Hi everyone, welcome to our second seminars in this fall. This belongs to our Climate Change and Health Center four seminar series on climate change and health and today we’re very fortunate to have Dr Virginia Pitzer. She’s an Associate Professor of Epidemiology from the Microbial Disease Department. So she’s also one of our pilot project awards winners. Her research mainly focused on the new mathematical modeling of the transformation dynamics of infectious diseases. So without further ado, the floor is Gina Pitzer.

Well thanks very much for the introduction and hopefully we can switch to my screen. I think you need to enable share screen. Gina, I think you’re good to go. Got it.

Okay, can everyone see the presentation now? Okay, well thanks very much for the introduction and for the opportunity to speak with you all today and so as Kai said, I’m gonna be talking about some of the ways in which we use different models as well as our mechanistic understanding of relationships.
in order to predict the impacts of climate change,
specifically for infectious diseases.

And so when it comes to predicting the impacts
of climate change on infectious diseases,
often the way that this is done
and let me see if I can get this working,
is to take a model of climate projections over time
and for example this is projections around variations
in temperature from the current day up through 2100
and to combine this with a model
for the incidence of disease given climate
and use this to get projections of the relative risk
data
on model projections of the relative risk of diarrhea
over time given the model forecast
for increases in temperature over time,
suggesting that by 2010 to 2039,
you should see a median increase
or relative risk of around 1.1 and by 2040 to 2069,
a relative increase, median increase
of around a relative risk of 1.2 and upwards of 1.3
by the 2070 to 2099 time frame
and voila, I mean that’s basically you know,
one of the ways that people have gone about doing this.
So you know, thank you.

I’ll take any questions now.
But of course if it were as simple as that,
this would be a very short talk
and I’d argue that really the most difficult part
of understanding the climate of this is
really understanding the true climate disease relationship and in particular, the causal effects of climate change on infectious diseases which is really far from straightforward and typically cannot be determined by the simple regression type analyses that were used in that previous study and are often used in many types of analyses of relationships between climate and chronic diseases for example. And so I'd argue that in order to really have a true causal model for linking climate to infectious diseases, there are really three main criteria that are needed to be met and these include that the change in infectious disease incidents really must occur at the right time, in the right place and in the right direction in order to be causally linked to a change in climate and the last of these criteria really requires that you have a hypothesis about the mechanism through which climate impacts on infectious diseases. well the first two really involve the careful analysis of spatiotemporal data and so in this talk I’m really going to begin by talking about the mechanisms through which climate can have important impacts on infectious diseases, including both the direct effects of climate as well as some of the indirect ways in which climate can impact on infectious diseases,
and then I will talk about how we go about identifying and quantifying these associations between climate and infectious diseases, including the types of data that are often used to draw these associations.

And I'll largely be drawing on examples from my own work, particularly when talking about these quantitative approaches.

And then finally I'm gonna end with just some challenges and some opportunities to really get further when it comes to making these predictions around climate change on infectious diseases.

And so one of the main ways in which climate can have an impact on infectious diseases is through the effects of climate on pathogen survival and, or replication within the environment.

And so one example here is work that's been done by researchers to understand the effects of temperature and humidity on the transmission of influenza where researchers use guinea pigs which are a great kind of model system for measuring influenza transmission, to examine how the level of transmission happened from an infected guinea pig to a susceptible

