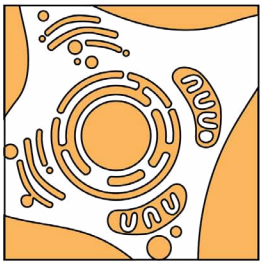
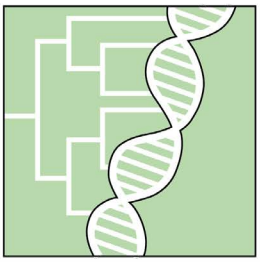
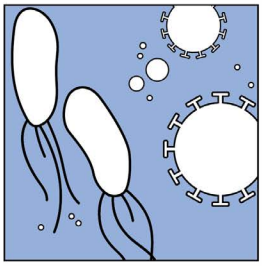




CAMB Student Newsletter

Volume 6 | Issue 3 | August 2021

In this issue

Letter from the Editors

Dear CAMB Students, Faculty, and Alumni,

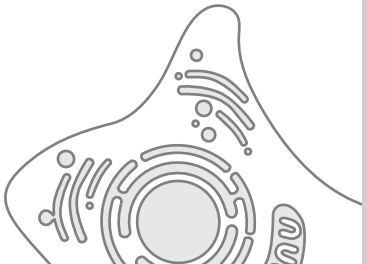
As we wrap up summer 2021 and look forward to the new academic year, we'd like to welcome to Penn the first-year CAMB graduate students! We're excited to meet you, in person or virtually, and to get to know you and your interests!

In the August issue of the CAMB Student Newsletter, we review Derek Liberti's recent publication in Cell Reports. Liberti, a fifth-year DSRB student, discovers how alveolar epithelial cells balance proliferation and differentiation to repair acutely injured lung. We chat with Dr. Marie Guerraty, an Assistant Professor of Medicine whose research focuses on the coronary microvasculature (a note to first-year students – Dr. Guerraty is accepting new rotation students!). We also discuss the disproportionate burden placed on early career women in science, and how the COVID-19 pandemic exacerbated these pre-existing burdens. Finally, CAMB Newsletter Writer Kanika Jain shares her first-person account of traveling in India during the height of the recent COVID-19 surge.

Lastly, check out page 10 for our new column – "Positivity Posts". We thank CAMB student Emily Shea for sharing her story of positivity with us, and congratulations on adopting your new dog, Rory!!

For additional articles, past publications, and to learn more about the CAMB Student Newsletter team, visit our blog at cambnewsletter.wix.com/blog. Current students interested in contributing to the CAMB Student Newsletter can contact us at camb.studentnews@gmail.com. We hope you enjoy the August 2021 issue!

Sincerely,
James Gesualdi and Hannah Kolev
Editors-in-Chief



Research Spotlight

AT2 alveolar cells balance differentiation and proliferation to repair acute lung injury

Aishwarya Pawar

Derek Liberti

Acute lung injuries can be caused by a variety of factors including smoke inhalation and microbial infections like pneumonia, COVID-19, and H1N1 viral infection. These injuries reduce the respiratory capacity of the lungs by destroying resident epithelial cells and damaging alveolar architecture. A recent study published by ***Derek Liberti****, a 5th-year DSRB candidate from Edward Morrisey's lab, elucidates how the alveolar stem cells choose between proliferation and differentiation during alveolar restoration following acute lung injury.*

Gas exchange between the blood and atmospheric air takes place across the thin epithelial surface of pulmonary air sacs called alveoli. Two major epithelial cell types make up the lung alveolar tissue: Alveolar Type 1 (AT1) and Alveolar Type 2 (AT2) cells. AT1 cells form the single-cell thick alveolar surface across which gas exchange takes place. AT2 cells generate and secrete surfactants to reduce surface tension and prevent the alveolar sacs from collapsing. However, outside of this homeostatic state, AT2 cells have another important function. Following lung damage caused by injuries like viral infections, AT2 cells act as pulmonary stem cells, dividing and differentiating into AT1 cells to restore alveolar architecture. The balance between proliferation and differentiation of AT2 cells is precariously maintained, and signaling through the Fibroblast Growth Factor (FGF) has previously been reported to develop alveolar architecture neonatally [1] and maintain AT2 cell homeostasis through its receptor FGFR2 [2]. However, how AT2 cells divide or differentiate into AT1 cells, and the role of FGFR2 in regulating this stem cell fate decision remains poorly understood.

