JUDY HOYT: Go ahead and get started with today's class. I've got a couple of announcements. There's one handout for today, which is in the back. Hopefully you all have a copy of that. And I've brought with me the clipboard with the project signup sheet. So I'd like you to put down your name on one of the lines here. And the most important thing today is whether you want to give a written or an oral. Written would be at 20-page written report. The presentation would be a 15 to 20 minute presentation to the class. And all of that is described in the handout I gave to you about a week ago, which hopefully you have. If you don't have a copy of it, it's on the web or it's here on this clipboard.

So I'd like to know today or Thursday at the latest whether you want to do a written report or an oral report. Even if you don't know your topic. This will help me in scheduling your oral presentations. I have to figure out when at the end of the term, which class periods I'm going to dedicate to oral presentations. And then if you know your topic, that'd be great. Go ahead and write that in. The last column on this signup sheet says whether it's approved or not. And I'll look through this over the next few weeks. And as time goes on, I'll look at your topics. If I have a question. I'll talk to you about it. And if I don't, I'll just go ahead and approve it. So when it's checked off, then your topic is approved.

So if you can fill that information out, the sooner the better, on the written versus oral, and as well as on your topic. If you have any questions about topics, feel free to ask me. So I'm going to start passing that around.

One of the things, homework. Homework number 3 is being graded. Hopefully we'll have that back to you soon. Homework number 4 went out last time. Hopefully you got a copy of that. If you didn't, it's all posted on the web. Everything is posted on the website. This is where we are on the class schedule. What I'm looking at here is this Excel file, which just sort of shows that we're at lecture 14. We're going to talk today about transient enhanced diffusion from Chapter 8. And you've got a couple of more lectures. The next homework due date is homework number 4, which is due a week from today, election day, November 2.

So let's go ahead and start with today's lecture. The notes are handout number 24, and this is the third lecture on chapter number eight. Chapter 8, hopefully you've been here reading through. Chapter 8 at this point, chapter 8 is all about ion implantation. But this lecture and the next lecture are about transit enhanced diffusion.

So let me just remind you what we talked about last time. We talked about the physics of nuclear stopping and electronic stopping, or electronic energy loss processes. We said that if you have a heavy ion-- say if you're implanting antimony-- and heavy and lighter always relative to the substrate. So antimony and arsenic, for example, are very heavy compared to silicon, then nuclear stopping tends to dominate over their entire path as they go into the silicon substrate and as they come to rest. Nuclear stopping is dominant.

If you have a heavy or a light ion, then nuclear stop stopping dominates towards the end of path, the end of range. So when the energy gets low enough. So as we saw last time, the nuclear stopping power goes down as energy increases, and the electronic stopping power goes up. So only at very low energies do you have a lot of nuclear scattering for a light ion. It's the nuclear stopping, not the electronic. It's the nuclear stopping, the collisions, that contribute to crystal damage clearly.

So the ions are coming in, and in a nuclear event, you have a billiard ball collision, and you're knocking the silicon off its lattice site. And you're creating, in that process, silicon interstitials and vacancies. And these results in something which we call the collision cascade. We talked a little bit to how people have calculated damage profiles. So they calculate the amount of energy deposited into these nuclear stopping processes as a function of depth. And the peak damage-- so the most amount of damage you do-- the profile tends to peak near the projected range of the primary ion. Maybe just slightly shorter or shallower than the projected range RP.

So let's say you're doing an ion implant of arsenic, and you can easily calculate, using one of the theories, the arsenic profile. You can use the rain statistics for the arsenic, whatever. You know where the RP of the arsenic is, and you can figure out where the most of the damage is going to be done. It'll be just about 90% or so. It will be the peak of that damage profile.

For a heavy ion like arsenic or antimony and silicon, the damage is more stable. So it doesn't tend to anneal out at room temperature. It's relatively easy to form an amorphous layer with a heavy ion at room temperature. And this can then be regrown. We talked about a process called solid phase epitaxy to regrow that amorphous layer. And so this is a relatively efficient way to do dopant activation. I'll say a little bit more about dopant activation in this lecture.

