improved diagnostic tools that aid physicians treating these people and promote improved quality of life and long-term survival. Working hand-in-hand with the physician groups at Rush, we use a range of technologies for these investigations that include Luminex-based immunoassays, novel immunoproteomic and quantitative proteomic methods for biomarker discovery driven by mass spectrometry, and expression proofing via RNA sequencing and/or microarray technology. Current research efforts focus on understanding the role of tumor metabolism in disease progression, discovery and characterization of surrogate biomarkers to better monitor aggressive tumor phenotypes, adoption of stem-cell like characteristics, and immune system monitoring.</p>					

Chen, Di MD/Ph.D.		Professor		<u>di_chen@rush.edu</u>	
Department of Biochemistry					
MS/Ph.D. Students for Laboratory Rotations	No	Master Thesis Students	No	PhD Dissertation Students	Yes
<p>Cyclin D1 SUMOylation and breast cancer development - Cyclin D1 is abnormally up-regulated in several types of cancers, especially in breast cancer (over 50% of breast cancer tissues have up-regulated cyclin D1). We found that cyclin D1 protein is SUMOylated and subsequently degraded in an ubiquitin/proteasome-dependent mechanism. We mapped SUMOylation site in cyclin D1 protein and identified a specific E3 ligase recognized SUMOylated cyclin D1. We are now testing if a SUMO activator (chemical compound) could inhibit breast cancer cell growth <i>in vitro</i> and <i>in vivo</i>.</p>					

Cohen, Frederic Ph.D.		Professor		Fredric_Cohen@rush.edu	
Department of Molecular Biophysics & Physiology					
MS/Ph.D. Students for Laboratory Rotations	No	Master Thesis Students	No	PhD Dissertation Students	Yes
<p>Cancer: We are studying how the chemical potential of cholesterol in cellular plasma membranes regulates cellular pathologies. The concentration of cholesterol is easily and routinely measured, but it is chemical activity, and not concentration, of a substance, including cholesterol, that has biological relevance. Chemical activity is the “effective” concentration and is rigorously quantified by chemical potential. A major obstacle to understanding has been the lack of an accurate procedure to measure cholesterol’s chemical potential. We have recently developed such a procedure. Implementing it, we have found that the chemical potential of cholesterol within plasma membranes significantly increases when cells become inflamed. This chemical potential also increases when cancer cells become metastatic. Further, we have developed a means to control and fix this chemical potential, and in doing so we found that preventing the chemical potential from increasing severely reduces metastasis and cellular inflammation. It thus appears that the chemical potential of membrane cholesterol is a key control in both inflammation and metastasis, and that interventions that regulate this chemical potential may be critical in controlling important pathologies.</p>					

Forsyth, Christopher B. Ph.D.		Associate Prof. ,Director, Basic Research, Digestive Diseases and Nutrition		Christopher_Forsyth@rush	
Dept. of Internal Medicine/Gastroenterology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Christopher B. Forsyth, PhD, AGAF is an Associate Professor of Medicine and Biochemistry. He received his PhD in Immunology-Microbiology from Loyola University-Chicago and his post-doctoral study was in vascular immunology at the Cleveland Clinic Foundation. He is the Director of Basic Research for the Division of Digestive Diseases and oversees the research laboratory for the Division of Digestive Diseases. His research focuses on intestinal inflammation and gut permeability as well as the microbiota role in diseases and microbiota-brain-gut axis, disruption of circadian rhythms, and effects of alcohol. Major diseases of interest in human studies and mouse models as well as in vitro models include inflammatory bowel disease, colon cancer, alcoholic liver disease, Parkinson’s disease, HIV/AIDS, obesity and metabolic syndrome and circadian rhythm disruption.</p>					

Gupta, Vineet Ph.D.		Vice Chair for Research and Innovation		Vineet_Gupta@rush.edu	
Department of Internal Medicine/ Immunology/Microbiology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Vineet Gupta, PhD is a member of the Departments of Medicine and Microbiology/Immunology. We are applying cross-disciplinary approaches to develop novel therapeutics in two research areas:</p> <ol style="list-style-type: none"> <u>Modulating innate immune response in Inflammation and Cancer:</u> Several years ago, we made a paradigm-changing observation that integrin activation is a novel mechanism for targeting innate immune cells. Subsequently, we identified novel small molecules to target integrin CD11b/CD18 (Mac-1) on neutrophils and macrophages and used it to show that such agents are highly efficacious in reducing cardiovascular inflammation and injury. Current studies are focused on unraveling its molecular and cellular signaling mechanism in models of autoimmune diseases (lupus and lupus nephritis). We have also made a surprising finding that these agents target tumor associated macrophages (TAMs), modify tumor microenvironment and reduce tumor growth, thus also have a significant effort in Cancer Biology. We are currently completing studies to be able to initiate a new Phase I clinical trial with these agents. <u>Protecting podocytes from damage in Chronic Kidney Disease:</u> Podocytes form the filtration barrier in the kidneys and their damage is a central hallmark of all chronic kidney diseases. Therefore, we recently developed a first-of-its-kind high content screening assay to identify compounds to protect podocytes from injury. Current studies in the laboratory are focused on understanding the molecular and cellular signaling mechanism of our novel hits as well as their efficacy in models of diabetic kidney disease and FSGS. <p>The laboratory currently has one junior research faculty member, three post-docs and three graduate students, in addition to several undergraduate interns. For further information please go to: http://guptalab.com</p>					

Maki, Carl Ph.D.		Associate Professor		<u>carl_maki@rush.edu</u>	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>We are examining two areas:</p> <p>P53 and metabolism: P53 is a tumor suppressor and potent cell growth inhibitor. P53 is kept at low levels by MDM2, an E3 ligase enzyme that binds p53 and promotes its degradation. However, p53 is stabilized and activated in response to DNA damage and other stresses. Activated p53 inhibits cell growth by inducing either a cell cycle arrest or apoptosis. We are examining how this decision (apoptosis vs arrest) is made. The ability of p53 to alter cancer cell metabolism is also believed to be important for tumor suppression. We are examining how p53 alters cancer metabolism, and how this affects cell fate.</p> <p>A novel breast cancer protein: We identified a protein that regulates signaling by G-</p>					

protein coupled receptors and also regulates breast cancer proliferation and metastasis. High expression of this protein is correlated with worse patient outcome. We are examining how this protein controls breast cancer growth and metastasis, and if targeting this protein can inhibit breast tumor growth in animals.

