

Utilization of Social Media Tools for Student-Driven Medical Education

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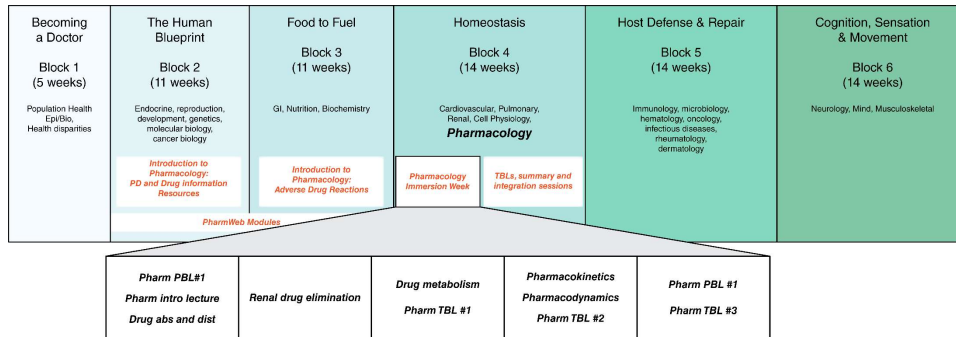
Western Reserve₂ (WR2) Curriculum

Foundations of Medicine and Health	Advanced Clinical Experiences and Residency Preparation	
Basic Sciences and Early Clinical Training	Core Clerkships	Acting Internships
	Basic Science Integration	Transition to Residency
	Electives and Board Study	Advanced Electives
	USMLE Step 1	USMLE Step 2 (CK/CS)
Research and Scholarship → MD Thesis		
1 st Year	2 nd Year	3 rd Year
		4 th Year

Pre-clerkship curriculum at Case Western

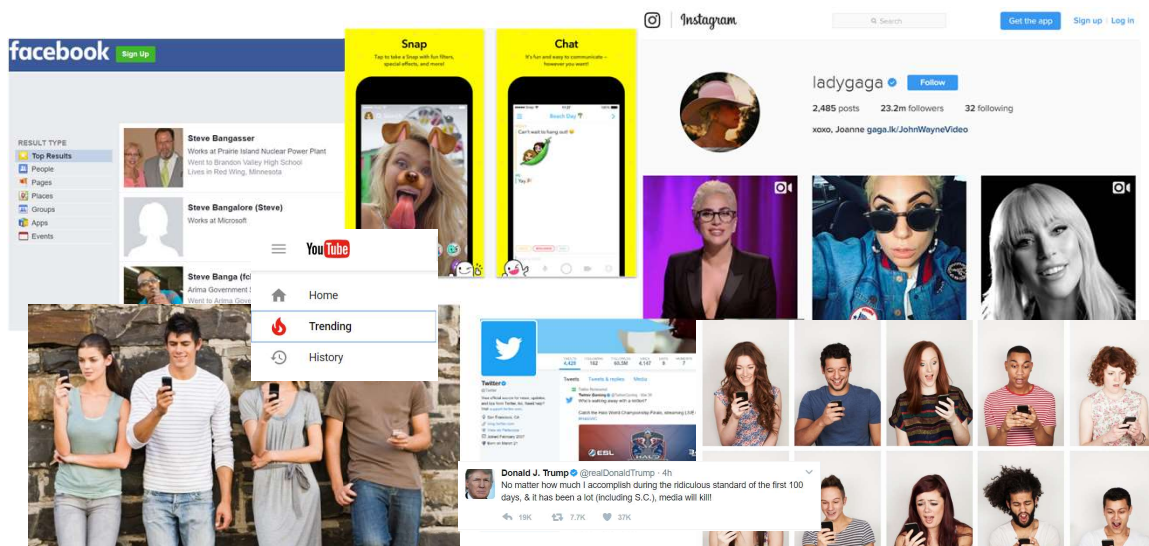
• Hybrid curriculum

- Half lectures/TBLs
- Half Problem-based learning
- Six sequential Blocks
- Main pharmacology contact time during Block 4



*Tight schedule limits student contact time with pharmacology faculty
Yet, students consistently request more pharmacology exposure*

How to connect with today's students?