And finally, we mentioned last time, we just started this topic, we said that the excess interstitials that are created by this implant, all these nuclear collisions, they can cluster into a particular type of defect called a 311 defect. Later, the 311s dissolve and they give off interstitials. And these interstitials then are what determine the kinetics of transient enhanced diffusion of boron and other dopants. So that's just a review of what we talked about last time.

What I want to cover this time, I have a few slides in this lecture on dopant activation just to go through that a little bit more carefully. And then most of the lecture is going to be talking about transient enhanced diffusion this, TED effect and how it's modeled. So let's go on to slide number 2.

Well I think I showed this last time, this cross-section PEM. We're going to talk about this case on slide number two, when the dose and the mass are high enough to amortize the silicon. And these are cross-section transmission electron micrograph images of a sample that has been ion implanted and amorphized. And then it is being regrown by putting it in a furnace at 525 degrees C. The initial implant was 200 keV of antimony. And look at the antimony dose. Quite high, 610 to the 15th per square centimeter. That's a high dose.

And so you can see-- this should say zero minutes, I'm not sure what happened to the zero-- but anyway, this means when you're close to starting up what you have is an amorphous layer from the surface down to some depth. And in fact here's the scale bar. This is 100 nanometers from here to here. So that's maybe 300 nanometers, 3,000 angstroms deep, something like that. It's all amorphized. The crystal structure is completely destroyed. You have an amorphous solid.

Now after 10 minutes, what has happened? Well the amorphous crystal interface has moved up. So we have regrowth. We have solid phase epitaxy, the layer by layer process. This epitaxy process is not that different from growth from the melt. The only difference here is we're talking about, we're getting a phase transition, or we're getting a transition from in the solid phase from amorphous to solid phase single crystal. And that's happening in a layer by layer fashion here.

After 10 minutes, I've grown up a little distance. 15 minutes, you can see the amorphous crystal interface has progressed. And in fact, this is a linear growth rate, you'll find. On the right hand side all the way on the right at 20 minutes, there's only a small amorphous layer remaining. And if we were to go longer, the amorphous layer would entirely regrow and you would have single crystal silicon completely in depth from the surface on down.

Then the only thing you're left with, you still have single crystal material, is down just below the original amorphous crystal interface, you are left with what's called these end of rage damage. It's a series of these dislocation loops and other sort of extended defects that don't anneal out. End of range damage, in general, unless she were to melt the sample or do something, it's something we most of the time, we end up having to live with to a certain extent. It's very hard to get rid of. But the amorphous layer is gone, and we have single crystal silicon.

So it's just a reminder when we're talking about amorphization and a process for restoring the crystal to its single crystal nature. So let's go on to slide 3. I wanted to just remind us about dopant activation. And this particularly applies to boron and silicon. It's much more difficult to anneal a silicon sample. That is, when I say difficult to anneal, really what we mean is it's difficult to activate, to electrically activate. So to get when-- what do we mean by electrically activate?

Well, for every ion I implant, I like to have it go into a substitutional place. And if it's a donor, I like to donate an electron so I get a free electron. If it's an acceptor, I'd like to get a free hole. That's the whole purpose of doing the ion implant. So if I implant a dose of 10 to the 15th of arsenic or boron, I'd like to get 10 to the 15th per square centimeter free electrons or free holes. That's what I mean by activating.

This activation is more difficult, ironically, when you've only partially damaged the silicon. Particularly intermediate doses are difficult. So let's just take the range of doses. I think we talked a little bit about this last time. If we have a very low dose, 10 to the 12th atoms per square centimeter, not much damage is done, and it's not too hard to anneal the sample, get the crystal structure restored to more of a perfect state and to activate all the dopants and substitutional sites.

A very high dose, you create amorphous layer like we just saw on the previous slide. And it's relatively easy to anneal out the amorphous layer by solid-phase epi. Solid-phase epi can take place at 500 or 600 degrees. So you don't have to go very hot. Typically you would go a little higher than that, maybe after this SPE regrowth step you might go to 800 or 900 to get a little better dopant activation. But that's not so hard.