To address this critical knowledge gap, Liberti et al investigated the function of FGFR2 signaling during postnatal lung development and during adult lung regeneration following acute injury. First, the authors determined the role of FGFR2 during alveologenesis, a period of postnatal lung development during which alveoli form. They performed a postnatal lineage trace of control and loss-of-function *Fgfr2* mutant mouse lungs using immunofluorescence and showed that loss of *Fgfr2* function from AT2 cells leads to an increase in their differentiation into AT1 cells. This finding suggested that FGFR2 restricts AT2 cell fate during developmental alveologenesis. However, when *Fgfr2* was inactivated in AT2 cells of the adult lung, lung architecture and morphology were maintained for 1 month, 6 months, and 12 months following the loss of FGFR2 expression. These findings demonstrated that FGFR2 is required to maintain AT2 cell identity during alveol-

ogenesis but is dispensable for adult AT2 lung homeostasis.

Following injury and in regions of acute damage, Liberti and colleagues identified a more active role for FGFR2 signaling. Lung damage caused by acute injuries is highly heterogeneous in nature and elicits varying degrees of regenerative and restorative responses depending on the degree of damage. This heterogeneity makes it difficult to study and visualize acute alveolar injury. Recognizing this caveat, Liberti et al developed a lung damage assessment program, which used computer vision on histological samples to assess the degree of lung damage and categorize injured regions as "*severe*", "*damaged*", or "*normal*". This new and robust analytical approach helped the authors in the assessment of lung regions and their regenerative response as a factor of the degree of injury.

"A major challenge in identifying therapeutic targets to ameliorate lung disease lies in our limited understanding of both the essential factors that control cell fate decisions and the consequences of those decisions for lung regeneration."

Combining their zonal analysis with immunohistochemistry, Liberti et al characterized these zones according to AT1 and AT2 cell behavior and density. They demonstrated that regions of *severe* injury following influenza infection were largely populated by Keratin 5 (KRT5)-expressing epithelial cells, which indicated a quick but temporary response to damage. These *severe* zones also had negligible AT1 cells but a small number of highly proliferative, non-differentiating AT2 cells. In contrast, *damaged* regions largely lacked KRT5+ cells, but contained both a large number of AT1 cells and rapidly dividing and differentiating AT2 cells.

After identifying these distinctive zonal characteristics, the authors next compared *damaged* and *severe* zones of control and *Fgfr2*-deficient AT2 cells following influenza infection. Using immunohistochemistry for Ki67, a marker of proliferative cells, the authors found that FGFR2 loss reduced the proliferation of AT2 cells in *damaged* zones, but not in the *severe* zones. This finding suggested that FGFR2 is especially important for AT2 cell proliferation for the purpose of tissue regeneration in *damaged* zones.

The authors next switched to an *ex vivo* organoid model to further define the function of FGFR2 in regulating AT2 cell proliferation. Control and *Fgfr2*-deficient organoids collected from the uninjured mouse lung grew comparably, however, control organoids showed a robust growth response to exogenously-supplied FGF7, an FGFR2 ligand. Conversely, *Fgfr2*-deficient AT2 cells did not proliferate in response to Fgf7. Given previous studies [3,4] suggesting a role for inflammatory inputs in regulating lung regeneration, the group next tested whether FGFR2 is required for the AT2-cell response to three cytokines: IL-1α, IL-1β, and TNF-α. Interestingly, the authors observed no significant difference in growth between control and *Fgfr2*-deficient organoids in response to these cytokines. These findings suggested that restorative AT2 cell proliferation in *damaged* regions can be activated in response to other stimuli besides FGFR2 signaling, for example, cytokine and inflammatory signaling.

Returning to an *in vivo Fgfr2* control and mutant AT2 cell model, the authors performed a second round of lineage-tracing experiments and determined that loss of *Fgfr2* function not only reduced the proliferation of AT2 cells but also increased the proportion of AT1:AT2 cells, resulting in imperfect alveolar structure of the mutant lungs with the formation of large extended air spaces in *damaged* zones. This result was possible either if FGFR2 indirectly suppressed differentiation of AT2 cells by promoting their proliferation, or if FGFR2 directly

controlled differentiation of AT2 cells. Using an *Ect2* genetic deletion model (where cytokinesis and cell division are blocked) the authors showed that AT2 cells paused in a binucleated state of cell division can still differentiate into AT1 cells in *damaged* zones. This result demonstrated that AT2 cells do not need to divide in order to differentiate into AT1 cells when restoring a *damaged* alveolar zone, suggesting that cell proliferation and differentiation are decoupled in AT2 cells through FGFR2 function.