Marzo, Amanda L Ph.D.		Assistant Professor		Amanda_Marzo@rush.edu	
Department of Internal Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>The overall goal of the lab is to understand the mechanisms underlying the induction and maintenance of tumor specific memory CD8 T cells. Over the more recent years the role of metabolism in cancer growth and cell survival has been an area that is growing in interest. A well-known metabolic hallmark of tumor cells is the shift in glucose metabolism from oxidative phosphorylation to lactate production in order to produce energy. Furthermore several key signaling pathways, oncogenes, and tumor suppressors have been linked to an increase in glycolysis seen in tumor cells. In addition cancer cells are highly proliferative and also need to produce excess lipids, nucleotides, and amino acids. In order to do this, a number of metabolic adaptations occur in cancer cells that help generate these metabolites, fuel growth, and may also aid in the evasion of apoptosis. The aim of this project is to characterize 1) the metabolic changes in effector CD8 T cells that accumulate within the tumor microenvironment and how PD-1 signaling impacts metabolism and 2) to identify factors that control metabolic adaptability of effector and memory CD8 T cells. This study would provide a link between nutrient availability and PD-1 and provide evidence that there is a metabolic basis for CD8 T cell exhaustion in cancer and targeting CD8 T cell metabolism could be effective in reversing exhaustion. Overall these findings will identify novel pathways in T cell metabolism that could be targeted to enhance the function and survival of tumor reactive T cells.</p>					

Morgan, Deri Ph.D.		Assistant Professor		Deri_Morgan@rush.edu	
Department of Physiology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>Dr. Morgan The Lab is currently investigating Potential roles for proton channels (Hv1) as a treatment option in breast cancer. Triple negative tumors currently have no treatment outside of chemotherapy and survival rates are poor. Triple negative breast cancers do not express estrogen, progesterone or HER-2 receptors (these are the most common and effective targets for breast cancer treatment). Recent studies and preliminary data have shown that highly metastatic breast cancer cells express the voltage gated proton channel (Hv1). This protein is NOT found in healthy breast tissue and we propose that breast cancer cells are using this to enhance their function. Furthermore, greater Hv1 expression appears to correlate with larger tumor size, more</p>					

advanced clinical stage, lower recurrence-free survival, and shorter overall survival. Our studies have shown that inhibiting the expression of Hv1 suppresses in-vivo tumor growth, demonstrating its functional significance. However, the mechanism by which Hv1 supports the growth of cancer cells remains unknown and methods to exploit it in treatments are yet to be discovered. Elucidating these mechanisms will lay the foundation for new targeted treatments. We are investigating the role Hv1 plays in regulation of intracellular acid, oxygen radicals, Calcium signaling and metabolism in triple negative breast cancer cells as avenues that Hv1 may enhance these cells pathogenic potential. We utilize a range of electrophysiological techniques, genetic manipulations, cell based assays and in-vivo methods to accomplish this.

Pratap, Jitesh Ph.D.		Assistant Professor		<u>jitesh_pratap@rush.edu</u>	
Department of Anatomy and Cell Biology					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Metastasis is a step in advanced cancer development that is lethal as cancer cells often spread to bone. Metastasis to bone is associated with liberation of growth factors from the bone matrix, which can feed back to enhance tumor growth resulting in the 'vicious cycle' of bone metastases. We are investigating cellular and molecular events during bone and cancer cell interactions that influence cancer cell survival. We combine molecular and cell biological approaches to address two aspects of bone metastasis:</p> <p><u>Growth factor signaling:</u> Our focus is to understand the survival mechanisms of metastatic cancer cells in the bone microenvironment. In the bone microenvironment, metastasized cancer cells activate signaling pathways for growth factors. We identified, in bone-derived populations of metastatic breast cancer cells, a novel regulatory axis for growth factor receptor (IGF-1R) via the Runx2 master transcription factor. Our current studies center on crosstalk between Runx2 and growth factor signaling in metastatic cancer cells. We utilize cutting-edge molecular approaches and involve state-of-the-art live animal bioimaging to address our biological questions.</p> <p><u>Metabolism and Autophagy:</u> The unique biochemical characteristics of the bone niche provide homing signals to the cancer cells. Cancer cells have high-energetic and anabolic needs and are known to adapt their metabolism by activating distinct nutrient metabolic pathways to be able to survive and keep proliferating under conditions of nutrient stress. Currently, we are examining the mechanisms of autophagy during metabolic stress by which metastatic cancer cells survive in the bone microenvironment.</p>					

Track for Function and Disorders of the Musculoskeletal System

AI-Ghoul, Kristin Ph.D.		Associate Professor		kristin_j_alghoul@rush.edu	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>The function of the ocular lens is variable focusing of light onto the retina. Cataracts are a loss of lens transparency that precludes this function, leading to visual impairment. My lab is primarily interested in the structural basis of cataract formation. Additionally, normal lens growth and differentiation of lens fiber cells depends on proper fiber end migration and elongation of lens fiber cells. We are exploring both normal and aberrant migration in several animal models. Current projects include: (1) Aberrant fiber end migration in diabetes and retinal degenerative disease. (2) The role of inflammatory cytokines in diabetic cataract formation. (3) The structural alterations to the lens after combined alcohol and circadian disruption.</p>					

Chen, Di MD/Ph.D.		Professor		di_chen@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>We are working on following projects which use transgenic mice as disease models:</p> <ol style="list-style-type: none"> 1. Skeletal development - We are now investigating the role of Axin1 and Axin2 (key negative regulators of β-catenin signaling) in long bone development. We are also investigating the interaction between Axin/β-catenin and BMP/Smad signaling during skeletal development. 2. Molecular mechanisms of genetic bone diseases - a) Post-Axial Limb Hypoplasia, including tarsal coalition, fibular hemimelia, and proximal femoral focal deficiency (PFFD); b) Humeroulnar-radial-synostosis. (<i>Axin1^{Ptx1}</i> conditional KO mice, <i>Axin1^{Ptx1}/Axin2^{+/-}</i> double KO mice) 3. Cartilage biology - We have been investigating roles of Wnt/β-catenin, TGF-β/Smad3, and BMP/Smad1/5 signaling pathways in cartilage biology. (<i>β-catenin(ex3)^{Col2ER}</i> conditional activation mice, <i>β-catenin^{Col2ER}</i> conditional KO mice, <i>Tgfbr2^{Col2ER}</i> conditional KO mice, <i>Bmp2^{Col2ER}</i> conditional KO mice, <i>Bmp2/Bmp4^{Col2ER}</i> double KO mice). 4. Regulation of mesenchymal stem cell (MSC) fate. (<i>Periostin-CreER;Rosa^{mT/mG}</i> mice, <i>Osx-CreER;Rosa^{mT/mG}</i> mice). 5. Molecular mechanisms of arthritis - a) Pathological mechanism of Osteoarthritis (OA) (<i>Tgfbr2^{Col2ER}</i> conditional KO mice); b) Pathological mechanism of Spondyloarthritis (SpA) (<i>β-catenin(ex3)^{Col2ER}</i> conditional activation mice, <i>β-catenin(ex3)^{Agc1ER}</i> conditional activation mice, <i>β-catenin(ex3)^{Gli1ER}</i> conditional activation mice). 					