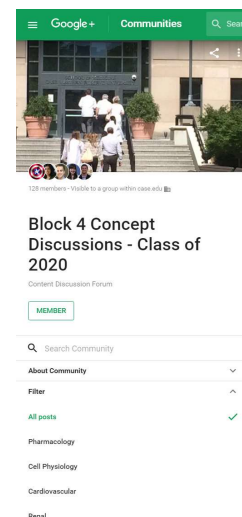
Potential benefits of using social media-based discussion boards for pharmacology education

- Provides a centralized forum for discussion of topics of specific relevance to the curriculum
- Allows students to ask questions after they have had time to digest, reflect on and assimilate information provided in lectures, PBLs or TBLs
- Allows students to efficiently share useful resources
- Allows students to answer each others questions
- All students can follow and contribute to the discussions on their own time
- Forces students (and faculty) to clarify their questions and answers in writing

Cheston CC, Flickinger TE, Chisolm MS. Social media use in medical education: a systematic review. Acad Med. 2013 Jun;88(6):893-901.
Davis WM, Ho K, Last J. Advancing social media in medical education. CMAJ. 2015 May 19;187(8):549-50.

Application of Google+ discussion forums for asynchronous pharmacology education

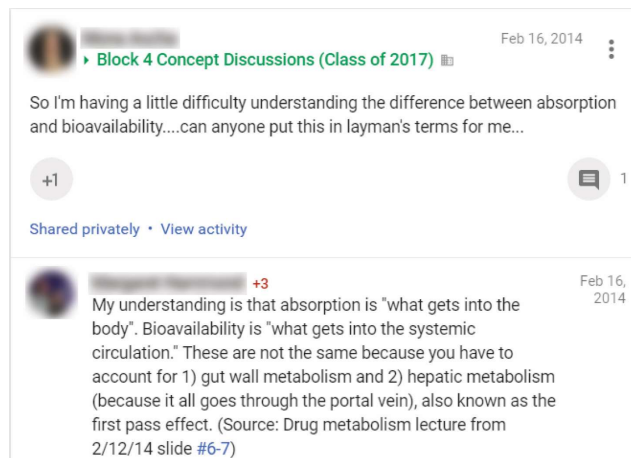
- Professional forum that can be set up for restricted access
- At CWRU, everyone has a Google+ account by default, no need for people to create an account
- Available as an app that runs on smart phones
- Provides helpful functionalities like image and video posting within the discussion box
- Mechanism to reward helpful posts (+1), similar to Facebook “like”, Twitter “favorite” or Instagram “love”
- Can “follow” people like on Twitter
- Community discussions are saved and easy to recall



Experience with Google+

- **Set up**
 - Straightforward except that inviting many people to join a community is cumbersome
- **Participation (initial pharmacology posts)**
 - 13 thus far - 2017
 - 5 – 2016
 - 9 – 2015
 - 11 – 2014
 - In most cases there are multiple follow-up posts
 - Multiple students contribute to the discussion
- **Post topics**
 - Tips on drug information research
 - Clarification of lecture material and assigned reading
 - TBL/PBL ideal answer clarification

Example discussion #1



Example discussion #2

Block 4 Concept Discussions - Class of 2020

Feb 12, 2017

I wanted to ask a question about this slide relating therapeutic index, to population therapeutic window, to the therapeutic window for an individual (these seemed like the 3 categories presented in lecture, but I could be wrong). If one goes up, do the others all necessarily follow? For instance, can you have a drug with a wide therapeutic index, but a very small therapeutic window (both for populations and an individual), and vice versa? If this can happen, it would seem like the therapeutic index alone could falsely lead one to believe that one drug was safer than the other drug...

Dose-related ADRs

Quantal population dose-response curves

Therapeutic response Adverse response Lethality

therapeutic index = $\frac{TD_{01}}{ED_{50}}$

margin of safety = $\frac{LD_{01}}{ED_{50}}$

Population therapeutic window: high probability of efficacy with low probability of toxicity

+1

Shared privately · View activity

- +1
- I see what you are saying, sort of like if the curve of one of the slopes was flattened (for example the adverse response curve above), it would more easily overlap, yet the TD50 and ED50 would remain the same. So I think your line of thinking is correct
- 7w
- Yea, exactly! But I wonder if that's true in general...
- 7w
- Philip Kiser
- Great observation. A shallower slope would mean more variability in the population making you less confident that a given dose will be effective in your patient without inducing toxicity. So it is true that you could have two drugs with the same TI but different actual safety levels. The margin of safety index is better in this regard.
- 7w

Example discussion #3

Block 4 Concept Discussions - Class of 2019

Feb 13, 2016

Can you have a pharmacokinetic drug-drug interaction where the first drug shuts down a reabsorption transporter in the kidney and it lowers the [serum] of a second drug?