Alveolar architecture provides more than $100m^2$ of surface area for the exchange of gases between the blood and the atmosphere, and encounters 5-8 liters of atmospheric air per minute. This high degree of exposure renders the alveolar epithelium extremely susceptible to injury via pollutants, microbial pathogens, airborne chemicals, and particulate matter. Any damage to lung architecture calls for urgent repair and restoration to reestablish the gas exchange capacity of the lungs. Liberti's results show how diverse signaling pathways, such as FGFR2 amd cytokine signaling, converge to restore and maintain the alveolar epithelium in injured lungs. His paper also introduces a revolutionary method of categorizing the degree of damage on heterogeneous lung tissue, and deftly shows how FGFR2 controls the cell fate of AT2 stem cells in *damaged* zones. Commenting on the translational potential of his study Liberti states that, "*A major challenge in identifying therapeutic targets to ameliorate lung disease lies in our limited understanding of both the essential factors that control cell fate decisions and the consequences of those decisions for lung regeneration. Without knowing the major signals regulating cell fate decisions in the lung, we have no way to enhance the regenerative process.*" Combined with his previous work on neonatal lung regeneration following lung injury [5], Derek Liberti's research contextualizes how important and precariously balanced cell fate decisions are for the purposes of restoring damaged tissue and maintaining tissue homeostasis.

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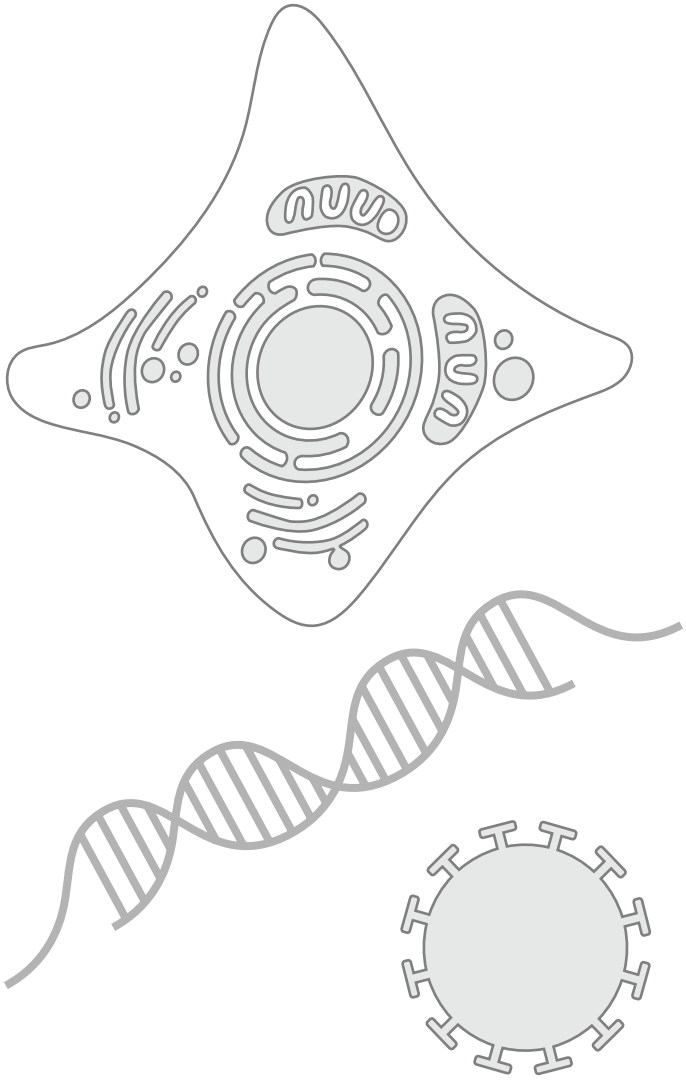