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an exposed guinea pig that was housed in a separate cage, but downwind of this infected guinea pig when they modulated the temperature and humidity of the cages in which these guinea pigs were housed. And generally what they found was that both survival and transmission of influenza virus was really enhanced at low temperatures and low relative humidities, and when colleagues went about and re-analyzed some of this data, what they were able to show was that it was really absolute humidity or vapor pressure which was even better at explaining some of these associations and in particular the combined effect of temperature and relative humidity and then based on this relationship, we were able to use some of this data and combine it with mathematical models of flu transmission to show that by incorporating the relationship between absolute humidity and flu transmission into these models, we could better forecast the timing of seasonal flu epidemics happening across the US each year where often the epidemic tended to be preceded by a dip in absolute humidity before each flu epidemic happening in each year. So another way in which climate might affect infectious disease incidents is through its impact on host defenses and host behavior and so you know, we’ve all been told to bundle up.
in the winter so that we don’t catch a cold and of course we know that while we don’t actually catch colds by being cold, there is actually some potential truth to this mechanism and so when it comes to colds which are caused by rhinoviruses as one example, it has been shown by work from Ellen Foxman and Akiko Iwasaka here at the med school that when the nasal cavities are exposed to warmer temperatures, they tend to exhibit higher levels of expression of Interferon gamma which is an important first line in defense against viruses such as rhinoviruses and other cold viruses. But these levels of Interferon gamma tend to be quite a bit lower when temperatures are lower, leading to potentially slightly impaired kind of first-line immune responses in the nasal cavities at colder temperatures which may be part of the reason why we do indeed tend to get colds more often during the winter season. And so another way in which climate can impact on infectious diseases is through its impacts on the risk of human exposure. And so for example it’s known that flooding can increase the risk of exposure to various waterborne pathogens including Leptospirosis which is a febrile illness that’s transmitted primarily through the urine of rats and so when you see these heavy rainfall events, the rat urine can get washed into the street.
0:11:33.02 –> 0:11:35.56 and into sewers where people are walking through
0:11:35.56 –> 0:11:40.411 and the bacteria can then enter into humans
0:11:40.411 –> 0:11:42.49 through small cuts on the feet
0:11:42.49 –> 0:11:45.134 when people are walking around in these floodwaters
0:11:45.134 –> 0:11:49.49 and mud that has been contaminated by this rat pee
0:11:49.49 –> 0:11:51.61 and this is something that has been studied
0:11:51.61 –> 0:11:56.61 by Albert Coe who’s the department chair in EMD here
0:11:56.79 –> 0:11:58.29 at the School of Public Health
0:12:00.38 –> 0:12:03.98 and then finally for vector-borne diseases,
0:12:03.98 –> 0:12:04.813 it’s very clear
0:12:04.813 –> 0:12:08.17 that climate can have really important impacts
0:12:08.17 –> 0:12:11.9 on the risk of disease often through its impacts
0:12:11.9 –> 0:12:14.496 on factors such as vector survival,
0:12:14.496 –> 0:12:17.555 vector fertility and development
0:12:17.555 –> 0:12:22.555 as well as the biting behavior of various vectors.
0:12:23.37 –> 0:12:26.77 And so this is why diseases like Dengue fever,
0:12:26.77 –> 0:12:30.04 Chikungunya and most recently Zika virus
0:12:30.04 –> 0:12:35.04 really tended to be confined primarily to the tropics
0:12:35.25 –> 0:12:39.353 since the adult Aedes aegypti mosquito
0:12:39.353 –> 0:12:41.591 as well as the Aedes albopictus mosquitoes,
0:12:41.591 –> 0:12:44.19 the survival of these mosquitoes is
0:12:44.19 –> 0:12:45.71 really temperature dependent
0:12:45.71 –> 0:12:47.93 and so at these warmer temperatures,
0:12:47.93 –> 0:12:49.84 they tend to live longer,
0:12:49.84 –> 0:12:54.84 allowing for the key time for these viruses
0:12:56.67 –> 0:12:59.74 to be acquired by the mosquitoes,
0:12:59.74 –> 0:13:03.38 develop within the mosquito gut and then be transmitted
0:13:03.38 –> 0:13:05.593 to a susceptible individual.
0:13:07.24 –> 0:13:10.31 Although these factors such as temperature
0:13:10.31 –> 0:13:12.29 really aren’t the only factor that needs
0:13:12.29 –> 0:13:13.51 to be taken into account
when predicting the risk of disease, since factors such as human behavior and the amount of time spent outdoors, housing development and whether or not there are screens on the windows and other actions that contribute to the prevention of mosquito breeding sites, can all play a really big role in the risk of vector-borne diseases across different climatic conditions and kind of across time. And another important factor is that while climate can affect the development rate of parasites and viruses within mosquito vectors, the extrinsic incubation period of malaria often tends to be shorter at higher temperatures, another important factor which is often not necessarily taken into account is that variations around mean temperatures can also play a very important role. So it was found using experimental system that diurnal temperature variations or the variation in temperature between night and day really can play an important role in modulating both the development time of mosquitoes, the EIP as well as the survival rate of mosquitoes at different temperatures where at lower temperatures, larger diurnal temperature variations tended to increase the survival of mosquitoes and decrease the development time whereas at higher temperatures.
that tend to be you know, typically more conducive to survival of mosquitoes, when you take into account the diurnal temperature variations, it can actually lead to lower survival than might be predicted in a higher developmental time. And so you need to not only take into account just mean temperatures, but also often these variations in temperature around the mean. And then finally there are other both direct as well as indirect impacts of climate on infectious diseases and these include impacts of climate on the geographic range, population dynamics and behavior of zoonotic reservoir species as well as effects on human behavior such as seasonal migration that may be linked to agriculture and pastoralism that lead to kind of movement and aggregation of individuals in different areas at different times of the year. Finally, climatic events can cause displacement particularly of climate refugees in different areas which can make them particularly vulnerable to various infectious diseases and then finally, climate can have important impacts on host susceptibility as we talked about earlier, but there are both climate related as well as unrelated causes of seasonal variation in host susceptibility for example linked to the length of day.
and how exposure to solar radiation can impact vitamin D metabolism and such which plays an important role.

Exposure to solar radiation can be an important co-factor in the immune system and so one of the ways in which we can identify and quantify the mechanistic impact of climate on infectious diseases is through experimentation. For example, this is what was done with the guinea pig experiment that I talked about earlier where they looked at the effects of temperature and relative humidity on flu transmission.

