The Summit Weekly Update: March 26, 2026

Chronic Illness and Vaccine Prevention Presentation – Sara Y. Tartof, PhD, MPH, Epidemiologist/Research Scientist, Kaiser Permanente Southern California Department of Research and Evaluation; Professor, Department of Health Systems Science, Kaiser Permanente Bernard J. Tyson School of Medicine

Sara Y. Tartof, PhD, MPH, summarized her research, which found that COVID-19 carries a higher risk of severe post-acute sequelae (PAS) than influenza, but vaccination and antiviral treatment may attenuate the risk in both types of infection.

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Post-acute sequelae were found in up to 30% of people with COVID-19 in the early days of the pandemic. To assess whether PAS is as common following other respiratory viruses, Dr. Tartof and colleagues compared people who received care for either COVID-19 or influenza through Kaiser Permanente of Southern California health system in the 2022–23 respiratory illness season (study available here). With a large member population from which to draw and capacity to review patient records in every setting from telehealth visits to hospital admissions, the study assessed nearly 75,000 cases of COVID-19 and almost 19,000 cases of influenza.

Key Findings

- People with COVID-19 had only modestly higher overall risk of PAS up to 180 days after the initial infection than people with influenza, but COVID-19 carried a greater risk of severe PAS requiring inpatient admission.
- People with COVID-19 whose initial infection warranted inpatient care were at higher risk of severe PAS than those hospitalized for influenza.
- Vaccination and antiviral treatment may attenuate the risk of PAS following COVID-19 and influenza.
- Multiple respiratory viruses are associated with PAS.
- PAS following influenza may be under-recognized and warrants further study.

QUESTIONS & ANSWERS

Q: Is it possible that some portion of the difference between PAS following COVID-19 versus influenza infection is related to the fact that vaccination rates are much higher for influenza than for COVID-19?
Sara Tartof (Kaiser Permanente): For those who are up to date [on their vaccinations], we don't see a lot of difference between patients with COVID and patients with flu, if that is what you're referring to here. And I think one of the key points is that it really looks like, from the analyses, the severity of your initial infection makes a really big difference. And being up to date with your vaccines, I think, really plays a role in your risk for severe infection and your risk for developing PAS, so I think it kind of evens the playing field a little bit more between the two different infections.

Q: If you flip it, what about the unvaccinated patients?
Sara Tartof (Kaiser Permanente): I think for the unvaccinated, you probably are likely to see a higher risk with COVID-19.

Q: The data are fascinating, thank you for sharing. It seems there's really an underappreciation of PAS from other respiratory viral infections. Also, it's very important to make sure people are aware that there was a meaningful positive impact of vaccines and antivirals.
Sara Tartof (Kaiser Permanente): I 100% agree. I think that is a really key message here. It kind of always comes back to that message in so much of the work that I do.

Q: What was the motivation for comparing PAS from flu versus COVID-19? What was the hypothesis? Why these two?
Sara Tartof (Kaiser Permanente): Oh, that's a really good question. I'm trying to go back. So, this was done in collaboration with the Centers for Disease Control and Prevention (CDC), and it really was a very highly collaborative project. I almost want to say that was a question that may have come from our CDC collaborators. I've done plenty of work on PAS with COVID, and it was the topic that everyone wanted to look at. Those of us who have studied influenza over time realize that the issue requires a little bit of context. I think sometimes when there's something new, we get really sort of excited about it, but we can forget that these things already existed, and we have to maintain our public health efforts for other things as well. A huge burden may already exist for other things, but they haven't been pointed out for a while. So, I think it might have been along the lines of just sort of providing some context for what we were seeing. And also, it was a question that a lot of people, I think, were asking CDC at the time.

Q: It's just interesting that there appear to be some of these other differences by organ system. I'm wondering if, in part, these two particular diseases were chosen because the rapid tests for both of them are so commonly used, so you would have adequate power to look at a large number of people. I mean, people don't really test for human metapneumovirus too much.
Sara Tartof (Kaiser Permanente): Respiratory syncytial virus (RSV) is another area of interest. We're always lamenting the under-testing of RSV. I mean, these are all molecular tests that we included here, and I almost wanted to change the axes, the scale of the axes for

this, because I feel like you could look at this and sort of think you're seeing a lot. You know, it looks like some things have a higher risk difference, but then when you look in the other time period, it doesn't look as much. … We did do a lot more analyses looking at the different systems, but it just felt like a lot to present everything.