Faculty Spotlight

Insights from Dr. Marie Guerraty of Penn's Department of Medicine

Yee Hoon Foong

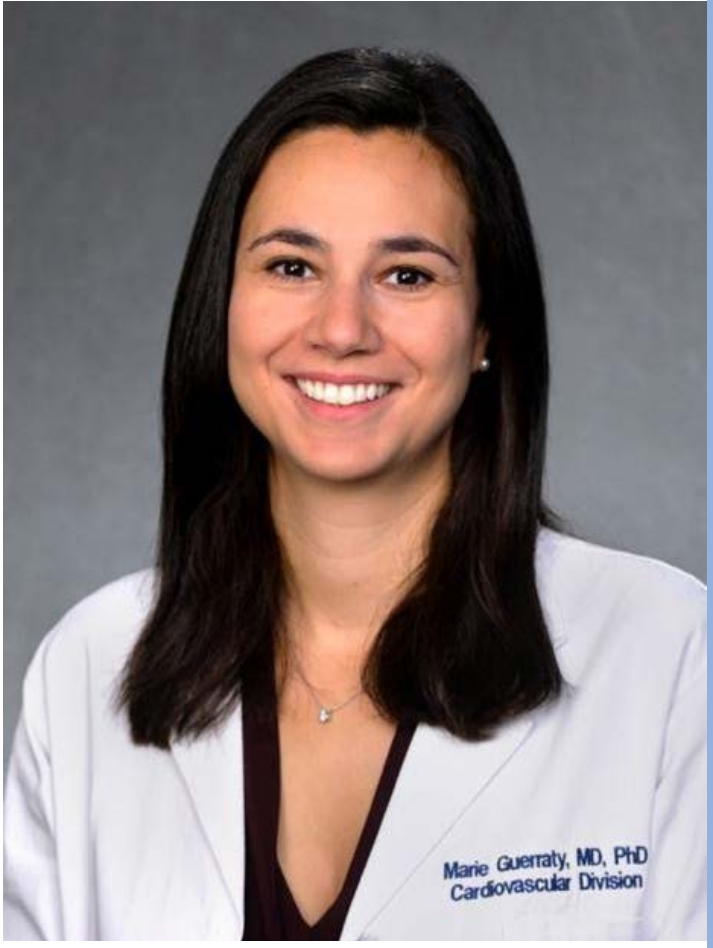
Dr. Marie Guerraty is a physician-scientist who divides her time between basic research and clinical practice as a cardiologist and cardiac imager. She is an Assistant Professor of Medicine in the Perelman School of Medicine and is launching her own lab investigating the coronary microvasculature.

YHF: Could you tell us a bit about your doctoral thesis training? What did you study and how did you become interested in the cardiovascular field?

MG: I received my PhD from the Department of Bioengineering in the Penn School of Engineering and Applied Sciences as part of the Medical Scientist Training Program. I did my dissertation with Dr. Peter F. Davies who is Professor of Pathology and Director of the Institute for Medicine and Engineering. My project was focused on understanding the role of endothelial cells in early aortic valve sclerosis in vivo.

I chose to work with Peter even before I knew how interested I would be in cardiovascular research as a field. He struck me as an ideal mentor in terms of his commitment to training and his ability to balance giving trainees independence while helping to guide them when they needed help. He is also a pioneer in the field of endothelial biology, and his passion for vascular biology was contagious. I also loved cardiovascular physiology in medical school. The cardiovascular system, and the clinical practice of cardiology, is very quantitative and logical, and there are concrete interventions and treatments available.

YHF: You have completed your PhD and MD trainings at Penn and are now establishing a new lab here. What drew you to Penn and why did you decide to stay?



Dr. Marie Guerraty

MG: Penn is such a wonderful and unique place, and the thing that drew me and then kept me here is the interdisciplinary and collaborative nature of the institution. Crossing disciplines and working with experts in other fields leads to more exciting science. There is no other place where the medical school and main hospital is so close to the engineering school, and the veterinary/nursing/law/business schools; there is a cyclotron in the middle of the Medical School campus minutes from where mouse, large animal, and human experiments are conducted. This physical proximity promotes scientific collaborations.

YHF: What are your fondest memories from your graduate training? Did you ever have an "epiphany" moment?

MG: My biggest epiphany moment during graduate training was when I realized that the challenges in making things happen and moving my project forward were part of the training. It wasn't a road block or a derailment. It was a key part of the

curriculum.

YHF: Can you briefly describe the research in your lab?

MG: My lab is a basic-translational lab focused on studying the coronary microvasculature. We are most interested in understanding how developmental programs regulate the microvasculature in adults. We combine small animal models and functional imaging with cell and molecular biology to understand how the coronary microvasculature is regulated and how it becomes dysfunctional. We also tie in our work on cellular mechanisms with in vivo physiology and ultimately with health and disease in people. Coronary microvascular disease accounts for 30-50% of the burden of ischemia heart disease, but, unlike atherosclerosis, it's a relatively young field which makes it particularly exciting and timely.

YHF: What is your mentorship style?