I also am showing here results of another experiment in which they looked at the effect of temperature on snail mortality which is an important host of Schistosomiasis and showed that the mortality rate of snails tended to be lowest when mean water temperatures were around 20 degrees Celsius in this experimental system, suggesting kind of the ideal climatic conditions for kind of greater survival of these snails which play an important role in the transmission cycle for Schistosomiasis. And another way to really identify and to quantify some of these mechanistic links between climate and infectious diseases is to use model-based approaches, but in this way, it’s really important to make sure that you’re following supposed links within the causal pathway.
And so for example, we have been working with researchers in Nepal based on some of the pilot funding that we received from the Climate Change and Health Initiative to try to quantify the impacts of rainfall on typhoid transmission within the setting and to estimate the incidence of typhoid fever that might be attributable to rainfall in this setting. You can see on the plot on the bottom left here that typhoid fever incidence tends to peak kind of during the rainy season within this particular setting, but there are also some of these important variations in seasonal incidents that are hard to explain just based on rainfall patterns alone. However, studies from our collaborators have shown that levels of bacterial DNA present in water sources such as these wells that are often used by individuals to obtain water tend to, the levels of the bacterial DNA tend to be slightly higher following increases in rainfall or these big rainfall events. However when it comes to trying to quantify these associations between infectious disease incident and climate, there’s a variety of different data types that are often used in order to do this and one of the most common types of data that is typically used to look at relationships between climate variables and infectious disease variables.
is data on seasonality,
since often infectious diseases do exhibit these seasonal variations in incidents,
however there often are a lot of things that vary seasonally and in these types of analysis,
you need to be really careful to avoid confounding just because things are correlated with each other,
it doesn’t necessarily mean that one thing is a cause of another.
So for example, this is data on murders by steam,
hot vapors and other hot objects in the US plotted in black here and the average age of the Miss America winner plotted in red which oddly enough are very highly correlated with one another, with a correlation coefficient of 87%,
but I have a very hard time seeing how these, one thing could possibly be causally linked to another and so just because there are correlations present,
doesn’t necessarily mean that any of these correlations are necessarily causal.
And so it’s best if you can also link when looking at these seasonal relationships,
link the between year variations in incidents and deviations from normal climatic conditions to anomalies in the infectious disease incidents.
So for example, one of the things that we found in modeling the relationship between absolute humidity and influenza, seasonal influenza in the United States was that there tended to be these dips
in the absolute humidity relative
to kind of normal absolute humidity expected for that time of year and these dips often preceded the onset of the seasonal influenza epidemic in different US states by around seven to 14 days, and this sort of provides good evidence that there’s actually sort of this relationship where in addition to the experimental evidence, that absolute humidity is really kind of pre or precipitating the influence epidemic each year. And another type of data that’s often used and can potentially be a very strong way to link infectious disease incidents to climatic variables is to take advantage of multi-annual variations in both climate and infectious disease incidents, and one of the best known multi-annual cycles when it comes to climate is the El Nino phenomenon or the El Nino-Southern Oscillation which has been linked to variation in cholera cases happening in Bangladesh since the 1990s where you typically tended to see higher peaks of cholera epidemics coinciding with years in which there were greater sea surface temperature anomalies happening and these are happening with a frequency of around five to six years. However, while these generally provide stronger evidence in favor of a climate disease link,
since fewer things will vary at these kind of multi-annual frequencies, you still need to be careful when it comes to drawing these causal links between variations in climate and these variations in infectious disease incidents, since infectious diseases can often exhibit multi-annual cycles that are driven instead by the internal dynamics of immunity and susceptibility which I’m gonna touch on in a couple slides. And then finally spatial data can often be useful as well. In particular, the geographic range limits of a particular pathogen may help to tell you something about how climate affects its transmission. For example, this is a distribution map for the Ixodes scapularis tick which is the main vector of Lyme disease within the United States, showing that the sort of suitable ranges in which we would expect to see the tick species overlap with the observed distribution of the tick. Sorry, I’m accidentally going forward too quickly. And the one caveat with doing this though is that you need to be careful not to over interpret some of the data, since there may also be other factors involved including behavioral factors or it just may be possible that the pathogen hasn’t been introduced yet, for example, to a region where you would predict the climate
0:25:08.88 –> 0:25:12.66 to be suitable, but you don’t see presence
0:25:12.66 –> 0:25:14.543 of the particular pathogen there yet.
0:25:16.664 –> 0:25:21.664 And so when it comes to methods for drawing these
links
0:25:23.97 –> 0:25:28.97 between climate and infectious diseases,
0:25:29.41 –> 0:25:32.01 one of the ways that this has traditionally been done
0:25:32.01 –> 0:25:37.01 for other diseases not necessarily infectious diseases is
0:25:37.14 –> 0:25:40.52 through the use of time series models.
0:25:40.52 –> 0:25:43.51 So for example, generalized linear models
0:25:43.51 –> 0:25:46.35 such as this Poisson type regression model
0:25:46.35 –> 0:25:50.1 which models the log of the number of cases at time t
0:25:50.1 –> 0:25:52.25 as a function of the baseline incidence
0:25:52.25 –> 0:25:55.07 as well as a variety of different predictors,
0:25:55.07 –> 0:25:57.973 some of which may be climatic variables,
0:25:59.18 –> 0:26:03.54 but the main limitations of this approach is
0:26:03.54 –> 0:26:08.54 that it really assumes that you have independent
outcomes
0:26:08.6 –> 0:26:11.968 or in other words that the number of cases
0:26:11.968 –> 0:26:16.968 of the observed disease at time t is independent
0:26:17.2 –> 0:26:20.39 of the number of observed cases of disease
0:26:20.39 –> 0:26:23.88 at time t minus one and we know for infectious diseases
0:26:23.88 –> 0:26:25.77 that that’s just not true
0:26:25.77 –> 0:26:28.01 because of the transmission process
0:26:28.01 –> 0:26:31.82 and because often the cases at time t minus one
0:26:31.82 –> 0:26:36.353 are actually causing the cases happening at time t.
0:26:39.19 –> 0:26:41.85 And so models that do not account
0:26:41.85 –> 0:26:45.05 for these underlying variations in susceptibility
0:26:45.05 –> 0:26:48.37 of the population may fail to identify
0:26:48.37 –> 0:26:51.37 some important climate disease relationships.
0:26:51.37 –> 0:26:54.81 And this is just an example plotted here
0:26:54.81 –> 0:26:57.9 in which we model the potential relationship
0:26:57.9 –> 0:27:00.55 between a climactic variable,
In this case, we're gonna say precipitation and we're gonna say that there's this link between precipitation and climate, which we're modeling. We're gonna say that there's this link between precipitation and climate, which we're modeling, and we're gonna say that there's this link between precipitation and climate, which we're modeling. The transmission rate, \( \beta_t \), which we're modeling up on the top here, shows a biannual pattern of precipitation with two peaks a year. These peaks cause the transmission rate to happen at different times of the year, resulting in a large peak and a minor peak in the transmission rate each year.

