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ACRONYMS

STUFF THAT'S NOT SCIENCE

FLOSS free/libre open source software

GPL GNU General Public License

GNU GNUs Not Unix

PLOS Public Library of Science

OS open science

SCIENCE

GPCRS G-protein coupled receptors

5-HT 5-hydroxytryptamine

CNS central nervous system

BHLH basic helix-loop-helix protein structural motif

VMNS ventral motor neurons

SHH sonic hedgehog

ASCL1 Achaete-scute homolog 1

1

"SCIENCE STUDIES, FEMINIST THEORY, CULTURAL STUDIES"

1.1 SCIENCE AS PRODUCED KNOWLEDGE: KUHN, FLECK, AND FOUCAULT:

re: Kuhn, Fleck, Foucault

Science is fake; or rather, it's made in a lab by people – it's the genesis of facts, not discovery

1.2 FEMINIST SCIENCE STUDIES:

Produced knowledge as a point to analyze: feminist science studies, re: Keller, Haraway, Harding, Fausto-Sterling, Roberts

standpoint theory & strong objectivity!

women in science: eh, boring

gender in scientific knowledge: much more interesting and to me, important to figure out in mol neuro.

1.3 BEYOND GENDER ANALYSES

not so much gender, but how science appears globally, currently. specifically sparked by the pen access "save the third world" rhetoric – obviously flawed, as per colonial/white feminism, e.g. movements to ban the burqa or "rescue" women in former European colonies

bored of asking where are women? (not in science) more interested in intersectional questions about queer, colored, trans, feminism/femme/women, poor etc;

2 REPRESENTATION & SCIENCE

ACRONYMS

OEAC Other Euro-American Countries, including Australia, NZ, and Canada

ROW "Rest of World", anything not already included

From a "purely" numbers standpoint, scientific production is unequally distributed around the world and on a variety of axes.

2.1 DEVELOPING COUNTRIES

Sumathipala et al. [17]

In a retrospective of 5 major medical journals, the authors examined contributions by 4 geographic areas: the UK, USA, Other Euro-American Countries, including Australia, NZ, and Canada (OEAC), and "Rest of World", anything not already included (RoW). RoW, on average, contributed 6.5% of the research literature. The highest seen was in the Lancet (12%), and of the 151 RoW articles involved, 68.9% involved authorship from a developed county in Europe or North America. 15 original papers in the journals had data from RoW w/ no RoW coauthors.

The 10/90 problem is a clever name for enormous inequity: only 10% of the world's resources are used for 90% of health problems; in the author's previous survey of psychiatric journals, only 6% arose from regions accounting for over 90% of the population.

Science has a WEIRD problem...but like, actually. this not even accounting for gender or age

This study selected the highest impact general purpose medical journals, of all issues in one calendar year (either 2000 or 1999), and examined authorship (by institutional affiliation) and the methods, to ascertain the geographic origin of data.

Some notable variations in the regional data: 2 countries (Japan and Israel) contribute a 5th of the RoW-published literature, while China and India combined (the two most populous countries in the world) contribute a total of 13%. Only 31% of the total articles were entirely independent RoW.

2.2 GENDER DISPARITIES

bal gender disparities in science

> "Although there are more female than male undergraduate [especially in "softer" scientific fields" and graduate students in many countries, there are relatively few female full professors, and gender inequalities in hiring, earningsg, funding, satisfaction and patenting persist."

Quantitition of gender disparities in measures of scholarly output and has been few and far between – "highly localized, mono-disciplinary, and dated"; they take little account of the rise in collaborative research etc. This paper analyzed the relationship between gender and research

output (authorship), collaboration extent (co-authorships) and scientific impact of publications (citation count, according to the Web of Science); analyzed >5 million research papers and reviews with more than 27 million involved authors, assigning gender using data from Social Security database.