MG: I see my role as a mentor to nurture curiosity and support trainees through the inevitable joys and challenges of doing science. I have benefited tremendously from mentors who have given me space to explore new ideas and have encouraged me to think creatively.

YHF: In your experience, what are important characteristics for students to thrive in graduate school?

MG: The most important characteristics for students to thrive in graduate school are patience and perseverance. It's not a coincidence that patience and perseverance are the also the most important characteristics needed for a scientist to thrive in science. It can be difficult to transition from a well-paved undergraduate training path with clear course work and testing requirements to a dissertation where the benchmarks are more abstract and progress is very non-linear. And a key part of patience is being comfortable with perpetual learning. I also try to model this. After so much training, I have learned a lot, which has served to make me even more aware of how little I know.

YHF: What is your advice for students progressing through their PhD or MD/PhD training?

MG: Enjoy the ride. You are not completing training and waiting for your life to start. Your life has started, and you should enjoy the challenges along with the successes. A failed experiment or a rejected paper is a time to rally with your mentor and colleagues, get support, regroup and try again. It is not an assessment of you as a person or a scientist.

YHF: What are your hobbies outside of the lab?

MG: I enjoy reading (and am eagerly awaiting Louise Penny's new book coming out in August), running, bike riding, going to the beach, and exploring new restaurants. My husband (whom I met in grad school at Penn!) and I have two little boys, and we've learned to blend our interests with things that they can enjoy with us.

YHF: What are your favorite summer activities?

MG: When I first moved to Philadelphia, it took me three years to realize how close Philadelphia is to the beach and how beautiful some of the nearby beaches are. Now we go whenever we get a chance for a short day trip or for a few nights. My other favorite summer activity is blueberry and strawberry picking followed by eating and baking with blueberries and strawberries. New Jersey is called the Garden State for good reason.

YHF: Are you interested in taking in rotation students and if so, what qualities are you seeking in the students?

MG: Yes! The most important quality is curiosity and eagerness to learn something new.

The Guerraty Lab is located in 11-167 South Perelman Tower. Any trainees interested in learning more about her work can contact her at marie.guerraty@pennmedicine.upenn.edu.

Special Interest

A Burden Unevenly Shared:

The COVID-19 pandemic and its disproportionate effect on early career women scientists

Sylvia Stankov

Graduate school is punctuated by (well-meaning) questions about where you're going. As soon as you've settled into your lab, and surely after you've passed your preliminary exam, faculty and family collectively declare open season. What are your next steps? When will you graduate? When will you get a "real" job? While many trainees aren't exactly sure about their dream career trajectory, they almost always have a hard line on whether or not they'll be staying in academia. At Penn, CAMB graduates are an even split; as of January 2020, 50.2% of alumni are in the academic sector[1].

Many factors influence the decision to pursue an academic career, and hesitations around work-life balance and perceived barriers, especially those faced by women, are paramount. Many studies put numbers to these hesitations. Women have earned more than 50% of all doctoral degrees since 2009 but as of 2018, women hold only 34.3% of full professor positions[2]. Women identifying as Black or Hispanic only make up a little over 8% of tenured faculty[2]. Moreover, a randomized controlled trial of women and men researchers with identical CVs found that CVs from male applicants received higher scores and were more likely to be seen as having leadership potential[3]. Studies have also underscored a persistent pay gap between men and women holding identical positions[4] and, in 2017, reports of sexual harassment were found to inflict "significant damage to research integrity" and led to loss of talent[5].

More recently, additional concerns have emerged from the COVID-19 pandemic, particularly for early career scientists with caregiving responsibilities. Drs. Michelle Cardel, Natalie Dean, and Diana Montoya-Williams from the University of Florida and CHOP penned their views and brainstormed solutions in their article "Preventing a Secondary Epidemic of Lost Early Career Scientists – Effects of COVID-19 Pandemic on Women with Children" [6].

Drs. Cardel, Dean, and Montoya-Williams deftly highlight pre-existing barriers for women with chil-

dren that have been exacerbated by the pandemic. They note that the prime reproductive years and family planning milestones (i.e., marriage and childbirth) overlap with the early stage of scientific careers. Shockingly, 43% of women (versus 23% of men) leave full-time STEM employment after their first child[7]. Even among high-achieving early career physicians and researchers, women shoulder more domestic and childcare responsibilities and are less likely to have a stay-at-home partner[8].

Over the past year and a half, this disproportionate burden on women scientists has only grown. As the workforce moved home, women took on more childcare, homeschooling, and elder care responsibilities. There's even a downward trend in manuscript submission among women[9]. These historical observations and recent events demonstrate that early career women scientists, and women of color, are particularly vulnerable in the academic pipeline[6].