Second question is if you can have a pharmacodynamic drug-drug interaction where they deactivate the same pathway?

+1

Shared privately · View activity

- +1
- Re: your first question, I was looking over a paper about renal DDIs and the authors pitch your idea as plausible, but later on they say it hasn't been observed clinically: "To our knowledge, no clinical drug interactions have been reported between 2 xeno-biotics competing at the active tubular reabsorption level."
- <http://www.ncbi.nlm.nih.gov/pubmed/9592621>
- Feb 13, 2016
- Philip Kiser
- For 1) also keep in mind that drug reabsorption from the glomerular filtrate is almost always a passive process, not mediated by transporters
- Feb 15, 2016
- For 2) what type of pathway do you have in mind?
- Feb 28, 2016
- For #2, would ACE inhibitors and losartan be an example since they both block the effects of angiotensin II, but using different mechanisms?

Example discussion #4



Block 4 Concept Discussions - Class of 2020

Feb 13, 2017



+2

Hi class,

I have a question regarding calculation of maintenance dose and loading dose.

On Wikipedia, the formula for both is a little different from what we have from class. For loading dose it has the formula $CpVd/FS$. For maintenance dose it has the formula $CpCL/F$. (Cp = desired concentration of drug; Vd = volume of distribution; F = bioavailability; CL =clearance.)

Could anyone explain the differences between the equations and whether there are times we would use one over the other. Especially for maintenance dose, in the textbook bioavailability is in the numerator, whereas in wikipedia it is in the denominator, so I find it confusing to understand.

As for the formulas you mentioned on Wiki:

loading dose = $CpVd/F$

^This is the formula in the powerpoint, but just solved for loading dose instead of for Cp (which is $C0$ in the powerpoint).

And as for the Wiki formula which has:

maintenance dose = $CpCL/F$

In the powerpoint, if we solve for D/t (which is the maintenance dose), we get $D/t = Cavg CL / F$, where $Cavg$ I think is the same as Cp .

Example discussion #4, cont.



+2
Hey -

Golan does give: maintenance dose = $Css \times CL$
Our slides give: $D/t = Cavg \times CL / F$

Maintenance dose is the same as D/t ; Css is the same as $Cavg$

So the only difference is that in our slides they divide by F , and in Golan they don't include F in the formula. But on p. 47 of the 2nd edition, at least, Golan does mention that after you calculate maintenance dose, you have to account for bioavailability, which you do by simply dividing your answer by F (for example, if the maintenance dose is 6 mg/day, but the F is only 0.50, then you should give 12 mg/day - which you calculated by just dividing 6 by 0.50). This then gives us the same equations.

Golan does mention "maintenance dose" and "dosing interval" as separate, which is confusing, but I think the term "maintenance dose" can refer to either: a) D/t , that is, maintenance drug dose per time or b) simply the numerator of D/t , in which case it's literally just the "dose" without the dosing interval. The interval matters because you can avoid toxicity by giving more frequent, smaller doses to achieve the same "maintenance dose" as if you had given larger doses at longer intervals. Back to our example, we are giving 12 mg/day (since only half is making it to systemic circulation); but if this is toxic, we can give 4 mg three times a day. This would be like the zig-zags getting smaller and closer on that graph. Ideally, we give just a very small amount but at every second, which would give us just the smooth curve and no more zig-zag (this would be like a continuous IV infusion).



Thanks for clarifying Faez! I still have a question regarding the maintenance dose. In Golan p41 it gives the equation for maintenance dose as $D \text{ maintenance} = \text{Clearance} \times C \text{ steady state}$. Which actually gives us $D \text{ maintenance} = \text{Bioavailability} \times \text{dose/interval}$. Also, this would make D/t and $D \text{ maintenance}$ different things, which is why I am still kind of confused.