Web of science, of course, is an extremely flawed way to do this because it barely includes anything that isn't the US or UK. See the sections on citation factors and global publishing

Some results:

1. in the most productive cntries, all articles with women in dominant author positions receive fewer citations than those with men in the same position
    a) accentuated by domesticity [nationality-wise] - women benefit less from the extra citations accrued by international collaborations
2. globally, women account for <30% of fractionalized authorship, men are >70%
    a) 1:1.93 women:men first authorships
3. South America & Eastern Europe had best gender parity
    a) female authorship generally more prevalent in countries and/or states with lower output
4. impact: when a woman is in prominent position (sole authorship, first-author, last-author), paper attracts fewer citations; holds for national and international collaborations

There are, of course, major limitations, the biggest one being obvious – age has an enormous role to play. Many observed trends are likely proxies of a different problem: that women aren't making it to the top ranks of sicence, and that those senior ranks bear the imprint of previous barriers.

Basically, this is quantitive numbers to back up what women ===have been saying for years – there are barriers, and they are tall. In 17% of countries, scientists are equally represented: but fewer than 6% Web of Science countries come even close to gender parity in # papers published.

West et al. published an extremely similar paper in PLoS one the same year, without the global analysis component but with even more papers as part of the analysis. Their source was the JSTOR corpus, "a body of academic papers...spanning five centuries. They look at similar variables – authorship order and overall percentages. This includes the humanities research as well, which is interested.

with the mention that publication counts obviously are relevant in tenure *discussions, e.g. hiring e.g. perpetuating cycle*

Also, HUGE points for their methods and reproducibility, e.g. creating the gender browser and communicating with me about making that something to apply to own libraries.

2.3 RESEARCH ACCESS & ASSESSMENT

2.3.1 *Global Citation Indices*

Test textWagner - Actually apparently this isn't a real problem. As in, only 4% of journals in *any* country are indexed. Which really just means like, why are we bothering with this?

2.3.2 *effect of open access publishing* on *access*

What is the influence of publishing OA on research attention? It's greatest impact is the development of world participation.

> Across sub-fields, the impact of commercial online availability was positive, statistically significant, and on average 40% larger than the OA effect, suggesting that most researchers rely on institutional subscriptions.
>
> The influence of OA was more than twice as strong in the develop- ing world but was less apparent in the very poorest countries where electronic access is limited (Fig. 1D).[8]

Good/bad/always more complicated

3 OPEN SCIENCE

3.1 WHY DO I CARE ABOUT OPEN SCIENCE?

I think the movement, generally, is trying to do the right thing. and connects people and researchs

I think technology *can* be amazingly helpful; I want everybody to have the tools they need/want.

I recognize that science is largely inaccessible, and that technology – "open" technology – can make it more accessible.

I think this maybe needs the context of neuroscience. Possibly. At the end though.

3.1.1 *Defining "open"*

"Open science" is a big, vague, nebulous term. First, the short and sweet version:

> "Open means **anyone** can **freely access, use, modify, and share** [content] for **any purpose**."

The "free" in that definition is, of course, free as in freedom, not as in beer. The open science movement is closely affiliated with the free/libre open source software (FLOSS) movement, and as such, there's a large overlap in terminology. Some preemptive clarification:

> "The terminology of and factions within this movement are complex, but, in short, "free software" tends to be associated with the ideology of freedom, "open source" with the openness of the development process, and "libre" with those concerned about confusion from the previous two. FOSS/FLOSS are used as monikers to refer to all of these meanings." (Reagle, 2012)

The open science movement's unifying[1] principle manifests with 3 (or 4, or 6, depending on who you ask) areas of interest:

1. open access - namely, to full-text published papers and research results
2. open data - publishing raw data pre- or concurrently with paper publication
3. open research - everything else, including, but not limited to:
    a) open code distribution - code for analyses, model generation, etc. should be hosted somewhere accessible
    b) open lab notebooks - tracing the entire research process with all dead ends and kinks included

1 unifying isn't actually the right word here – single shared point? overlap? lots of smaller movements all agreeing on this one

There's also a hundred other subcategories that fall under the umbrella; new systems of distributed, ongoing, or otherwise "open" peer review or community discussion of publications, like the work at PubPeer, billed as an "online journal club". So open science is frustratingly broad and inconsistent, just like any other community. But there are some commonalities in intent and goals, and from that some general open science values can be extrapolated.

3.2 VALUE(S)

I think open science is really interesting as a BIG IDEA, largely because of its ties to the free and open source software movement, and specifically in the realm of community-driven goals. A lot more analytical work has been done on FLOSS sociological values than the narrower and newer niche of OS.