The hard one is the last one listed here. These are the intermediate doses. And this is a dose that is high enough to do some damage, but it's not high enough to create complete amorphous layer. So you cannot have solid phase layer by layer regrowth. So it's right in the middle that you have a problem. And this leads to a lot of complex behavior where secondary defects, other types of defects besides just interstitials and vacancies, can form.

And particularly for boron, which is a light ion, because of the nature of the type of damage it does, this intermediate dose range is a real problem. And this complicated behavior can occur over a wide range of boron doses that are used in practice in CMOS fabrication. And it's because of the nature of the damage created by boron is different. It's a light ion. So it's not doing that much very effective nuclear damage, at least until you get towards its end of range.

So you'll hear people say, oh, boron is a lot harder to activate than arsenic. Indeed it is. It's harder to repair the damage from a boron implant in silicon than from an arsenic implant in silicon. What do people do about this? Well, there are a couple of approaches I think we mentioned last time. Boron is light. OK, that's a problem. In some sense it's doing damage, but not enough damage. So within the dose range that you want to implant it.

Sometimes what people do is they don't implant pure boron. They implant a molecule, BF2. So these two extra fluorines add quite a bit of extra mass to the species that your ion implanting. So it's not just boron 11, it's boron with two fluorine atoms attached. And this is the ion that you ionize and the ion implanter, and that you impart energy and you put into the silicon. So when the BF2 molecule hits the silicon, what happens?

Well, it dissociates, most likely. It has so much energy, and the boron goes along and the fluorine goes in. And they both impart energy though, they both can do some kind of nuclear damage. So we can kind of treat this, we can say that the energy is apportioned, a certain amount to the boron-- in ratio to its z number-- and a certain fraction of the energy goes to the fluorine. And having the extra fluorine in there is a way to amorphize the sample. So BF2, sometimes people use to try to improve the activation. It's also a way of getting, effectively, a lower energy implant. Because if you have a certain energy imparted to BF2, the boron only shares a certain fraction of that when it actually hits the sample.

The other thing people do is they do what's called pre-amorphization. Pre-what? That means prior to implanting the boron, they modify the sample with another species. Typically you would not want to use an anti-dopant, because then you're compensating the boron. So typically you use either silicon or germanium, which are not electrically active per se. Right? Silicon is the same as silicon, so it's not going to produce any anti-dopant effects. Germanium is also in column four, it turns out it does not produce a doping effect. That is, it does not add p or n type character to the substrate. And it's heavy, so it's easy to amorphize.

So you'll see people doing prior to a shallow implant, they may implant silicon and liquid nitrogen or germanium at room temperature, create an amorphous layer. Into that amorphous layer, implant the boron, and then do a relatively low temperature activation to try to activate the boron and get a little bit of a better annealing behavior.

Pre-amorphization also has another advantage if you want to think about it. Remember when we were talking about ion implantation a few lectures ago? We were talking about ion channeling, and the fact that ions can be knocked into these channels because you have a perfect crystal that can go a long ways before they stop. And this creates these long tails, it makes it very hard to get an abrupt, shallow junction. Well, if you amorphize the top 3,000 angstroms prior to putting the boron in, that eliminates the possibility of ion channeling in those top 3,000 angstroms. There is no crystal. It's not single crystal, it's completely random.

So pre-amorphization has two benefits. You can get a little better activation of the boron, and it eliminates ion channeling, so you can do shallower junctions. It costs more money, it takes time, it does create end of range damage, so there are tradeoffs in using pre-amorphization as a way to do boron implants. So that's mainly comments I wanted to make about dopant activation. One more comment before I leave slide number 3, because we probably have a homework problem coming up in homework 5 about activation.

When you're using Supreme, Supreme can model dopant activation in a very crude way. If you do an ion implant in Supreme-- you let's say you implant arsenic to a certain concentration or dose-- and then you anneal it at just about any temperature, the default assumption is that everything is activated almost immediately within a few microseconds or something. So Supreme doesn't really have any good kinetics, any time dependence of the activation. So you do a one minute anneal at 800, it assumes you're getting the same amount of activation as a 30-minute initial at 800. It doesn't have kinetics to activation built in.