If you model the incidence of a disease with a low \( r_0 \) or a lower transmission rate within the population, you see a similar predicted pattern of cases happening through time, where a peak in incidence follows the peak in precipitation or the peak in the transmission rate. If you take the same model and simulate it with a higher \( r_0 \) or a higher baseline transmission rate, you can get into these patterns. However, if you take the same model and simulate it with a lower \( r_0 \) or a lower transmission rate within the population, you will see a similar pattern of cases happening through time, where a peak in incidence follows the peak in precipitation or the peak in the transmission rate.
in which you see a very large epidemic happening, kind of the first time climate is sort of favorable to transmission, but then you've kind of overshot the susceptible population such that you don't have enough susceptible people around to cause an epidemic the next time climate is favorable to transmission happening and so there's no epidemic happening even though conditions are favorable this year and that you have to wait another year until climate conditions are both favorable as well as there's enough susceptible individuals around to have another epidemic occurring. And you can see that in this instance it would be very much more difficult to link your climate driver to the observed incidence of cases happening in the population as modeled here. And so as a result, there's a variety of different methods that can be used and are often used when specifically looking at the climate disease relationship for infectious diseases. and these vary from the traditional statistical methods including your generalized linear models through to models that do account for autocorrelation within data such as ARIMA models and time-varying coefficient models to methods such as time series decomposition and wavelets,
semi-mechanistic models known as TSIR-type models, down through the fully transdynamic models such as transmission dynamic models or individual based models. And similarly, there are spatial methods that can be applied as well varying from static risk maps through to dynamic risk maps and I’m just gonna touch on a few of these different examples, kind of using some of our own work to illustrate it. So for example, one of the things we’re working on currently is to try and understand links between climate and diarrhea incidents across different districts within Ghana as modeled or as shown in this map here on the right where we have the observed incidents per 10,000 individuals on the left and the model predicted incidents on right where we’re using a simple time series Poisson regression model where the log number of cases at time t is a function of the baseline incidence plus a function of the mean temperature in the given district at time t, the diurnal temperature variation, a model for wetness prevalence or the presence of wetness which incorporates precipitation data as well as often using harmonic terms for annual and possibly biannual variations in incidents.
where you can see the model provides a reasonably good fit to diarrhea incidents in Navrongo which is a city, a small city in the northern part of Ghana as well as Accra which is the main capital in the southern part of Ghana, but one of the interesting things when you look at actual correlations and the coefficients within these models is that you see opposite relationships between your climatic variables including the mean temperature in this panel, the second panel as well as the wetness prevalence or a measure of precipitation in the fourth panel here in the northern part of the country versus the southern part of the country, where here we’re plotting the Pearson correlation coefficient across these different areas and showing that you see negative associations between temperature and diarrhea incidence in the north and more negative associations found in the south. And so it’s I think gonna be difficult to really kind of tease apart what are the main drivers of these differences and what are the other factors that are involved that really explain sort of the differences in climate, in the role that climatic factors play in diarrhea
in this setting, and one of the ways that we can do this is using spatiotemporal models and this is a previous study in which we use spatiotemporal hierarchical Bayesian models to look at diarrhea and the associations between climate and diarrhea incidents in Afghanistan. Using these methods, we're really kind of able to show that higher diarrhea incidents which tended to be concentrated around the population centers in the northeast as well as in some of the other kind of northern outlying regions is really associated with both positively with aridity and fluctuations in mean daily temperature as well as negatively with changes in average annual temperature. Where colder parts of the country tended to have a higher incidence than might be expected. And another way in which we can use different or another approach rather to using models to tease apart these climate disease relationships is to use what's called a TSIR type model which is a semi-mechanistic model which estimates the susceptible population through time at each time point as well as the affected population at each time and incorporates it into a regression type of framework such as this where we can kind of model out the transmission rate through time.