Comment: There are many studies that have been done by lots of different researchers all over the place looking at cardiovascular complications of influenza. But it got a lot more attention, in part because of the younger males that had some risk of myocarditis after [COVID-19] vaccination. We know that influenza caused significant cardiovascular complications with acute infection. I think that's another takeaway: just a reminder about influenza and all the cardiovascular sequelae from that. So, thank you.

Q: Regarding the increase in [PAS in the] inpatient setting among COVID versus flu patients: Were the patients just sicker than those with flu in the inpatient setting at baseline?
Sara Tartof (Kaiser Permanente): No. Patients with COVID were overall sicker. Were we perfect in our adjustments? Perhaps not, but we did really try to balance those two groups for comparison. The nice thing about working here at Kaiser is we really do have fairly complete [patient data]. Actually, we only included people who have been members for a while, so we know that we were capturing their underlying comorbidities. We do feel that our capture of whether somebody has a lot of comorbidities or not is complete, and so I don't think that would be the reason.

Association Between Shingles Vaccination and Slower Biological Aging: Evidence from a Population-Based Study – Eileen M. Crimmins, PhD, University Professor University of Southern California (USC) Leonard Davis School of Gerontology, AARP Chair in Gerontology; Co-Director, Multidisciplinary Research Training in Gerontology PhD Program; Co-Director, USC/University of California, Los Angeles Center on Biodemography and Population Health

Eileen M. Crimmins, PhD, summarized research confirming that shingles (specifically the now discontinued Zostavax®) vaccination slows aging and protects against dementia but does not appear to have an effect on cardiovascular health.

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Some literature suggests that the shingles vaccine has broader benefits beyond protection against shingles—namely, reducing the risk of dementia and heart disease. Dr. Crimmins and colleagues looked at seven measures of biological aging and a composite score of all seven measures among subjects of an ongoing national survey. The study (available here) looked at uptake of Zostavax from its introduction in 2006 until 2016 and controlled to eliminate bias toward healthier people who get vaccines.

Key Findings

- The shingles vaccine slows down molecular aging, according to epigenetic and transcriptomic measures. The effect is meaningful, amounting to about 2–3% slower aging per year.
- Vaccinated people had a lower inflammatory load, calculated using innate and adaptive immunity measures. Chronic inflammation is considered a core mechanism of diseases of aging.
- Vaccination significantly lowered composite scores. Changes in epigenetic, transcriptomic, and composite scores showed up earlier and were sustained over time. The benefits on inflammatory load showed up later.
- Shingles vaccine is strongly related to reduced risk of dementia and mild cognitive impairment, and the effect persists for at least 6 years.
- The study did not find an association between shingles vaccine and concurrent heart disease or onset of heart disease within 6 years of vaccination.

The findings appear to be unique for Zostavax® vaccine; neither influenza nor pneumococcal vaccine has the same impact on biological aging. It appears that the vaccine breaks the link between the latent virus and chronic immune stimulation. Studies of the currently available Shingrix® vaccine are underway; it may have an even stronger effect on slowing biological aging. Vaccination may be a key factor in healthy aging.

QUESTIONS & ANSWERS

Q: Are you talking about shingles vaccination of any type, or is it a specific vaccine?
Eileen Crimmins (USC/UCLA): Zostavax®. That was the only thing available for a long time. One of the nice things about that is it was clearly available one year, it was clearly stopped one year, and then the other vaccine came right in, so there's sort of good timing. You're not seeing a lot of overlap.

Q: So, you'll be able to kind of identify whether it is a zoster vaccine, or is it something specific to Zostavax?
Eileen Crimmins (USC/UCLA): Right. Or is it something even better with the newer vaccine that's adjuvanted?

Q: For clarification, it's not that the vaccine slows aging, but that protection from the varicella zoster infection slows aging. So, something about the latent infection with varicella that then erupts in later life as shingles?
Eileen Crimmins (USC/UCLA): Well, I think we think some of these latent viruses are really the thing that challenges your system. So, yes, that's the problem, that the viruses are there, and that's one. And also, cytomegalovirus (CMV) is a gigantic one, and we're hoping a vaccine comes for that soon. So, those are things that you have to sort of maintain control of, and, as you age, it gets harder and harder for your system to sort of tamp down what they're trying to do to you. So, yes, I agree. You don't change your aging, what you do is you change the force that the virus is having on you. So that's the cause, and that is changing the rate of aging, essentially.