Hallah, Nadim Ph.D.		Professor		<u>Nadim_Hallab@rush.edu</u>	
Department of Orthopedics, Immunology, Anatomy Cell Biol.					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr Hallab teaches graduate courses in biomaterials and biocompatibility. He is the founder and CEO of two small companies that perform services for the orthopedic industry which act as a catalyst for collaborations between academia and industry. Dr Hallab's research involves the study of implant degradation and biologic reactivity to soluble and particulate implant debris with four areas of focus: 1) Immune reactivity to implant debris, from both an adaptive (T-cell) and innate (macrophage) perspective, 2) Implant connections (modular junctions) and implant fretting corrosion, metal release and metal-protein complex formation, 3) Peri-implant cell toxicity responses to implant degradation products such as metals, 4) Study of how material surfaces can be used to control immune and cell function such as bone deposition. Over the years his group has found different types of implant debris (ions vs particles) bind to different specific serum proteins in people with total joint replacements, these differences translate into quantifiable person- and material-specific immune responses that can be used as diagnostic measures of performance. They have developed methods for diagnosing metal sensitivity that are being used clinically to help people with or receiving orthopedic implants. They have quantified toxicity responses of many implant metals (e.g. Al, Co, Cr, Cu, Fe, Mo Nb, Ni and Zr) to peri-implant cells. The aim of these areas is to enhance implant performance through greater understanding and interventional strategies in the field of orthopedic-immunology.</p>					

Hannah J. Lundberg, Ph.D.		Assistant Professor		Hannah_Lundberg@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Hannah J. Lundberg, PhD is an Assistant Professor in the Department of Orthopedic Surgery. She is interested in the biomechanical function of human joints (natural and implanted). Her laboratory combines novel computational and experimental modalities to better represent joint function in vivo and improve surgical outcomes. Current emphases are using computer modeling to predict: 1) total knee replacement forces and behavior during everyday life, 2) wear of total knee replacements, and 3) the biomechanical behavior of total hip replacement modular taper junctions.</p>					

Natarajan, Raghu, Ph.D.		Professor		Raghu_Natarajan@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>Transition Syndrome: Biomechanical contributions to Adjacent Segment Disc Disease: Several in vivo and invitro studies have shown increased mobility of the adjacent spinal segment as a result of fusion or placement of disc replacement or degenerative disc disease or a combination of all these variables. There are large number of variables that can cause this disease such as fusion of single or multiple discs, single or multiple disc degeneration of varying grades of degeneration, alignment of the spine during fusion, instrumentation used for fusion, osteoporosis of the vertebra etc. The research involved is as follows: Build a 3D finite element model of a lumbar spine and introduce these variables into the model and provide an order of priority list of the variable which will help the clinicians to better serve the patient.</p>					

O'Keefe, Joanne, Ph.D.		Assistant Professor		<u>Joan_a_okeefe@rush.edu</u>	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr. O'Keefe's expertise is in measurement of gait and balance impairments in movement disorders using state-of-the-art technologies. Our research program is concentrating on the neurodegenerative disorder known as Fragile X Tremor and Ataxia Syndrome (FXTAS) which occurs in some carriers of the Fragile X gene. It is well known that neurodegenerative disorders begin long before abnormalities can be observed on a neurological exam. One of our goals is to develop an early detection model for FXTAS and identify additional molecular risk factors for developing this disorder in collaboration with Drs. Elizabeth Berry-Kravis and Deborah A Hall, world renowned Fragile X Syndrome and FXTAS experts, respectively. We are also investigating the impact of cognitive dysfunction on gait and balance impairments using neuropsychological testing and what are known as "dual task" cognitive interference motor tests in FXTAS, Parkinson's disease, Huntington Disease, and Essential tremor. These studies will lay the foundation for identification of fall risk in these patient populations and eventually in the development of preventative treatment strategies. We are also using our sensitive motor outcome measures in exciting clinical trials in FXTAS and the neurodegenerative disorder known as Niemann Pick C disease.</p>					

Pourzal, Robin Ph.D.		Assistant Professor		Robin_Pourzal@rush.edu	
Department of Orthopedics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>The implant lifetime of total hip replacements (THR) is often limited by adverse local tissue reactions (ALTR) caused by metal ions and debris that are generated within modular junctions of the implant. My research focuses on corrosion processes and how they are related to the quality of the implant alloy, the surgeon's implantation technique, and the patient's physiology. The result of in vivo corrosion processes is evaluated by retrieval analysis which entails the implant components as well as periprosthetic tissue that have been retrieved during revision surgery or postmortem. Material loss of the implant can be quantified by using a 3D surface profiler and damage modes are evaluated by scanning electron microscopy. The type of tissue reaction and corrosion product can be determined by means of Immunohistopathology and Fourier Transformed Infrared Spectroscopy, respectively. The implant quality is characterized by means of standard metallographic methods. Additionally, patient characteristics will be collected (e.g. blood ion levels) in collaboration with orthopedic surgeons at Rush. This enables us to find correlation between material and patient factors, and the occurrence of specific corrosion processes, corrosion products and tissue reactions.</p> <p>The impact of the surgical technique will be determined in simulated THR surgeries, where THR assembly load impulses applied by surgeons will be measured during different surgical approaches. The gathered data can be utilized for finite element analysis (collaboration with Dr. H.J. Lundberg) to determine the optimal load impulse range to ensure sufficient stability of the modular junction to prevent micro-motion and corrosion.</p>					

Pratap, Jitesh Ph.D.		Assistant Professor		Jitesh_Pratap@rush.edu	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Deregulation of skeletal cell differentiation is a major cause of several skeletal diseases such as osteoarthritis and osteoporosis. Differentiation of these cells (e.g., osteoblasts and chondrocytes) is characterized by a rapid increase in gene expression and protein secretion activity. This differentiation requires high metabolism and can be regulated by autophagy. During autophagy, cytoplasmic components and damaged organelles are captured by autophagosomes followed by fusion and degradation by the lysosome resulting in release of metabolites as energy sources to meet metabolic demands. Very recent studies with cell type specific knockout mice of autophagy-related genes show that autophagy is required for differentiation and mineralization of osteoblasts and chondrocytes. However, the regulatory mechanisms of autophagy in these cells are unknown. We are investigating how autophagy is regulated during differentiation.</p> <p>To understand the regulation of autophagy, we use osteoblasts and chondrocytes from genetic mouse models of skeletal cell differentiation and</p>					

examine the autophagy pathway by biochemical, live cell confocal imaging and electron microscopic approaches. Our recent results show that autophagy is impaired in Runt-related transcription factor-2 (Runx2) deficient cells. We found that Runx2 facilitates acetylation of microtubules (MTs), a critical step in formation of autophagolysosomes. Based on these findings, our research is focused on 1) examining the mechanism of microtubule acetylation in Runx2-mediated autophagy and 2) Runx2-dependent autophagy control of cell metabolic pathways (glycolysis and oxidative phosphorylation). Understanding of these mechanisms can lead to the development of novel therapeutic strategies for skeletal diseases.