and make it a function of different climatic variables and this is an approach that we used along with colleagues from Princeton to examine the relationships between humidity, rainfall and cases of Respiratory syncytial virus or RSV across different parts of the US and Mexico under both current and future climates. And using this approach, we were able to show that the transmission rates of RSV which is indicated by the various colors here, tended to depend both on the level of humidity within the population where it tended to be higher transmission happening at lower specific humidity as well as on the level of precipitation within the population where particularly at kind of middle precipitation played a larger role in modulating transmission of RSV and this really helped to explain some of the very different patterns that we see in sort of the seasonality of RSV across different parts of the US where we see this sort of biennial every other year pattern of large followed by small epidemics often happening in Upper Midwestern states such as Minnesota, kind of regular annual seasonal outbreaks happening in the winter in states such as New York and Connecticut, earlier epidemics with kind of more year-round transmissioning happening
0:36:18 –> 0:36:19.96 in Florida and these sort of biannual
0:36:19.96 –> 0:36:23.67 two peaks a year happening in parts of Mexico.
0:36:23.67 –> 0:36:27.1 And by linking this kind of specific relationship
0:36:27.1 –> 0:36:31.35 between the transmission rates to these climatic factors,
0:36:31.35 –> 0:36:33.16 we’re able to make projections
0:36:33.16 –> 0:36:36.938 about the impacts of climate on future disease incidents
0:36:36.938 –> 0:36:41.4 which is shown on the plots over here
0:36:41.4 –> 0:36:45.12 on the map on the right in which we predict
0:36:45.12 –> 0:36:50.12 that overall transmission rates of RSV will be lower
0:36:51.51 –> 0:36:54.21 in the future in the Upper Midwest
0:36:54.21 –> 0:36:56.13 and Northeastern United States,
0:36:56.13 –> 0:36:58.44 but potentially higher seasonal differences
0:36:58.44 –> 0:37:01.87 in transmission in the west as well as the south,
0:37:01.87 –> 0:37:03.34 although there’s a lot of uncertainty
0:37:03.34 –> 0:37:05.35 in some of these model predictions,
0:37:05.35 –> 0:37:07.81 partly related to uncertainty
0:37:07.81 –> 0:37:10.133 in rainfall predictions going forward.
0:37:11.17 –> 0:37:13.48 And we’ve also used this approach to look
0:37:13.48 –> 0:37:17.701 at the relationship between rainfall
0:37:17.701 –> 0:37:22.65 and typhoid fever using historical data from the US
0:37:22.65 –> 0:37:25.04 where we had data from 19 cities
0:37:25.04 –> 0:37:27.37 across different parts of the US
0:37:27.37 –> 0:37:29.92 and found this really kind of interesting differences
0:37:29.92 –> 0:37:31.71 in seasonal patterns between cities where
0:37:31.71 –> 0:37:33.55 for example in New York,
0:37:33.55 –> 0:37:36.98 we saw very strongly seasonal epidemics peaking
0:37:36.98 –> 0:37:39.29 of typhoid fever before,
0:37:39.29 –> 0:37:43.6 this is data from like the late 1880s, early 1900s
0:37:43.6 –> 0:37:46.66 where you saw these typhoid fever epidemics peaking
0:37:46.66 –> 0:37:49.54 every summer, early fall
0:37:49.54 –> 0:37:51.89 whereas in a city like Philadelphia
which was right next door,
there’s very little kind of seasonal variation in climate
and one of the things that we were able
to identify oh sorry, seasonal variation
in the typhoid transmission rate,
and by teasing apart these variations
in the transmission rate,
one of the things that we identified was that the amount
of seasonal variation in the transmission rate
really tended to vary depending on the primary water source
for the city where cities that relied on reservoirs,
often reservoirs that were outside of the city
such as the New York reservoir which is located in upstate New York
as well as outside of Boston and in Baltimore,
tended to exhibit these kind of stronger overall seasonal variations summarized in the plot
on the bottom right here,
compared to cities that relied on data
or a water from nearby rivers or rivers that ran through the city such as in Philadelphia
cities that drove their water from the great lakes
which actually had the lowest seasonal variation in transmission rates and so taking into part,
taking into account kind of other factors
such as water sources is really important
in understanding some of these relationships.
And overall, this relationship between kind of temperature and you know,
why transmission rates tend to peak
in the summer months was consistent with results of a systematic review that we conducted looking at associations between climate and typhoid fever incidents that generally showed that temperature on the bottom here was a stronger correlate of typhoid fever incidence at lags of zero to two months across different latitudes and studies conducted across different latitudes compared to rainfall which is really kind of only associated often in studies conducted in the monsoon belts where, and you often also saw potentially negative associations between rainfall and typhoid fever incidents in places such as the Middle East.