But it can plot for you. It will plot the active carrier concentration. And it will clip it off at the solubility limit. So if you implant arsenic to a peak of 10 to the 21, let's say, a huge peak per cubic centimeter, and then you tell it, I want to anneal it at 1,000 degrees for ten seconds, fine. It'll give you the active arsenic. It'll take it up to at 1,000 degrees, whatever the solubility limit. Is say it's 3e20, and just clip off the profile and go down from there. So it imposes a very kindergarten-like, sort of elementary activation model. But it can still be useful if you're trying to figure out what your sheet resistance should be. But it doesn't have a lot of these kinetics built in.

People need to do that more empirically. Do an implant, do an anneal at various temperatures, and see what your activation ratio is. OK, so let's go on to slide 4. And this is, again, review from last time, but I think it's important to review this before we talk about transient enhanced diffusion.

Last time we talked about this model with a funny name called the plus 1 model of implant damage, or plus-one model for residual damage. It was published by Giles back in 1991. And the idea was this. Or what the model says is most of the recoiled silicon interstitials and vacancies recombine very rapidly in the first few milliseconds, or hundredths of a second, during the implant, or else just when you just put it into the anneal.

So you create a huge number of these. But most of the interstitials find a vacancy, and they find a place to go back to.

How about the remaining? It's only the net remaining that we care about. So it's the distribution of the remaining interstitials, or the recoils, shows a net-- tends to have a net excess of vacancies near the surface and a net excess of interstitials towards the bulk.

So in fact, if you plot on this plot-- this is a plot from Giles-- concentration versus step, this is the total number of interstitials up here, in the 10 to the 21 range, or high 10 to the 20s for this phosphorus implant. And the total vacancies, you can't even distinguish the two curves. They're practically-- they're really on top of each other on the log scale.

When you subtract them from each other, what you find-- so you subtract interstitials from the vacancies-- that the net vacancy concentration looks something like this. That's what's shown by this little profile here.

So near the surface, we have some extra vacancies of a concentration, in this example, of about 10 to the 17th. In the bulk, a little bit deeper, we have net interstitials. But their total numbers is orders of magnitude lower, because a lot of them have recombined.

And look at their order of magnitude. What is their height? Well, lo and behold, the height of these net interstitials, its concentration is very close to the concentration of the phosphorus implant-- very, very close-- so of order of magnitude.

So to first order, what Giles said, is that all of the original implant damage recombines and leaves behind only one excess interstitial for every dopant atom you ion implanted. So if I ion implanted, in this case, 10 to the 13th phosphorus, then he would say that the number of interstitials left behind is 10 to the 13th per square centimeter-- because every phosphorus atom eventually finds a home on a silicon lattice. And those 10 to the 13th silicon atoms must then be interstitials.

So that's the most elementary model, so to speak, for the amount of damage that's produced. And it's easy to do. You just take the dose, and that's the answer more or less.

Actually, if we want to be a little more sophisticated, people since that time have come up with what they call a plus-n model, where n is of order 1 but not exactly equal to 1. And this is Pelaz et al. from the MRS meeting in 1997-- came up with this sort of plus-n model.

What they were saying is that the multiplier on the dose for the original ion implant dose, the multiplier may not be exactly 1. It may be 2. It may be 3-- something of that order. So this plus 1 approximation, which says that the number of interstitials created exactly equals the dose, it's reasonably good, but it's not necessarily perfect. And it may be particularly not as good for a heavy ion-- a low energy, or a low dose, particularly for heavy ions.

The reason we don't think it's so great-- it's not as perfect-- is because there's a lot of recoils. If I implant arsenic, it's a very heavy ion. It comes in, it can impart a fair amount of energy to a silicon that's originally at rest. And then that creates more recoils. So you can have a large population of recoils for each ion relative to the ion population.