Rios, Eduardo Ph.D.		Professor, Section Head		Eduardo_Rios@rush.edu	
Department of Physiology & Biophysics					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	No	PhD Dissertation Students	Yes
The laboratory of Eduardo Rios works on the mechanisms of control of rapid calcium signals in excitable cells. While crucial to many cells, we focus on their workings in striated muscle, skeletal and cardiac, where they support movement, locomotion, and the cardiac beat. Currently we explore the roles of a protein, calsequestrin, which constitutes the main device for storage of the calcium ions that convey these signals. A number of proteins, including calsequestrin, link together mechanically in a supra-molecular device, named the couplon, which controls the calcium signals. Mutations of the couplon proteins cause a group of diseases, with both diverse and similar features, named "couplonopathies". Through studies in cells of patients, we are trying to understand the alterations in normal mechanisms that underlie these couplonopathies, with the long term goal of contributing to their treatment.					

Ross, Ryan D., Ph.D.		Assistant Professor		Ryan Ross@rush.edu	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
My research interests are skeletal mineralization and bone quality. I am currently working closely with Dr. Rick Sumner on two related projects: 1) Using a novel rat model of induced skeletal remodeling we will be testing the effects of pharmaceuticals, specifically those currently in use as treatments for osteoporosis, on the process of skeletal mineralization. This project will determine how these treatment impact the mineralization of bone tissue and assess the mechanical impact of altered mineralization. Relevant research techniques include; scanning electron microscopy, Fourier transform infrared microspectroscopy, micro-computed tomography, and three-point bending mechanical testing. 2) I am also working to establish the connection between two bone-derived proteins critical to bone formation and mineralization, sclerostin and fibroblast growth factor 23 (FGF23). This project aims to determine how sclerostin upregulates					

FGF23 and what impact this has on skeletal mineralization, with the hope of eventually targeting sclerostin as a means to correct diseases of hypophosphatemia. The first phase of this project is in vitro so relevant research techniques include; cell culture, RT-PCR, western blotting and ELISA. The second phase will involve use of a genetic mouse model; relevant research techniques include, mouse husbandry, micro-computed tomography, mechanical testing, ELISA, and RT-PCR.

Schmid, Thomas M. Ph.D.		Professor, Director Musculoskeletal Track		Tom_Schmid@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>My expertise is musculoskeletal biomarkers, collagen, cartilage, growth plate, cell culture, immunochemistry, mineralization, and lubrication.</p> <p>Project 1 – Role of superficial zone protein/lubricin/PRG4 in synovial joints. What cells make it? Can they be maintained in long term in culture? Which cells make the source of SZP in blood? Which cells make the sialidated form and which ones do not? Is this molecule a useful biomarker for musculoskeletal disease or forms of cancer?</p> <p>Project 2 – Characterization of Peyronie’s plaque tissue (post-surgery). What methods can be used to visualize the collagenous fibrotic plaque in Peyronies tissues? One third of these tissues mineralize, what kind of mineral is present in these tissues? How can it be dissolved in vitro and in vivo? What cells contribute to this disorder?</p>					

Sumner, Rick D. Ph.D.		Professor, Chair & Director MicroCT/Histology Core		Rick_Sumner@rush.edu	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	No
<p>My specific areas of research interest are bone regeneration, orthopedic implant fixation, and the role of bone in osteoarthritis. Over the last 30 years, my lab has been funded by the NIH, DoD, NASA, industry, private foundations, and philanthropic gifts. We frequently interact with other groups at Rush as well as with labs from throughout the U.S. and internationally. Currently, our projects are focused on (1) early detection and treatment of particle-induced peri-implant osteolysis, (2) bone matrix maturation in the adult skeleton, (3) the effect of space flight on bone quality, and (4) the relative timing of change in cartilage and bone during the development of osteoarthritis. Many of these projects rely on advanced imaging techniques performed here at Rush as well as in several national laboratories, including micro computed tomography, backscatter scanning electron microscopy, and Fourier transform infrared spectroscopy. Other major methods include histology, mechanical testing, biomarkers, and proteomics. Can it be easily dissolved? Under what conditions? What cells contribute to this disorder?</p>					

Viridi, Amarjit Ph.D.		Associate Professor		amarjit_virdi@rush.edu	
Department of Cell and Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>We use both <i>ex vivo</i> and <i>in vivo</i> approaches to enhance bone regeneration and to elucidate underlying mechanisms at the molecular, cellular and tissue levels.</p> <ol style="list-style-type: none"> 1. In situ tissue regeneration (Stem Cell Mobilization): This is a newer concept in regenerative medicine built on how the body reacts to injury and carries out the healing process. Cell recruitment to the injury site is an important component of the cascade of events that takes place throughout healing. This includes, but is not limited to, immune cells and stem cells from local as well as distant sites. The process of translocating stem cells from their resident niches throughout the body to the injury site is complex and may be a rate limiting step. We are working to test the hypothesis that deliberately mobilizing stem cells from niches and their recruitment/engraftment at the injury site with pharmacological agents will aid healing. While we are also interested in <i>ex vivo</i> expanded stem cells, we believe this approach of in situ tissue regeneration holds great potential and will be time- and cost-effective. 2. Anabolic enhancements (Local and Systemic Delivery): Growth-factor guided enhancement of bone has a proven track record in orthopedic research. In a number of publications we have shown that bone healing is enhanced and accelerated when cell stimuli are applied locally or systemically. We continue to test newer molecules and biologics in animal models of implant fixation and large bone defects. Much work still needs to be done to refine protocols for optimizing doses and treatment times/duration. 3. New generation of smart biomaterials: Orthopedic surgical procedures rely heavily on synthetic and biological materials. We are collaborating with investigators at UIC to develop and apply smart materials that can provide mechanical stability as well as allow improved bone formation on the engineered prosthetic surfaces. The approach involves texturing implant surfaces at the nanometer scale so that they are more conducive to cell attachment and differentiation. Bone grafts are commonly used for filling large gaps in orthopedic trauma or surgical resection. Allografts used in these procedures provide mechanical support but are slow to integrate with the host tissue. We are working to modify the allograft material to assist local cellular activity in augmenting tissue recovery and function. 					