And then finally, one of the last ways in which we can estimate the climate disease relationship is to incorporate climate models into fully mechanistic dynamic models which explicitly account for the susceptible, infected and recovered populations through time. And so for example the way this works is to assume in your population that all individuals are born susceptible to a particular disease and that they become infected at a rate which we're gonna call lambda and remain infectious for a period of time after which they recover and have some immunity to future infections of the disease and the important part about these models is that this lambda parameter or the rate...
from going to susceptible to infected depends on the current prevalence of infectious individuals within your population through time, such that the lambda at time t is gonna be a function of our transmission rate at time t, times the number of currently susceptible individuals and times the number of currently infectious individuals within our population. And so our incidence of new cases is dependent not just on the transmission rate or climatic variables which may affect the transmission rate, but also on the current prevalence of the infection within the population. And then within these models, we can decompose this transmission rate or this beta parameter at time t to be a function of various other factors and often the way we model it is as a function of a baseline transmission rate plus some seasonal variation which we may not understand kind of all the factors leading into the seasonal variation, but using a harmonic term and then can incorporate our various climatic predictors as coefficients in this equation for our beta t parameter. And this is something that we’ve done to look at the impacts of climate on rotavirus diarrhea in particular in Bangladesh where we’re using this slightly more complicated model specific to our understanding of immunity and natural history of rotavirus infections.
which is depicted on the left here and modeling our incidence rate at time \( t \) as a function, not only of sort of the baseline incidence and these harmonic terms accounting for kind of annual and bi-annual potential differences but also climatic terms including the diurnal temperature variation which is plotted in the middle here showing kind of a larger diurnal temperature variation happening in the kind of winter months or early parts of the year and less diurnal temperature variation in the middle of the year, as well as the wetness presence which tends to be more higher in the middle parts of the year coinciding with the monsoon season and you can see the rotavirus incidence in this particular setting, if you kind of average it over time shows these sort of bi-annual peaks where you have a larger peak happening kind of in the cooler, dry season and then a smaller secondary peak happening and we can use kind of this relationship to try and tease apart some of those relationships and how they’re associated with climate factors as well as other factors within the population and you can see that we have plotted here the incidence of rotavirus diarrhea from the 1990s
0:43:55.45 –> 0:43:57.83 to early 2000s as well as incidents
0:43:57.83 –> 0:44:02.51 over kind of a later time period from 2003 to 2013
0:44:02.51 –> 0:44:05.85 where if we fit models to the early time period
0:44:05.85 –> 0:44:08.6 and use it to predict the later time period
0:44:08.6 –> 0:44:11.9 which is shown in blue here,
0:44:11.9 –> 0:44:15.07 we can generally kind of capture some of these patterns
0:44:15.07 –> 0:44:18.7 in which we observe a stronger kind of bi-annual pattern
0:44:18.7 –> 0:44:22.32 or two peaks a year happening across the 1990s
0:44:22.32 –> 0:44:26.34 and early 2000s, but a much more kind of annual pattern
0:44:26.34 –> 0:44:29.473 that emerges in the later 2000s,
0:44:31.417 –> 0:44:35.09 in 2000 through 2013 where you’re starting to see
0:44:35.09 –> 0:44:38.57 much greater predominance of this annual
0:44:38.57 –> 0:44:42.44 kind of winter dry season peak and it doesn’t seem
0:44:42.44 –> 0:44:44.39 that this is necessarily related to variation
0:44:44.39 –> 0:44:47.62 in climate over time, but really is more driven
0:44:47.62 –> 0:44:50.15 by actually a decline in the birth rate