In the FLOSS movement, there's a sharp ideological divide between the free/libre and the open licenses. Advocates of the libre licensing model list many advantages, but their main focus is libre software as a social imperative. User freedom (or their conception of it) is paramount. Open source, on the other hand, was created and remains in an explicit attempt to side-step the social values and ideological connotations of the term "free software", as laid out in the GNU General Public License (GPL) . It instead has a narrow focus on access and production of source code - the 'practical' benefits of distributed production.

Bearing that distinction in mind, trivial as it initially seems, I think the usage of *open* in open science (OS) is a valuable point of entry into the ambivalent moral aspects of open science. Lots of proponents of OS either come from and/or explicitly draw from the F/LOSS movement, but the chosen movement nomenclature is "open". The same disavowal (or maybe just lack of acknowledgment) of social values seen in open source is apparent in a lot of the open science movement. It's not about science helping (or empowering) people, it's about doing "better" science – more reproducible, more reliable, more powerful (disruptive, even!). Open science is defined almost entirely by the *mechanisms*, in the same way open source is *just* focused on production. Even projects that pitch themselves as a place for citizen scientists can be construed as just a way to shift labor from cheap graduate students to even cheaper laypeople.

3.2.1 *"Biohackers"*

Delfanti, in 2013, published "Biohackers: The Politics of Open Science". It lies at the "the intersection of digital cultures, science communication and science and technology studies," a grouping that bears no little similarity to my own interests. Delfanti's book is a delight, and covers in brief detail a number of relevant topics.

Lineages of open (science)

> "The most common narrative about open science tells us that, once upon a time, science was an ethical enterprise: sharing, equality, disinterest and the common good drove the everyday work of scientists. Then evil corporations entered science and changed the rules of the game, patenting

> life, enclosing the commons, and eventually destroying the willingness to share data, information, and knowledge. But today, so the story goes, we have new tools that together with the old open science spirit an be used to rebel against evil, defeat it and allow scientific knowledge to flow freely again. These tools are open source and open access science, and they can be used to tear down the barriers to the access of scientific knowledge." (Delfanti, p 5)

Open science, in almost all narratives I've seen, does exactly this. Drawing on a mystical past where information flowed free ("In the 17th century, journal publication *was* open science!), and in so doing, constructing a seamless narrative where open science is part of every major discovery since Newton. Or whoever, pick your scientist. The construction of a history lends legitimacy to the moral appeal of the movement.

Public image management

Delfanti profiles 3 projects in his book:

1. The J. Craig Venter Institute and J. Craig Venter himself, representing venture capitalists and open science for monetary and prestige
2. Ilaria Capua, a vetinarian virologist in Italy, who during the 2006 global avian influenza crisis, pushed the WHO through public shaming into changing their data policies to an open access model to better combat the disease
3. "DIYBio", a loose network of homegrown biologists.

Both Venter and Capua used publications in both prestigious journals and in more public forums (*Scientific American*, *The New York Times*) as a way to get the public involved in their work and on their side; public pressure, as conveyed by the media, was influential in both their success stories.

citations here for when I find them

While these are particularly high profile cases, science has an intimate relationship with the media, and both parties use each other as a tool for various goals. The media gets a reputable scientist figure to bolster their claims; the scientist gets a public venue to couch their ideas or goals in objective-but-positive ways.

Corporate uses of open science

Open science, by definition, can be used by anybody for anything. That also means corporate entities can borrow methods, tools, and data released into the public sphere by government-funded organizations

and other organizations, but...beginning analysis

Delfanti does not, however, draw on explicit feminist analysis of race and gender in the spheres he discusses. The intricacies of who can or will practice (open) science on a social access are almost entirely absent.

3.2.2 *Feminist Open Science(s)*

Who gets to create?

Generally, white men. In the free software world, men outnumber women to a much greater degree than in more traditional development structures.[13] Minorities fare even more poorly.

The dismal representation in FLOSS derives in part from a flawed perception that social stratifications and classifications "don't matter" on the internet. Code is race- and gender-agnostic (except that, for somewhat obvious reasons, it's *not*). Thus, collaborative, decentralized community structures like those in FLOSS usually fail to include any ways of dealing with societal differences. As a result, misogyny and racism run rampant and often unchecked.