Wimmer, Markus, Ph.D.		Professor, Director Gait Laboratory		Markus_A_Wimmer@rush.edu	
Department of Orthopedic Surgery					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Markus Wimmer, PhD is a Professor in the Department of Orthopedic Surgery with conjoint appointments in Cell and Molecular Medicine, and Rheumatology. He is the Director of two laboratories, the Human Motion Analysis lab and the Tribology lab. Research in the Motion Analysis lab seeks to improve the physical capabilities of those</p>					

who suffer from musculoskeletal ailments. Students and scientists study the functional performance of people during activities of daily living, measuring the kinematics and kinetics of natural and artificial joints. Current research involves exploring kinematics and stability of movement in total knee replacement, the pathomechanism of abnormal gait, and the development of rehabilitation strategies to either delay or halt the progression of osteoarthritis.

Tribology research applies physical principles of friction, wear, and lubrication to natural and artificial joints to improve the material properties of implants and the patient's well-being. The goal is to contribute to long-lasting treatment solutions for the osteoarthritic joint. Although, the main focus is directed towards artificial implants, there are also efforts to better understand the effects of loading and motion on living cartilage and bridge the fields of biomechanics, cell biology and chemistry. We are currently implementing multiple daily activities on joint simulators to conduct more accurate clinical wear testing. In tribocorrosion experiments we investigate the degradation mechanisms of taper joints. Particulate and ionic debris is then tested in cell cultures to estimate the biologic reaction. In addition, we started studying cartilage wear of living tissue using a joint bioreactor.

Track for Function and Disorders of the Nervous System

Lena Al-Harhi Ph.D.		Professor, Interim Chair		Lena_Al-Harhi@rush.edu	
Department of Microbial Pathogens and Immunity					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr. Lena Al-Harhi is a Professor and Interim Chair in the Dept. of Microbial Pathogens and Immunity at Rush University Medical Center in Chicago, IL. She is also the Program Director of Rush Initiative to Maximize Student Development (RUSH-IMSD), an NIH funded PhD training grant for underrepresented minority scientists enrolled in the Integrated Biomedical Sciences (IBS) program at Rush graduate college. Dr. Al-Harhi's research over the past 18 years has focused on HIV/host interactions, with a special emphasis on bridging basic and clinical science in the HIV/AIDS field. Because of her experience in HIV molecular biology, immunology, and for the past ten years in neuroAIDS, she has been able to probe mechanistic questions that are clinically relevant to HIV/AIDS. She has over 70 peer-reviewed publications and invited reviews/book chapters. Her research focus is on understanding the dynamic cross talk between Wnt/b-catenin signaling, inflammatory mediators, and HIV as they regulate HIV transcriptional activity and pathogenesis in the CNS. The Wnt/b-catenin pathway is vital for proper CNS development and homeostasis. Her group has identified the β-catenin signaling pathway as an important regulator of HIV replication in multiple compartments, including the central nervous system, and in playing key roles in HIV neuropathogenesis. Her lab is also evaluating the role of host factors (e.g. inflammatory mediators), HIV and co-variants (e.g. drugs of abuse) in modulating β-catenin which will in turn impacts overall CNS homeostasis. Further, she is investigating the role of astrocytes in HIV latency. Lastly, she has an interest in neuroimmunology, as it relates to anti-HIV responses in the CNS. Her lab uses cutting-edge methodologies and tools to address these important topics.</p>					

Arvanitakis, Zoe MD		Professor		Patricia_Boyle@rush.edu	
Rush Alzheimer's Disease Center and Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
Links among neurodegenerative disease, cognitive aging, and vascular disease in humans					

Elizabeth Berry-Kravis, MD/PhD		Professor		Elizabeth_Berry-Kravis@rush.edu	
Department of Pediatrics/ Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>I have been working in the field of fragile X syndrome and neurodevelopmental and neurodegenerative disorders since 1988 and have had a Fragile X Clinic, dedicated specifically to the care of patients with fragile X syndrome (FXS) and their families since 1992. In 2000-2002, in collaboration with researchers at UC Davis and University of Colorado, I participated in research leading to the initial discovery and characterization of FXTAS as a new disease, occurring in carriers of an <i>FMR1</i> premutation. A significant part of my research program has been devoted to studies of molecular relationships, clinical manifestations, risk factors, and biological and neuroimaging markers of FXTAS and FXTAS risk. I currently have the second largest Fragile X Clinic in the world and follow over 600 patients with FXS in clinic, and have seen over 200 family members with FXTAS through clinical referral or research studies. My laboratory has been performing <i>FMR1</i> genotyping since 1992, is CAP/CLIA certified for molecular diagnostics including <i>FMR1</i> testing for FXS, has run thousands of <i>FMR1</i> genotypes for clinical diagnosis and research projects, runs several DNA banks for FXS and other projects, has run genotyping for multiple studies evaluating gene-phenotype associations, and has recently implemented <i>FMR1/ASFMR</i> mRNA assays and an FMRP ELISA and Luminex assay. A major area of focus for my research program over the last decade has been translational research in FXS including the design and conduct of clinical trials of medications directed at treating underlying neural mechanisms. This work has included validation of outcome measures and biomarkers to assess treatment effects on cellular mechanisms of disease and characteristic clinical phenotypes in FXS. In the past 14 years, my center has enrolled over 250 FXS participants into 19 clinical trials for targeted treatment in FXS, and over 300 FXS subjects into outcome measure or biomarker studies. I have been overall PI on or Co-PI for 10 clinical trials in FXS and part of the design team or advisory board for all 20 trials in which my site has participated and 2 trials not at my site. I am a PI on the CDC-funded set of FORWARD projects to define the natural history of FXS, associate natural history phenotypes with biomarkers, and am piloting use of ERP to track neurophysiological progress of individuals with FXS. I have also run clinical trials in Down syndrome, Rett syndrome, Angelman syndrome, Neimann-Pick type C, PKAN, creatina transport deficiency, am a Co-I on RDCRN projects on the natural history of Rett syndrome and Phelam McDermid syndrome and am PPI of a NeuroNext trial to study a new plasticity/learning paradigm for drug development in FXS.</p>					