0:44:50.15 –> 0:44:54.438 within Bangladesh and particularly within Dhaka
0:44:54.438 –> 0:44:57.36 which is sort of interacting
0:44:57.36 –> 0:44:59.76 with the different climatic factors
0:44:59.76 –> 0:45:03.043 to change the sort of the predominant way
0:45:04.79 –> 0:45:08.37 in which the climate influences transmission of rotavirus
0:45:08.37 –> 0:45:11.36 within the setting, where kind of even modeling
0:45:11.36 –> 0:45:14.16 kind of same relationships between climate
0:45:14.16 –> 0:45:16.66 and rotavirus transmission over time,
0:45:16.66 –> 0:45:21.66 we can capture this shift from kind of more biannual peaks
0:45:22.2 –> 0:45:27.2 to greater predominance of annual peaks over time
0:45:30.72 –> 0:45:33.07 and so finally, I just wanna talk a little bit
0:45:33.07 –> 0:45:36.617 about how we can kind of pull everything together
0:45:36.617 –> 0:45:40.34 and how we can make these predictions
0:45:40.34 –> 0:45:44.26 around the impacts of climate change on infectious diseases.
And again, the way that we do this is to really combine our climate model projections with a good understanding of the incidence of disease given climate, but then there’s still a number of big challenges. Often the climate models have poor resolution and wide uncertainty which needs to be propagated throughout the relationships of predictions going forward. Infectious diseases may often vary based not only on mean climate, but can also show important variability on shorter spatial and temporal scales. Such that things like diurnal temperature variation or changes in climate kind of through the day can have important impacts often on climate. I would say non-linear, when it comes to infectious diseases and climate models are often bad at predicting some of the extremes and extremes in temperature and rainfall for example which have been shown to be kind of important drivers of a transmission of infectious diseases. And then finally, another important caveat is that the impacts of climate change may be quite small relative to the impacts of human interventions and demographic changes as we saw with the rotavirus example, but as we also see with models of projections.
0:47:17.35 –> 0:47:19.81 around malaria risk over time
0:47:19.81 –> 0:47:21.76 where if you were to just simply look back
0:47:21.76 –> 0:47:25.52 at the malaria risk map from the 1900s
0:47:25.52 –> 0:47:30.37 and compare it to the malaria risk map from 2007,
0:47:30.37 –> 0:47:33.67 you’ll see that the overall regions
0:47:33.67 –> 0:47:37.59 in which you see malaria has contracted significantly
0:47:37.59 –> 0:47:39.96 where we no longer see malaria happening
0:47:39.96 –> 0:47:42.35 in parts of the US for example,
0:47:42.35 –> 0:47:45.77 but really being more confined to parts of Africa
0:47:45.77 –> 0:47:48.58 and Asia and other places.
0:47:48.58 –> 0:47:53.58 But at the same time, the climate has actually been warming
0:47:54.64 –> 0:47:57.78 and this would suggest that you would see
0:47:57.78 –> 0:48:00.13 more favorable conditions for malaria climate
0:48:00.13 –> 0:48:04.54 if you’d only take into account climate over time
0:48:04.54 –> 0:48:07.16 and so while what’s actually been observed is
0:48:07.16 –> 0:48:09.51 mostly been driven by human interventions
0:48:13.39 –> 0:48:15.24 If you don’t take into account those changes,
0:48:15.24 –> 0:48:17.49 you would totally misunderstand
0:48:18.53 –> 0:48:20.26 or misrepresent the climate associations
0:48:20.26 –> 0:48:21.92 that we know are there.
0:48:23.42 –> 0:48:25.14 And so in terms of the way forward,
0:48:25.14 –> 0:48:29.27 what really needs to be done is to take on
0:48:29.27 –> 0:48:31.35 some of these climate disease relationships
0:48:31.35 –> 0:48:34.91 in which we have experimental systems set up
0:48:34.91 –> 0:48:38.05 and we have a better understanding of the relationship
0:48:38.05 –> 0:48:41.86 between climate and how it impacts on infectious diseases.
0:48:41.86 –> 0:48:43.9 Furthermore, some of the climate change productions are
0:48:43.9 –> 0:48:47.01 gonna be more reliable and so those infectious diseases
0:48:47.01 –> 0:48:50.25 that rely on or have been shown to vary
based on climate variables which are more predictable, I think, are gonna be kind of more amenable to making predictions around the impacts of climate change.