Whether representation is *worse* in open science than science at large is something I don't know yet, but it's probably fair to say that open science carries professional risks, and women and minorities in the sciences tend to disproportionately suffer for taking risks.

Who is it designed for?

I don't know yet! But probably I won't like it.

3.2.3 *Global Definitions of "open" and "free"*

Free software and open science are built on European and U.S. legal, moral, and social codes. Probably and definitely an issue, especially when "open science" is supposed to level the playing field but other communities, sciences, etc. are actually consulted in how they would like that leveling to be done.

Relevant citations: Christian[5], Dahdouh-Guebas et al.[6], Gorelick[9], Jolliffe[10]

3.3 GLOBALIZED AND/OR INDIGENOUS SCIENCES

If indigenous sciences are local sciences, specific to the time and place and users involved, then they may not be generalizable to a mythic global community of "scientists".[2] And *if* we create a system where one of those many sciences is targeted as the right one to be spreading, and making access to it the ultimate success or bridge to success, local knowledges are crowded out and erased. Indigenous sciences lose by virtue of what they are – non globalized, local knowledges – with no excuse to *not* use globalized standardized bits of (scientific) knowledge.

2 Destroy the idea of a global community of scientists. If science is local, there is no homogenous community.

4

SEROTONIN, TRANSCRIPTION FACTORS, AND DIFFERENTIATION

"every biological organism is inherently individual"

4.1 WHY SEROTONIN?

[1]Serotonin is 5-hydroxytryptamine (5-HT), a monoamine neurotransmitterbinding 14 classes of receptors, all but one of which are G-protein coupled receptors (GPCRs). With a wide array of receptors distributed across the brain, nearly every basic central nervous system (CNS) function, including mood, cognition, sleep, pain, motor function and endocrine secretion, is in some part modulated by serotonin signalling. As such, disruptions in serotonergic transmission is implicated in many psychological diseases (eg schizophrenia etc), as well as some of the mechanisms behind the relevant therapeutic drugs.[12]

Model systems for serotonergic signalling are fundamental to drug design and development for depression and whatever the other stuff is that serotonin does, and in the case of hallucinogenic drugs, present a potential method to link molecular and sub-cellular processes to cognition and thought.

obviously hallucinogens are what I want to write abut but it's very far away from what I'm doing in lab, so not clear on how they'll get in yet

also, hella reductionism. I don't know if this is BAD though

4.2 MODEL SYSTEMS

If I want to study second messenger signalling pathways, transfecting cells with the cloned receptor doesn't make sense, because the messenger pathways wouldn't necessarily be consistent with fully differentiated neurons – or at least that's my current theory and original motivation.

Current model system options for serotonin systems/work with 5-HT2a receptors and flaws? aka why mine is better for any reason besides $$

other advantages

- exclusion of external elements
- isolated cell type
- controlled enviro

and my own experience with CHO cells and flaws in studying receptor internalization

*Bea thought these were important to discuss, but I'm not convinced I *need* to explain why model systems are useful to normal science. Normal in the Kuhn sense, obviously*

1 **Acronyms:**

GPCRs G-protein coupled receptors

RA retinoic acid

5-HT 5-hydroxytryptamine

CNS central nervous system

BHLH basic helix-loop-helix protein structural motif

VMNs visceral motor neurons

SHH sonic hedgehog

ASCL1 Achaete-scute homolog 1

- genetic manip
- specific cell. manipulations
- support cells (microglia, astrocytes, etc)

Ideally, of course, one uses primary neuron cultures to study signalling – but those are expensive and hard to maintain.

Thus, differentation of fast-growing and easy to maintain cells, that nonetheless express a relatively fully functional GPCR signalling intracellular network.

The available cell line to me is P19 stem cells, which are known to differentiate into poorly-characterized neuron-like cells with the use of retinoic acid. The trick is to direct expression towards serotonergic neurons instead of *just* neurons. So how can we manipulate the P19 stem cells earlier in the process? We can (hopefully) set cell fate early on via expression of important transcription factors in serotonergic differentiation, and then allow cells to develop serotonin-receptor GPCR machinery relatively normally.

as far as cancerous, genetically manipulated, and traumatized rat cells can be "normal"

4.3 P19S

P19s are derived from rat embryonal teratocarcinoma cells. They retain pluripotency under normal cell culture conditions, and upon application of retinoic acid (RA) differentiate into CNS cells, including glia, neurns, and fibroblast like cells.