Q: Did you look at differences in aging markers for people in your sample who had a history of shingles disease?
Eileen Crimmins (USC/UCLA): No, we did not. And, it's not clear we can do that, because we don't have the health records.

Q: Are there other interventions you have tested or are aware of that show similar geroprotective effects?
Eileen Crimmins (USC/UCLA): I think the first things that came out have been diet and exercise, and they are sort of protective of these same mechanisms. So, one of the reasons that these kinds of measures evolved was that the Food and Drug Administration (FDA) didn't allow interventions that tested things that people thought delayed aging, or where aging was the outcome, because there was no good way to measure aging. And the FDA required, if you were going to have a trial, you had to have a diagnosed disease. So, it was a 10-year process by which people in aging [research] developed these measures of biological aging, basically so they could get a measure that was acceptable to the FDA to actually use in things like drug trials and interventional trials. So, yes, these kinds of measures are the things that are being used in all kinds of trials. One of our colleagues is investigating now the effects of 150 interventional trials on these kinds of things. So yes, there's lots of stuff going on, and it has to do with supplements and lifestyle changes, plus drugs.

Q: The use of the word health-span extension versus lifespan extension: is that a deliberate choice?
Eileen Crimmins (USC/UCLA): I think most of us in aging now are interested in increasing the length of healthy life, not just the length of life, per se. Part of the reason is that what we've done over the past 25 years has probably increased the length of life by saving people, but they've lived with disease and disability, and healthy life hasn't increased as much. So, it is a focus on healthy life.

Q: With regards to the mechanism, there is something about latent varicella zoster virus infection that is impacting aging, and if we somehow protect from that by improving our immune response to the virus, we prevent the damage from the latent infection. In other words, there's nothing in the vaccine itself that's the fountain of youth; it's the protection from the damage from the virus. Or, I would argue, maybe it's also the reduction of your biological stress in keeping the virus latent. That could be the other thing: You're not working so hard to suppress the virus from erupting.
Eileen Crimmins (USC/UCLA): Yes, that's what we think.

Q: Would individuals who are immunized with the chickenpox virus vaccine as children show delayed aging due to a lower risk of shingles?
Eileen Crimmins (USC/UCLA): As I understand it, the need for the shingles vaccine is not going to go away as we actually get a population that has been immunized as children. From what I've heard, we're going to continue to need the shingles virus because, already, people do have a little bit of varicella virus, even from the vaccine they initially got as children. I thought it was going to go away, this need, but I have been told it is not.

Q: I was just curious about the clinical implications. Do you think that recommending the shingles vaccine for folks that are younger would potentially be something that you would consider?
Eileen Crimmins (USC/UCLA): So, people, when they decide how to recommend vaccines, they sort of have a complicated calculation of how long it's going to last, and whether you're going to need another one, and how much it costs, and everything, and that's why they started at 60, but they've gone down to 50. And we all know a lot of people who are younger who got shingles. I'm not a clinician, so I don't make recommendations, but if I were the least bit immunocompromised and I were 35, I'd get it if I could. I think the fact that they went down to 50 means that there was no magic number at 60. And there's probably no magic number at 50, it's just the probability gets less and less as you get younger. You're able to control it.

Q: Is there a lower risk of aging, because there is now less infection?
Eileen Crimmins (USC/UCLA): We actually think that the lower rate of infection that has occurred over the last hundred years in children has led to stronger bodies and stronger brains, as we have instituted all kinds of things that prevent serious infectious diseases in children. So, yes.

Announcements

- Registration is open for the 2026 National Adult and Influenza Immunization Summit, May 19-21, 2026, at the Crowne Plaza Atlanta Perimeter at Ravinia, 4355 Ashford Dunwoody Rd, Atlanta, GA 30346 (https://www.izsummitpartners.org/2026-naiis/). Applications are no longer being accepted for the poster session. The Summit will present its annual awards at the event.
- Registrants are encouraged to book a hotel room using this link by April 23: https://book.passkey.com/event/51059389/owner/55306/home