So, what's the plan? Past efforts to equalize the playing field haven't had much success. In fact, gender-neutral tenure clock stopping policies for family-related reasons substantially reduced female tenure rates while increasing male tenure rates[10]. These findings are likely because women use parental leave time to physically recover from childbirth, feed, and bond with their baby. Meanwhile, men also bond with their newborn, but report greater flexibility in that time. Clearly, equal policies are not necessarily equitable policies.

Drs. Cardel, Dean, and Montoya-Williams suggest policies and strategies to make meaningful changes for women in the academic pipeline[6]. They highlight a need for infrastructure that identifies child care resources and that allows for flexible working arrangements. The authors also encourage increased funding opportunities for early career researchers, especially "women-only" funding opportunities, extensions on current grant periods or submissions, and administrative supplements to offset costs of pausing and resuming research activities. Concerning promotion and tenure, Drs. Cardel, Dean and Montoya-Williams suggest that editors prioritize women-authored papers, monitor sex breakdowns in promotion and tenure, and monitor allocation of new teaching service loads. Their biggest call, however, is for a shift in what defines academic success; Drs. Cardel, Dean and Montoya-Williams value scientific impact beyond manuscripts and grants. They believe that science communication, community-based implementation, dissemination, effective mentoring, and advocacy work are equally meaningful indicators of success in academia.

"...a diverse faculty pool means that underrepresented mentees will be able to identify someone who looks like them and as a result may have similar lived experiences, fears, hopes, and challenges."
-Dr. Montoya-Williams

While Drs. Cardel, Dean, and Montoya-Williams's article centers on women with children, all early career scientists can stand to benefit from a more equitable and inclusive academic environment. Recruiting and retaining a broader talent pool means more opportunities for quality mentorship of the next generation and strengthens current research endeavors. Dr. Montoya-Williams highlights that "a diverse faculty pool means that underrepresented mentees will be able to identify someone who looks like them and as a result may have similar lived experiences, fears, hopes, and challenges." She also believes that while "racial or gender concordance is not necessary for a fruitful mentorship relationship, shared backgrounds can increase the chances that a mentee might feel safe to really be themselves, share their struggles and receive advice or guidance that comes from a place of deeper or shared understanding." Dr. Cardel adds that the onus is "on the academic institutions who must realize [that] their future competitiveness and social impact" depend on these changes. As for current faculty and trainees, Dr. Montoya-Williams emphasizes how important it is "to be inclusive, to reach out, to speak names of mentees in rooms they are now privileged enough to be in...to change environments and cultures into ones that foster collaboration and are uplifting for all." While half of us will likely pursue careers outside of academia, embracing these ideals and empowering our peers and col-

leagues wherever we are can only strengthen our community and further scientific progress.

For additional reading, check out Dr. Cardel's 2020 article on "Turning Chutes into Ladders for Women Faculty: A Review and Roadmap for Equity in Academia" (https://www.liebertpub.com/doi/full/10.1089/jwh.2019.8027)

To read Dr. Montoya-Williams full interview responses on diverse mentorship and achieving personal goals during graduate school and early training, head over to our blog.

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Positivity Posts

We asked for you to share your positive, happy, or uplifting stories. In this issue, we share Emily Shea's story!

"I adopted a two-year-old hound dog (and named her Rory after Gilmore Girls)!"

Congratulations, Emily! If you would like to share your positive stories, please do so **here**!

Opinion Piece

India's Gasp for Oxygen

with a commentary on global vaccine rollout

Kanika Jain

After a year-long global travel ban, safety debates, and strict lockdowns, I finally got the opportunity to visit my family in Delhi, India during late March this year. COVID cases in India had maintained a steady decline since November 2020, with an average of 10,000 cases/day reported country wide. Vaccination rollouts had successfully commenced in major cities for frontline workers and the elderly population aged sixty years and above. COVID-appropriate behavior like avoiding mass gatherings, social distancing, and wearing face masks had become the new normal. Being the 2nd most populated country in the world, since the onset of the pandemic in March 2020, epidemiologists had warned India to be analogous to a fire-pit, where if lit uncontrolled, COVID cases would spread like a wildfire. However, almost everyone in the country was complacent that India had successfully fought and controlled the spread of COVID-19.