But overall, there’s really an important need for interdisciplinary work between climate scientists, lab scientists and microbiologists who can help test some of these mechanisms, and infectious disease modelers who can quantify the relationships between climate and infectious diseases to really move forward some of the field when it comes to trying to make impacts on infectious diseases, and in doing so we really need to take into account factors such as human adaptation and the impacts that climate may have on human behavior and population distribution since these are often kind of greater drivers of infectious disease incidents than factors affecting pathogen survival for example. And so really there’s this need to move beyond just the simple climate disease correlations that other I think previous attempts to predict the impacts of climate change have relied upon. And so finally I just want to quickly acknowledge some of the people who I’ve worked with on these various projects including collaborators and lab members here at Yale.
as well as collaborators elsewhere and funding from the Yale Climate Change and Health Initiative as well as NIH, the Gates Foundation and Welcome Trust and James McDonald. So I’d be happy to take any questions. Thank you.

Very wonderful presentation. I think it covers all the aspects when we talk about conscientious infectious disease from modeling the climate disease relationship to how to better project the future impacts. So we do have a lot of questions from the students. So because we only have very limited time, so I will summarize two questions from the students and if we have more time, then maybe our audience can speak for their questions. The first question is kind of follow up your later part talking about the inferences of the non-climatic drivers. You’re showing that actually human intervention can have much larger impacts. So students are wondering how do you consider this in projecting the future climate change impacts? Yeah I mean I think that’s often the difficulty when it comes to making predictions about the future is understanding how you know, you can make projections kind of assuming all other things remain the same and climate’s the only thing that’s changing.
but the reality is that climate’s never gonna be the only thing that changes over time
and so you have to have either some other model for how human behavior may change over time
and human development or things like that may change over time and that may just be sort of trying to make simple extrapolations or maybe based on sort of more sophisticated kind of sociological or sociopolitical models of say, development or things that other factors that may affect like interact the risk of disease. So for example when it comes to malaria, obviously sort of some of the developmental factors and industrialization that happened and things like that played a huge role in kind of why we no longer see malaria in parts of the world where it was previously, but yeah, I mean I think you need a separate model to really account for how we see those things changing over time separate from or potentially related to climate. Wonderful, so while Gina is answering questions, you can type your questions in the chat box if you do have any questions, you can raise your hand and then we can ask you the questions. So I have actually a couple questions from the students given we are under you know the COVID 19 pandemic. We are very interested in like
0:53:49.248 –> 0:53:51.88 what’s your answer to the climate inference
0:53:51.88 –> 0:53:54.05 on the transmission of COVID 19
0:53:55.373 –> 0:53:57.31 and what are the potential challenges
0:53:57.31 –> 0:54:02.31 in using the approaches that you are talking about
today
0:54:02.61 –> 0:54:05.797 to study the relationship between COVID 19
0:54:05.797 –> 0:54:07.403 and all the climate drivers.
0:54:08.654 –> 0:54:09.68 - Yeah, I mean I think that that’s definitely something
0:54:09.68 –> 0:54:11.43 that some people have tried
0:54:11.43 –> 0:54:14.24 to kind of tease apart using data
0:54:14.24 –> 0:54:16.39 I think mostly from kind of different locations
0:54:16.39 –> 0:54:18.55 and trying to understand kind of how
0:54:19.69 –> 0:54:23.89 perhaps how quickly the epidemic has taken off
0:54:23.89 –> 0:54:27.13 in different locations could potentially be explained
0:54:27.13 –> 0:54:31.638 by some of the differences in climate possibly
0:54:31.638 –> 0:54:35.17 and so I mean I think that that is potentially one
approach
0:54:35.17 –> 0:54:39.18 to take, but really doesn’t factor in
0:54:39.18 –> 0:54:43.503 all of the other things that may potentially affect
0:54:47.64 –> 0:54:49.93 whether it’s climate that’s driving
0:54:49.93 –> 0:54:52.96 how quickly the epidemic takes off across different places
0:54:52.96 –> 0:54:54.52 or whether it’s other factors,
0:54:54.52 –> 0:54:59.52 for example just kind of the extent of social distancing,
0:55:00.06 –> 0:55:01.41 the extent of other interventions
0:55:01.41 –> 0:55:04.1 and how all of those things have played a role
0:55:04.1 –> 0:55:06.59 or just chance in terms of when the virus was introduced
0:55:06.59 –> 0:55:08.38 in different places in determining
0:55:08.38 –> 0:55:10 kind of how quickly the epidemic has occurred
0:55:13.69 –> 0:55:15.25 Another approach that has been taken is
0:55:15.25 –> 0:55:20.13 to look at our understanding of other Coronaviruses
0:55:20.13 –> 0:55:22.78 within the human population
and there are kind of at least two other human Coronavirus species that cause cold like illness every year that circulate within the US and we know that those other Coronaviruses tend to peak in the fall, early winter time period and likely the reasons behind why they peak in the fall and winter time period is really related to climate conditions favoring transmission, be it from what I talked about earlier in terms of host defenses and host defenses being slightly weakened at that time or potentially direct relationships with virus survival or potentially you know, seasonal differences in behavior such as aggregation of kids in schools in the fall period, but I think trying to bring that to bear and directly predicting the incidence of the SARS-CoV-2 virus at this time is gonna be very difficult because I think the biggest factor kind of underlying transmission right now is differences, I mean we have a virus in which everybody is susceptible and so it’s gonna be able to spread efficiently kind of regardless of climate conditions across different settings and so I think that climate is gonna play kind of less of a role now in terms of determining when these seasonal peaks happen compared to just all the other factors in terms of social distancing and other interventions
and when some of these things are relaxed, when people become complacent and stop you know taking all the precautions during the summer months for example, more so than the role that climate is gonna play right now. So I think it’s really a situation we’re gonna have to wait and see kind of what are the major climate drivers of the SARS-CoV-2 virus and how much is it really modulating transmission. Thanks, that’s very insightful. So I think we have some time from the audience to ask a questions. So there’s one question was wondering, if the terms adjusted for the annual and the bi-annual pattern only account for seasonality, how about the long-term change? And a further question is if one disease shows a bioannual pattern, why still use the annual term in the model? Okay, so that’s a, I think very-- if it’s specifically around kind of rotavirus in Bangladesh, certainly I think that there is potentially
also long-term trends happening in transmission rates over time and often we do need to account for these potential like linear or long-term trends happening in baseline incidents over time which may be important as well and it’s often something that we do kind of explore incorporating into the models and is potentially able to explain some of these you know, unusual shifts that we might see for example from the biannual to more annual epidemics happening in Bangladesh in conjunction with the sort of the decrease in birth rate, we’re probably also seeing a decrease in potentially transmission rates over time in that setting. But in the question around kind of why do you incorporate both biannual and annual terms in a model. The reason for that is often because if you’re only incorporating a sort of biannual harmonic term that assumes inherently that the size of the two peaks is the same whereas when you add an annual harmonic, it allows for two peaks of varying size happening throughout the year and so it can sort of basically lead to a larger peak and a smaller peak throughout the year whereas if you only have a biannual term, you can only have those two peaks
0:59:45.72 –> 0:59:47.67 by definition have to be the same size.
0:59:48.77 –> 0:59:50.2 - Great, thank you.
0:59:50.2 –> 0:59:55.2 So I think we have reached the end of this seminar
0:59:55.76 –> 0:59:59.73 and thank you Gina for this wonderful presentation
0:59:59.73 –> 1:00:02.02 and thank you all for coming.
1:00:02.02 –> 1:00:05.97 Our recording will be available later
1:00:05.97 –> 1:00:10.91 and we will have our next seminar in November.
1:00:10.91 –> 1:00:15.14 So looking forward to see you soon, bye.
1:00:15.14 –> 1:00:16.097 - Great, thank you.