> "P19s have valuable charactersits – they are immortal, allowing the creation of "almost unlimited amounts of material for analysis, and easy to grow and maintain as undifferentiated cells, but can also be efficiently induced to differentiate by manipulation of culture conditions. "[2]
>
> The P19 embryonic carcinoma cell line has proved to be a particularly tractable system for studying neuron and glia differentiation. P19 cells are pluripotent and can be induced to differentiate into derivatives of all three germ layers: endoderm, mesoderm and ectoderm, depending on the nature of chemical inducers and the culture conditions (Runnicki and McBruney, 1987). After treatment with high concentrations of retinoic acid (RA) and aggregation, P19 cells can differentiate into neurons, glia and fibroblast-like cells (Jones-Villeneuve et al., 1982).
>
> RA has also been implicated in the development of vertebrate nervous system in vivo. RA is involved in the stimulation of axon outgrowth, the migration of neural crest cells and the specification of rostrocaudal position in the developing central nervous system (Maden and Holder, 1992). Studies of RA-induced P19 neural differ- entiation have led to the discovery of a number of genes that are important for neural development in vivo[19]

4.4 THE 5-HT SYSTEM

The 5-HT system is one of the most complex projection networks in the CNS. It has three descending efferents to the spinal cord, while the

Genes	Expression	5–HT defects	KO lethality	5–HT-specific TF
Nkx2.2	ventricular zone	100% except in r1 P0	P0	lost except in r1
Mash1	ventricular zone	Almost 100%	P0	all lost
Gata2	VZ, postmitotic	100%	E10-12.5	all lost
Lmx1b	Postmitotic	100%	P0	all lost
Pet1	Postmitotic	70%	mostly viable	unknown
Gata3	Postmitotic	mostly in caudal	E11.5-13.5	unknown

Table 1: Summary of the phenotype of knockout mice lacking individual transcription factor in 5–HT neurons. (from Chen and Ding)

B5-B9 neurons project via 5 different routes to almost every region of the CNS. This large network means serotonin signalling not only transmits information, but plays a large role in *modulating* the rest of brain function.

Brain anatomy.

5-HT *actual* distribution

4.4.1 *embryonic development*

Stem cells = embryonic cells; hence, knowing and examining the embryonic precursors to 5-HT neurons and then mimicking them => 5-HT neurons in culture

During embryonic development, the different networks of 5-HT neurons are much less distinct. In mice, they're some of the earliest generated neurons, appearing between E10.5 and E12.5, starting in the rostral regions along the brainstem axis. By E12.5, almost all of the 5-HT neuron classes are present. Induction and specification of diverse neurons, and specifically 5-HT neurons, requires a set of signaling molecules, converted into a combinatorial transcriptional code.

4.5 TRANSCRIPTION FACTORS IN 5-HT DIFFERENTIATION

4.5.1 *in 5-HT progenitor cells*

Nkx2.2

Nkx2.2! (**Nkx2.2!**) is a homeodomain-containing transcription fator that specifies the ventral cell type in response tosonic hedgehog (Shh) signals in the spinal cord and hindbrain. Expression of *Nkx2.2* ceases as soon as 5-HT cells become post-mitotic. Upstream of *Phox2b*, *Nkx2.2* plays a role in

1. earliest transcription factor required for 5-HT specifications
2. dispensable in r1 5-HT; Nkx2.2 KO neurons derived from r1 are unaffected
3. in the absence of Phox2b, adopts a default pathway to promote 5-HT fate

*Ascl1/Mash1 **

expression patterns & time course

Mash1 is the mouse, human, and rat basic helix-loop-helix protein structural motif (bHLH) homologue of Achaete-scute homolog 1 (Ascl1), first discovered and explored in *Drosophila* as a pro-neural gene.

It's a key fate determinant for many neuron types during fetal and adult neurogenesis. In the embryonic hindbrain, it's the only known pro-neural gene expressed in the domain of 5-HT progenitor cells, and is co-expressed with **Nkx2.2!** during vMN and 5-HT production.

Mash1 KO mice do not effectively product 5-HT neurons. visceral motor neurons (vMNs), which derive from the same set of cells, are produced normally, but without Mash1, none of the normal post-mitotic 5-HT transcription factors express. Likely, this is due to the loss of Notch signalling that ordinarily leads to 5-HT neurogenesis.