Two weeks into my vacation, India was gripped by a devastating second wave of coronavirus that crippled the entire nation. On April 7th, India recorded a daily average of 100,000 new cases, and these numbers soared higher during the weeks to follow. The positivity rate in the capital, New Delhi, rose to 36% and statewide lockdowns were imposed. The steep increase in cases by the highly transmissible 'Delta' variant overwhelmed one of the best healthcare systems on the continent. There was an increasing shortage of healthcare equipment like ICU beds, oxygen cylinders, drugs, and injections. Even basic amenities like oximeters and thermometers were becoming harder to find.

On the ground, these numbers translated to devastating stories of individuals gasping for breath. The high transmissibility of this variant meant that if one person in the family was positive, the entire family would soon contract the virus, including children and young adults. The symptoms reported were more severe than those reported earlier in the pandemic, ranging from previously reported loss of smell and taste, dry cough and cold to debilitating body aches, weakness and fatigue, high fever, headache, loss of appetite, severe abdominal pain, and diarrhea. In the second week of the infection increasing numbers of people reported sudden drop in their blood oxygen levels, severe chest pain, and inability to breathe. These symptoms often lead to blood clots and heart attacks, and required immediate hospitalization. A majority of people who lost their lives belonged to the unvaccinated demographic ranging between forty-five to fifty-five years of age, orphaning many young adults in their twenties.

An over-burdened healthcare system resulted in shortage of trained staff and resources to handle the increasing load of RT-PCR testing. People were requested to stay at home upon onset of mild symptoms in an effort to save hospital beds for others in severe conditions. As a result, with no specific antivirals known, people were encouraged to do a course of strong antibiotics like azithromycin, multivitamins including Vitamin C and Vitamin E, zinc tablets, anti-diarrheals, and antipyretics with the onset of mild symptoms. News channels soared with stories of oxygen shortage even in well-established private hospitals, continuous debates between central and state governments playing the 'blame games' for the abysmal condition of the country, and overburdened crematoriums. Personally, I can never forget attending a funeral for a distant family friend virtually, the constant state of fear for the safety of my family and friends, mental exhaustion and disarray. For the first time since the start of the pandemic in March 2020, it truly felt like the inevitable end of the world, an apocalypse in totality!

It was heart wrenching to see social media abuzz with pleas for hospital beds, anti-viral Remdesivir injections, and oxygen cylinders. Volunteers groups composed of college students, retired government and military officials, religious groups, social workers, doctors from other countries, and people from all walks of life came together and worked day-and-night to report the availability of healthcare resources, manage contact tracing, provide emotional and psychological aid, or just help elderly and un-abled with groceries and transportation. Gurudwaras (holy place of worship for Sikhs) organized drives to feed the homeless and daily wage workers, healthy families cooked food to deliver to the families infected, both known and unknown. There were reports of doctors and nurses converting their homes into makeshift hospitals to admit patients due to the growing shortage of hospital beds, auto-rickshaw drivers transporting patients for free, and volunteers helping to cremate bodies of people who had died and didn't have anyone to take care of the final rites. It was deeply inspiring to see a diverse nation like India, united in their sorrows, fighting the horrors of the pandemic together.

The delay in vaccinating developing countries severely threatens the social and economic recovery in developed nations by leaving large reservoirs of coronavirus circulating, providing grounds for the virus to mutate and transmit to developed countries.

More than 400,000 lives have been lost within India since the start of the pandemic. Questions have been raised on the credibility of the state and central governments in power, political blunders like allowing super-spreader religious and political mass gatherings, government's strategies to combat the situation, and most importantly the delay in vaccinating middle-aged and younger demographic of the country. While the steep rise in the number of positive cases was met with an equally steep decline in the positivity rate of infection, the second wave of the pandemic is far from being over. Availability of vaccines from multiple vendors, standardization of vaccine prices by the central government, and an increase in vaccination drive in both urban and rural India remain critical for the overcoming of the pandemic.

The resurgence of the devastating second wave of COVID-19 in India at the same time when COVID-19 restrictions were being relaxed in the other parts of the world, highlights the striking gap in equitable access to vaccines between developed and developing nations.