Google+ wishlist

- Anonymous posting (that can be controlled by moderators)
- Easier, more reliable, way to add members *en masse*
- Tracking number of views of a post

Example discussion #5

Block 4 Concept Discussions - Class of 2020 Feb 18, 2017

The ideal answer in a recent SEQ describes the mechanism of Digoxin as the following: "The consequence of decreased Na⁺ extrusion is an increased influx of Ca²⁺ which provides for an increased force of contraction."

Both Lilly (388, 5th ed.) and Costanzo (144), however, claim that the increased Ca²⁺ leading to increased force of contraction is not a result of increased flux of Ca²⁺ but rather a build-up of Ca²⁺ concentration from the decreased flux of the Na⁺-Ca⁺ exchanger.

How should we reconcile these differences in explaining the MOA of Digoxin?

Thanks,

+1 1

Shared privately · View activity

8w

The second answer is the correct one, I'd think the professor might have just misspoken when typing the ideal answer

8w

Agreed, I think you can argue the ideal answer is correct as well - there's two stages/pumps going on.

1. Increased intracellular (cytoplasmic) Ca²⁺ levels from decreased flux of Na⁺/Ca²⁺ exchanger -> 2. increased influx of Ca²⁺ into sarcoplasmic reticulum (through action of SERCA Ca²⁺/ATPase pump). Ultimately, it's the increase in Ca²⁺ levels in the SR not the cytosol responsible for increased myofibril contraction/observed clinical result. Long story short, calcium isn't pumped out from cytosol, allowing for its influx into the sarcoplasmic reticulum.



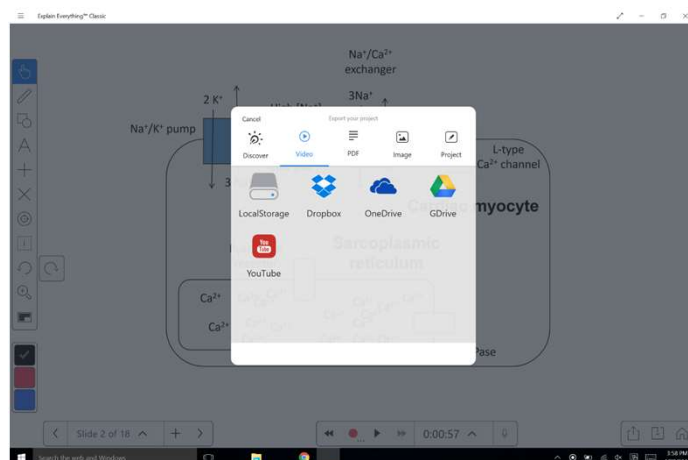
Customized videos to address student questions



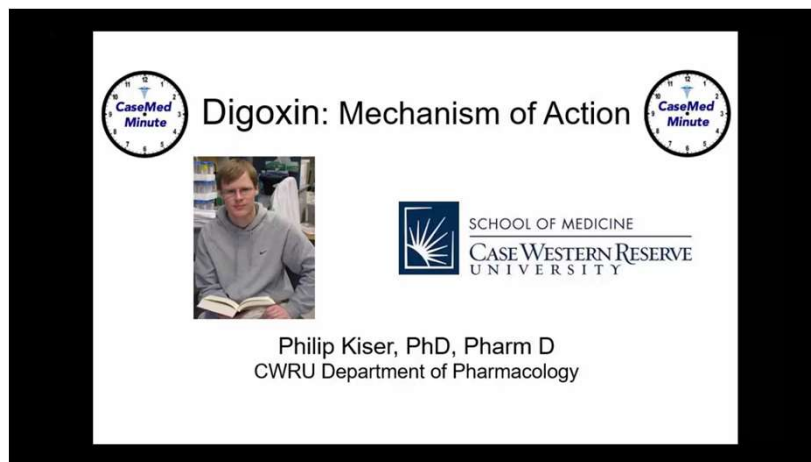
Available for Windows/Mac/Android

Free form or Powerpoint/pdf-based presentation options available

Separate recording for each slide (easy to edit)



Example video



Conclusions, implementation tips and future directions

- Google+ communities are useful for asynchronous pharmacology education
- Faculty promotion and monitoring of the forum is critical for student participation in my experience
- Faculty videos are often helpful supplements to discussions (don't underestimate the amount prep time needed for them!)
- Future efforts will aim to quantify the efficacy of the discussion forum in improving understanding of core pharmacological concepts