Mash1 possesses specific 5-HT characteristics; cannot be replaced by other pro-neural bHLH factors. At the same time, it is not *sufficient* to induce 5-HT differentiation; it requires cofactors, like *Nkx2.2*.

4.5.2 *in the ventricular zone & post-mitotic 5-HT neurons*

Gata2, Gata3

1. Members of the GATA family containing zinc-fingers which bind to core (A/T)GATA(A/G)
2. Gata2 precedes -3;
    a) E9.0 in r4 and transiently in r2
    b) E10.5 expanded to all rhombomeres and detected in VZ progenitor cells and in post-mitotic cells
3. Gata2 KO:
    a) severe anemia; death between E10 and E11
    b) defects in neurogenesis generally; early lethality precludes 5-HT specific examination
    c) *in vitro* test; KO tissues lack all 5-HT neurons, even in presence of Gata3
4. Gata2 affects different 5-HT cluster development differently. It is necessary and sufficient in R1 neurons, and capable of inducing other 5-HT specific TFs in r1.
    a) capable of inducing other 5-HT specific TFs in r1
    b) necessary and sufficient in r1
    c) necessary but not sufficient
5. Gata3 - relevant in caudal development of raphe nuclei
6. conflicting reports of Gata2 + Gata3 interactions
    a) Gata3 operates either independently or downstream of Gata2 and Lmx1b
    b) poorly understand epistatic relationships

5 CRITICAL NEUROSCIENCE

5.1 HISTORICAL NEUROSCIENCE?

"Neuroscience" as a discipline constructs a history tied to Galen and Aristotle and ancient Sumerian hieroglyphs, but in it's current state is a product of a reductionist "molecular gaze".[1]

5.2 SOME QUESTIONS

- Do stem cells have sex?
  - YES. They do. See[15]
  - also, see http://genderedinnovations.stanford.edu/case-studies/stem_cells.html
- where does molecular reductionism come from? [genomics]
- where does molecular red. lead us?
- can/should we rely on cell studies to give us answers about cognition?
  - or therapies, generally
  - what do we lose when we make diseases "solvable" in vitro; what nuances?
    - "personalized medicine" – still reductionist to genomic/-cellular information
  - model systems as a philosoph

5.3 SOME THOUGHTS AND STUFF

Neuroscience is essentially, and intentionally, interdisciplinary. Choudhury and Slaby, in the introduction to Critical Neuro. say propose "interdisciplinary" as a code word for scientism. Neuroscience is a scientific-only approach to a centuries old question of consciousness/humanity/what it is – or at least that's what we want to think about neuroscience. Potentially, and largely, missing out on a holistic embedded version of humanity. I think relevant is Gorelick's brief discussion of indigenous sciences as a process, and specifically the proposal that science-as-a-process is consistent with a verb-based language. "A bay" is English; the verb-based version is "to be a bay", which is not inanimate but moves in dynamic relationships with the wind and shoreline and other parts of the world. To be human or conscious or think is to have a similar dynamic and evolving relationship. On a very basic level, to be alive is to be in concert with the environment. #epigenetics.

So cellular and molecular neuroscience is missing a great deal of things.

Situating the brain/cognition w/in a culture is a trend - epigenetics as "from soma to society". But this also creates new "at rick" populations, incorporating social culture into scientific dogma. –[11,14]

A sensible research strategy (isolationism/reducitonism) becomes a metaphysical *commitment* to a system, where the system *is* reality. For us, cell lines are indicative of some reality. It's plausible that scientists themselves know their work to be misisng enormous sweeps of holistic knowledge, but any written work rarely acknowledges it. Grants and papers imply and argue that molecular work is *the* answer to XYZ – and not just the answer, but the problem. See the "gene for asthma" for why this is obviously wildly incorrect.

Big narratives of simplicity – simple simple simple, so we can understand. Many simple pieces to make a big complex system. Hoes this work? Eh, probably not. But what does it do when it doesn't work? How does it go wrong? Or how does it go right?

There's some arguments about neoliberalism and reductionism. I hear a lot of usage of "nature" and "science" to support some sort of status quo, of "free market forces"

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