Factors such as over-purchase of vaccines by rich nations to vaccinate their entire population several times over, lack of transparency in quoting global vaccine prices, stringent policies around intellectual property rights and technology transfer, and lack of adequate healthcare infrastructure are some of the major causes for this inequity. The second wave of the COVID-19 pandemic in India dealt a serious blow to The Serum Institute of India (SII), the largest vaccine producer of the world, and one of the major suppliers for vaccines under COVAX (global initiative to ensure rapid and equitable access to COVID-19 vaccines for all countries, regardless of income level). As a result, only 41 million doses of the initially planned 200 million doses have been rolled out by SII amongst more than 100 countries around the world, further exacerbating the problem. The delay in vaccinating developing countries severely threatens the social and economic recovery in developed nations by leaving large reservoirs of coronavirus circulating, providing grounds for the virus to mutate and transmit to developed countries. There have not been graver times than this to remember that we all see the same sunset. Let's be united under humankind and adopt joint collaborative efforts to ensure equitable vaccine access throughout different parts of the world, thus pushing the pandemic to its end.

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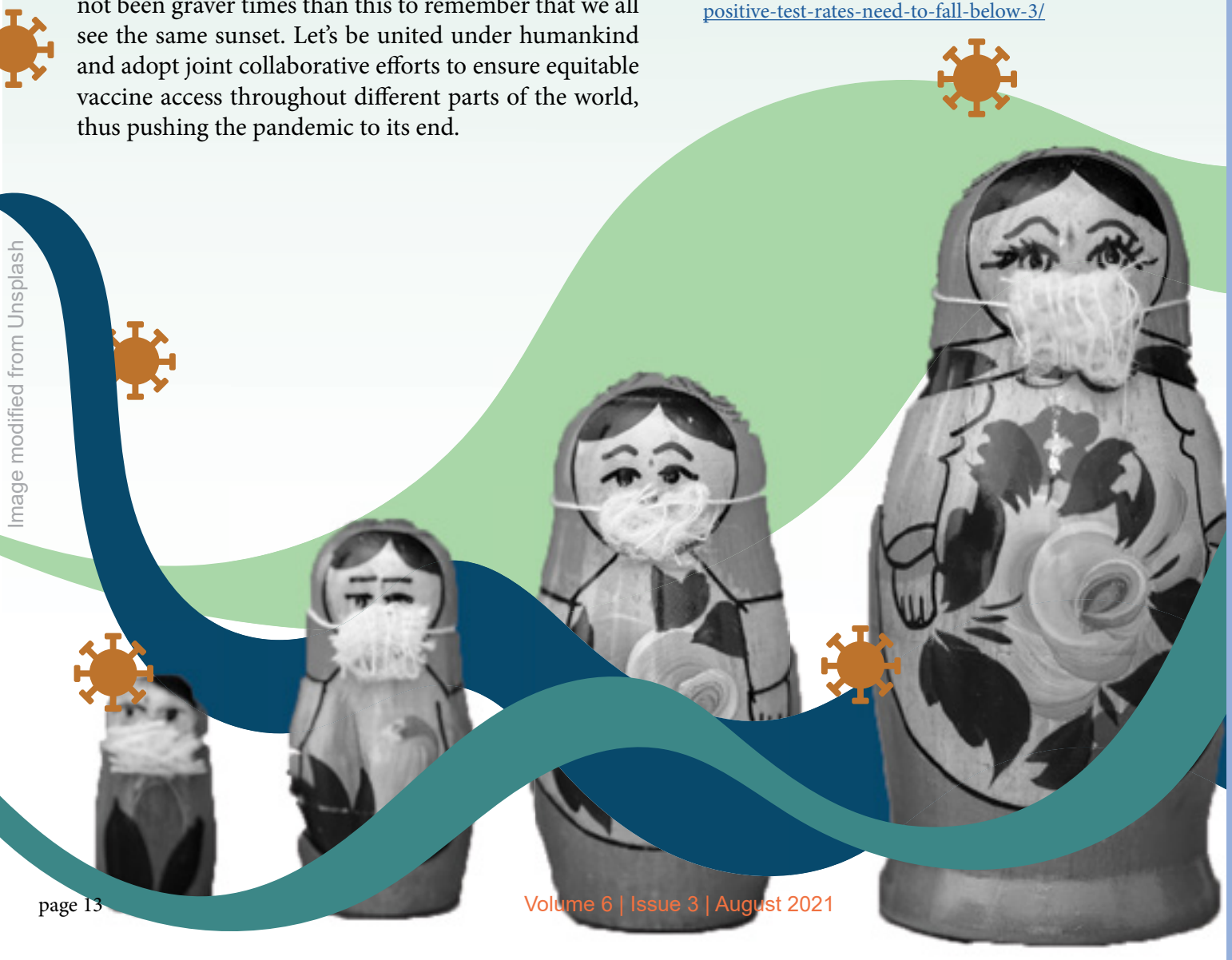

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