Burgess, Helen PhD		? Professor		Helen_Burgess@rush.edu	
Department of Neurological Sciences/ Department of Behavioral Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Sleep and circadian rhythm mechanisms, as well as their changes in relation to psychiatric and neurological disorders</p>					

Crowley-McWilliams, Stephanie PhD		Associate Professor		Stephanie_Crowley_McWilliam@rush.edu	
Department of Behavioral Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>I am trained as an Experimental Psychologist, and my broad research interest is sleep and circadian timing. Much of my work has been of human circadian timing with an emphasis on interventions, like bright light, exogenous melatonin, and timed sleep/wake schedules to correct circadian misalignment in situations like night shift work and jet lag. Over the past 10 years, my research has also focused on changes to sleep patterns and sleep regulatory systems during adolescent development; specifically, examining possible mechanisms underlying late sleep in teenagers, how these biological changes interact with a changing psychosocial context (e.g., early school start times), and developing behavioral interventions to increase sleep duration of adolescents.</p>					

Gaiteri, Chris PhD		Assistant Professor		Christopher_Gaiteri@rush.edu	
Rush Alzheimer's Disease Center					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>How can the growing body of biomedical information deepen our understanding of biological systems in a way that will revolutionize health care? While there is great potential for data-intensive approaches to lead this revolution, to date they have resulted in few effective treatments for neurodegenerative diseases. Therefore, approaches that utilize a wider range of available data, or which make more accurate predictions, are needed for a fundamental transformation of our drug discovery process. To deliver on the promise of data science for disease research, Chris Gaiteri develops such approaches that integrate diverse sources of information to assess the state of the brain in disease, and then accurately identify specific molecules that can push the brain back to a healthy state.</p> <p>In order to contribute to disease understanding and drug discovery, he take an integrated approach to computation, in the broader life-cycle of research. Within this integrated approach, he focuses on 1) combining different types of biological data and 2) making more accurate computational predictions. The integrated process starts even before experiments begin: meeting with wet lab biologists to discuss their goals and provide guidance on experimental design that will provide information with long term benefits. Then he oversees statistical aspects of data normalization, computational issues related to big data storage and processing. Because many diseases of aging have complex multi-system etiology, single data types are insufficient, so he begins to align information from multiple omics for high-confidence results. At this stage, the 2nd focus of his research - making more accurate predictions comes into play, as he builds causal models of intricate molecular regulatory mechanisms that utilize data from multiple sources. With these detailed computational models, we can accurately predict</p>					

key points for perturbation experiments. At this point, the life-cycle of experimentation begins again, as we either correctly predict key disease mediators, or acquire valuable information on where the models are incorrect and must be improved.

Applying this pattern of start-to-finish computational integration with biological experiments based on data from the Alzheimer's Disease Center will feed into collaborations with wet lab biologists and clinicians. Together we verify and refine the predictions to contribute to better health care programs for age-associated neurological disorders. Chris plans to continue interdisciplinary collaboration between theories and experiments, so that the integrated framework advances our understanding of neurodegenerative diseases.

Goetz, Chris MD		Professor		Christopher_Goetz@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
Director of the Movement Disorders Program. My major interest is the pharmacology of various movement disorders, including Parkinson's disease, Gilles de la Tourette's syndrome, Huntington's disease and pharmacologic studies of dystonia. I am particularly interested in non-motor aspects of movement disorders, including specifically, hallucinations, cognitive decline, and depression.					

Goldman, Jennifer MD		Associate Professor		Jennifer_Goldman@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
Dr. Goldman is conducting research studies to determine what causes cognitive, behavioral and emotional changes in patients with movement disorders, particularly Parkinson's disease, Dementia with Lewy Bodies, and Huntington's Disease, and how to improve treatments for these problems.					

Hall, Deborah MD/PhD		Associate Professor		Deborah_A_Hall@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
Dr. Hall's work in Parkinson's disease focuses on early interventions, such as neurotrophic factors and exercise, genetics and genomic causes of disease, and treatment for complications including falls. She also conducts research in the field of ataxia, specifically fragile X-associated disorders, by investigating epidemiology, clinical features of movement and balance, and interventions.					

Hu, Xiu Ti MD/PhD		Associate Professor		Xiu-Ti_Hu@rush.edu	
Department of Microbial Pathogens and Immunity					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
My research over the past 24 years has been mainly focusing on the elucidation of the mechanisms that underlie substance abuse-associated neuronal dysfunctions in the mesocorticolimbic dopamine system. In the past seven years, I also have applied my expertise in drug addiction research and electrophysiology to explore potential mechanisms that underlie HIV neuropathogenesis and the comorbidity of drug addiction (e.g., cocaine and methamphetamine) and neuroAIDS. My studies reveal a critical role of L-type Ca ²⁺ channel dysregulation (e.g., over-activation/expression) in HIV-induced dysregulation of the medial prefrontal cortex; and methamphetamine or cocaine-induced dysregulation of L-type Ca ²⁺ channel and/or K ⁺ channels in cortical and striatal neurons, as well as in astrocytes. My current research focuses on defining potential mechanism(s) by which methamphetamine/cocaine, HIV-1 proteins, and anti-retroviral drugs alter functional activity of forebrain neurons and astrocytes; and on evaluating potential novel therapeutic strategies that will exploit the usefulness of reducing cortical neuronal hyperactivity mediated by L-channels in HIV-associated neurocognitive disorders (HAND).					

Koralnik, Igor MD		Professor and Chair		Igor_Koralnik@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
Dr. Koralnik is the Chair of the Department of Neurological Sciences at RUMC. His laboratory is involved on investigating the clinical, translational and basic science aspect of viral infections on the Nervous System. In particular, Dr. Koralnik's group has been investigating the pathogenic aspects of the polyomavirus JC, the agent of Progressive Multifocal Leukoencephalopathy (PML). They have discovered and characterized other conditions associated with JC virus infections of neurons, meningeal and choroid plexi cells. The Koralnik laboratory is now investigating the effects of Zika virus on the nervous system, and are characterizing the entire virome in clinical samples using a					

novel, deep sequencing based platform called ViroFind.
Finally, the Korálnik Lab is also studying the auto-immune mechanisms of Narcolepsy and Cataplexy, a sleep disorder associated with a specific HLA allele.

Kordower, Jeffrey PhD		Professor		Jeffrey_Kordower@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes

For a number of decades, I have been an international leader in the field of Parkinson's disease (PD) and related disorders. I have particular expertise in studies performed in rodents. My expertise is documented, in part, by the following: Ranked #75 as an author in terms of citations in the field of PD in the last 30 years and #96 in history (Movement Disorders, 2011); Founding and current member of the Michael J. Fox Foundation (MJFF) Scientific Advisory Committee, Member of the MJFF Executive Scientific Advisory Committee, Past-President of the American Society for Neural Transplantation, and current member of the Movement Disorders Society International Executive Committee. I have published over 400 peer reviewed manuscripts and chapters, most in the area of PD and related disorders. Many of these papers are citation classics such as the ones documenting the first surviving fetal grafts in patients with PD (e.g.; Kordower et al., New England J. of Medicine, 1995), the first demonstration that gene delivery of GDNF was effective in nonhuman primate models of PD (Kordower et al. Science, 2000), the first demonstration that dopaminergic stem cells can survive, maintain phenotype, and restore function in a rodent model of PD and survive in a nonhuman primate model of PD (Kriks et al., Nature, 2013), the co-discoverer with Patrik Brundin that fetal dopaminergic grafts surviving in patients for greater than 10 years can develop Lewy bodies (Kordower et al.; Nature Medicine, 2006). I have also coauthored studies reporting ground breaking transplantation (e.g. Olanow et al., 2003) and gene therapy (e.g. Olanow et al, 2015) clinical trials. I have translated 6 different therapeutic strategies (both cell and gene therapy) from rodent and nonhuman primate studies to clinical trials. Indeed, my experience in nonhuman primate models of PD, cell replacement strategies, and their translation to clinical trials serves well the path of the lab's current direction.

Verhagen Metman, Leo MD/PhD		Professor		Leonard_A_Verhagen@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes

Deep brain stimulation and other treatments for movement disorders

T. Celeste Napier, PhD		Professor		Celeste_Napier@rush.edu	
Department of Psychiatry					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Dr. T. Celeste Napier is a Professor in the Departments of Pharmacology and Psychiatry at Rush University Medical Center, Chicago, IL. She also is the Director for the Center for Compulsive Behavior and Addiction at Rush University.</p> <p>Dr. Napier's research revolves around substance use disorders (methamphetamine, cocaine and opioids) and impulse control disorders (gambling disorders), including those that are comorbid with neurological disease (Parkinson's disease, neuroAIDS). Her research pursues the subcellular, neurophysiological and behavioral consequences of reward-motivated behavior using rodent models of human brain disease. Ongoing projects include the following:</p> <p>Methamphetamine abuse and Parkinson's disease. Recent studies of methamphetamine-abusing humans indicate an increase in the propensity to develop Parkinson's disease later in life. This project seeks to determine the underlying mechanisms that drive this phenomenon. Methamphetamine self-administering rats are used to examine Parkinson's disease-like pathology in the brain and gut.</p> <p>Psychostimulant abuse comorbidity with HIV/AIDS. This project evaluates the convergence of HIV-1 proteins (e.g., the transactivator of transcription [Tat] and in HIV-1 transgenic animals) with the neurophysiological changes imposed by cocaine or methamphetamine self-administration. The comorbidity on inflammation in the brain and gut is also studied.</p> <p>Impulse control disorders in Parkinson's disease. Dopamine agonists are an effective and widely used therapy to improve the motor symptoms of Parkinson's disease. But a significant portion of treated patients develop various forms of impulse control including problem gambling, hypersexuality and compulsive behaviors (shopping, eating). For this project, rats performing delayed and probability discounting tasks wherein intracranial self-stimulation serves as the positive reinforcement are used to study impulse control. Dopamine receptor-mediated subcellular changes imposed by chronic exposure to dopamine agonists signal transduction are examined. These approaches are used to identify targets that will allow for treating the motor symptoms of Parkinson's disease without involving neuronal pathways that regulate impulsivity.</p> <p>Medication development for addictions. The above described methods are being used to test the utility of compounds to reduce drug and behavioral addictions and impulse control disorders. Focus is on compounds that engage glutamatergic and serotonergic systems.</p>					

Nicholson, Dan PhD		Associate Professor		Dan_Nicholson@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>The laboratory of Dan Nicholson, PhD, is interested in the neurobiology of cognitive aging, Alzheimer's disease and epilepsy. We bring to bear numerous techniques to unveil the events and pathogens that ultimately lead to brain failure, including electron microscopy, patch-clamp physiology, immunofluorescence array tomography, immunoprecipitation assays and mass spectrometry.</p> <p>The majority of our work utilizes animal models of cognitive aging and Alzheimer's disease. With our colleagues, John Disterhoft, PhD, and Matt Oh, PhD, at Northwestern University, we use 2-photon glutamate uncaging and 2-photon calcium imaging of individual dendritic transients in mouse models of Alzheimer's disease and behaviorally characterized rats, ranging in age from four to 30 months of age. In the mouse models of Alzheimer's disease, the vast majority show learning and memory impairments by mid-life. Among aged rats (~28 months of age and older), some learn hippocampus-dependent behaviors as well as young adult rats (aged, cognitively unimpaired), whereas others show severe learning impairments (aged, cognitively impaired). We use animal models to investigate the cellular mechanisms of both Alzheimer's disease-linked (i.e., the mouse models of Alzheimer's disease) and non-Alzheimer's disease-linked (i.e., the aged impaired rats) cognitive failure. After recording synaptically evoked, individual dendritic calcium transients, we reconstruct the imaged dendrites using the super-resolution light microscopy technique array tomography and probe for ion channels and other important signaling proteins with single-dendrite, single-spine resolution. This allows us to generate single-dendrite ion channel gradients for dendrites that represent the normal range of signaling in mice and rats (i.e., similar to wildtype/young adult values), as well as those dendrites with signaling abnormalities. We therefore have multidimensional abstractions of dendrites with both normal and abnormal calcium signaling, allowing us to identify the ion channel or channels that are tied to signaling problems.</p> <p>A powerful aspect of this approach is that we can use array tomography on brain tissue from human Alzheimer's disease, mild cognitive impairment and non-cognitively impaired cases to perform the same exact experiments on neurons that have been filled iontophoretically with fluorescent dye. We can then infer how dendrites in the human brain might have signaled by comparing their multidimensional signaling interactome to those from the rat and mouse experiments.</p>					

O'Keefe, Joanne PhD, PT		Associate Professor		Joan_A_Okeefe@rush.edu	
Department of Cell & Molecular Medicine					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>I am an Associate Professor in Cell and Molecular Medicine with conjoint appointments in Neurological Sciences and Occupational Therapy. My expertise is characterizing gait</p>					

and balance impairments and the interaction of cognition in a variety of movement disorders. I am a Physical Therapist with doctoral/postdoctoral training in Neuroscience. I collaborate with numerous faculty in Movement Disorders and Pediatrics. I predominantly study Fragile X Tremor and Ataxia Syndrome (FXTAS) which occurs in some premutation carriers of the Fragile X gene. I am working on the following projects:

- 1) an NIH funded project to develop an early detection model for FXTAS using cutting edge, inertial sensor technologies to measure balance and gait and interacting molecular risk factors for its development (in collaboration with Dr. Deborah Hall in Movement Disorders)
- 2) comparing the distinct motor and cognitive phenotypes in FXTAS, Parkinson's disease and essential tremor in order to reduce misdiagnosis in FXTAS.
- 3) a collaboration with Dr. Elizabeth Berry-Kravis in the conduct of a clinical trial to determine the efficacy of the drug Hydroxypropyl Beta Cyclodextrin in Niemann-Pick C, a neurodegenerative disorder in children and young adults.
- 4) a dual task cognitive interference study to characterized its impact on motor function and fall risk in Huntington's disease (in collaboration with Dr. Jennifer Goldman in Movement Disorders).
- 5) creating a normative database of gait and balance outcome variables using inertial sensor based technologies that we anticipate will be very useful in the conduct of future clinical treatment trials in children and young adults with neurodegenerative disorders.
- 6) comparing balance and gait in Parkinson's disease patients who carry a GBA mutation versus non-mutation carriers (in collaboration with Dr. Gian Pal in Movement Disorders).
- 7) rehabilitative intervention trials in FXTAS
- 8) assessment of turn strategies in cerebellar ataxias
- 9) development of technologies/algorithms to quantify repetitive stereotyped behaviors in neurodevelopmental disorders such as autism.

Gait and balance impairments and the interaction of cognition in a variety of movement disorders (FXTAS, Parkinson's disease).

Pahan, Kalipada PhD		Professor		Kalipada_Pahan@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes

One of the pathologic hallmarks of Alzheimer's disease (AD) is the presence of extracellular amyloid plaques containing A β peptides, which originate from the amyloidogenic proteolytic processing of amyloid precursor protein (APP), through the sequential action of β - and γ -secretases. In contrast, APP can also be cleaved by a non-amyloidogenic pathway by α -secretase, precluding the formation of A β peptides. However, mechanisms by which non-amyloidogenic metabolism of APP is controlled are poorly understood. Peroxisome proliferator-activated receptor (PPAR) α is a transcription factor that regulates fatty acid metabolism. Although hippocampus does not metabolize fat, recently we have delineated that PPAR α is constitutively expressed in nuclei of hippocampal neurons (Roy et al. & Pahan, 2013, Cell Reports 4:724-737) and astrocytes (Roy et al. & Pahan, 2015, Cell Metabolism, 22:253-265). Surprisingly, PPAR α controls the metabolism of APP via direct transcriptional regulation of ADAM10 α -secretase (Corbett et al. & Pahan, 2015, Proc. Natl. Acad. Sci. USA 112:8445-8450). Therefore, in

an attempt to isolate novel physiological drugs to control plaque formation and memory loss in AD, we have isolated a few ligands of PPAR α from normal hippocampus. We will examine if these hippocampal drugs stimulate the metabolism of APP towards the α -secretase pathway in hippocampal neurons and if treatment with these drugs reduces cerebral A β load and improves memory and learning in an animal model of AD via PPAR α .

Persons, Amanda PhD		Assistant Professor		Amanda_L_Persons@rush.edu	
Department of Psychiatry					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes

Our research focuses mainly on substance use disorders and Parkinson's disease. We seek to determine how methamphetamine abuse increases the risk to develop Parkinson's disease, and how cocaine or methamphetamine abuse exacerbate the effects of HIV-1 proteins in the brain/gut axis. We also wish to determine how certain dopamine therapies for the treatment of Parkinson's disease leads to impulse control disorders, and how to attenuate those behaviors with adjunct therapy.

Stebbins, Glenn 'Skip' PhD		Professor		Glenn_Stebbins@rush.edu	
Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes

Dr. Glenn Stebbins, is a professor in the Department of Neurological Sciences at Rush University Medical Center. His research interests center on the effects of normal and pathological aging on cognitive function in humans. Using advanced neuroimaging (e.g., fMRI, Diffusion Tensor Imaging, SPECT) and behavioral techniques, his studies are designed to assess the relationship between structural and functional changes in the CNS and age-related behavioral changes. In addition, Dr. Stebbins has extensive experience with rating scale testing and development, having participated in the development of the Unified Dyskinesia Rating Scale (UDysRS) as well as serving on the Steering Committee of the Movement Disorder Society revision of the Unified Parkinson's Disease Rating Scale (MDS-UPDRS). He is actively involved in the development of validated non-English translations of rating scales including the UDysRS and MDS-UPDRS. He currently serves as the Co-Chairperson of the Rating Scale Program for the International Parkinson and Movement Disorder Society. This Program oversees a portfolio of 13 rating scales used in movement disorders and is focused on the validation of existing rating scales as well as the development of new rating scales. Dr. Stebbins serves as consultant and chief statistician to numerous clinical studies in Parkinson's disease and other movement disorders.

Stoub, Travis PhD	Assistant Professor	Travis_Stoub@rush.edu
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Department of Neurological Sciences					
MS/Ph.D. Students for Laboratory Rotations	Yes	Master Thesis Students	Yes	PhD Dissertation Students	Yes
<p>Travis Stoub, Ph.D. is currently Co-Director of the Rush Epilepsy Center Multimodality Neuroimaging and Neuroengineering Laboratory whose research interests focus on using multimodal neuroimaging and neurophysiological methods to develop novel markers for epilepsy and its comorbidities. He is currently principal investigator on a study that aims to develop structural and functional brain markers of depression and anxiety among individuals with epilepsy and to relate failed or successful treatment of depression, with anti-depressive medications, to structural brain changes. In addition, he is using a novel technique, brain network activation (BNA), to uncover alterations in brain networks that are the result of repetitive seizure activity. Rush University Medical Center is the leader in Chicago for resection surgeries to treat drug resistant epilepsy and is uniquely positioned to apply microscopic and physiological techniques to freshly resected tissue. In collaboration with Dr. Nicholson's state-of-the-art cellular imaging laboratory, he is studying the molecular basis of neuronal hyperexcitability in humans with temporal lobe epilepsy using resected human tissue. The goal is to examine the functional and structural anomalies in these patients and to identify target areas that can be approached with interventional strategies such as drug delivery or alterations in the surgical approach. This may ultimately result in seizure freedom or a reduction in seizure frequency or severity, improving the quality of life for the patients and their families.</p>					