And so that means they can essentially do a little more damage. So a simple approximation using a plus-n factor, which is a function of the ion species, the energy, and the dose. And here's an example of a plot from that paper.

This is a plot of the n factor. So what do we mean by the n factor? That's the multiplier that you need to multiply the dose of the primary ion. And that will tell you the number, then the dose of excess interstitials.

And so let's say if you're a boron. So let's look at this solid line. And we're doing boron somewhere around 5 to 10 Kev. Well, the n factor is very close to 1. So if I implant 10 to the 13th per square centimeter, I get 10 to the 13th per square centimeter interstitials excess created.

Look at BF2, is a little heavier. And in that same range, the n factor is 1.5. So if I implant 1 times 10 to the 13th BF2 per square, then I would assume that the number of interstitials is 1.5 times 10 to the 13th in this model.

And look at arsenic. It's actually almost up to a factor of 3 to 3.5. So somewhere between 1 and 3, and particularly at low energies where there's a lot of nuclear stopping, the n factor tends to increase. At high energies, they all approach one. And particularly for heavy ions, the n factor can be greater.

Now the reason we're emphasizing this, you need to know the dose of interstitials. You need to understand that, because it's those excess interstitials that are going to be responsible for transient-m enhanced diffusion. So that's why people pay a lot of attention to these damage models.

OK, so that's the next topic. We go on to Slide Number 6 on TED. I think I showed this last time as a way of finishing up. This is kind of an anomalous plot. It's a concentration as a function of depth. These are boron profiles.

And what's anomalous about it is if you look at the blue profile here, it's a 10 second at 1,000 degrees at a very high temperature, as opposed to two minutes at 800. The 1,000-degree profile-- this is after being ion implanted-- the 1,000-degree profile is actually shallower-- less motion at a higher temperature.

So this is very much non Fickian. This is very difficult to explain from a simple single diffusivity, which is exponentially activated.

Because we know, if you look in your text for the intrinsic diffusion of boron, that at 1,000 degrees it's like two or three orders of magnitude higher. And yet here this cannot be normal regular intrinsic diffusion, because you're getting a lot less diffusion at 1,000.

And so this is because of TED. And it'll turn out that-- we'll talk about in the modeling in this lecture why it is that at low temperature, we actually can get higher amounts of transient-enhanced diffusion-- higher amounts of this damage-induced diffusion than at high temperatures.

People didn't see this for many years. This TED was first started to be observed in the mid '80s, and then by the early '90s it became pretty prevalent. And why not? Why didn't people see it?

Well, for years annealed devices were very large. They were large in lateral dimension. They were large in vertical dimension. So annyas took place at reasonably high temperature, and they're very long. And so the TED effect-- the damage effects were completely masked by normal diffusion.

Because TED, what does that mean? Transient. It only lasts for a limited period of time-- a short period. So if you put a wafer in a furnace at 1,000 degrees, or 800 for four or five hours-- a couple of hours-- you're not going to see it.

It's only when people started trying to get very little amount of [INAUDIBLE], and they cut back on the time-- 10 seconds at 1,000, or a few minutes-- so development of rapid thermal annealing techniques where the anomalous diffusion became obvious. Because the thermal budgets today are so small that, in fact, TEDs were actually the dominant effect that determines many junction depths, not ordinary diffusion that we taught you about earlier, a couple lectures ago.

And even sometimes not even concentration-dependent diffusion-- doesn't really matter that much. A lot of it is dominated by the transient diffusion effect, at least for certain dopants-- particularly boron.

So the type of enhancement we're talking about here, it may only last for a short period, but we're talking about maybe 20,000 times the diffusivity-- the ordinary diffusivity at 700, or maybe even 400 times the diffusivity at 1,000. So these are not negligible. These are huge enhancements.

And there was a lot of work in the early days spent at trying to figure out what this could possibly be due to. So let's go on to Slide 7.

The basic model for TED, what we assume is that all the implant damage recombines very quickly except for the one interstitial generated per dopant atom. So the easy way we think about it is just use the Giles plus 1 model, or plus n, if you want to be really accurate. But for now, we'll just do plus 1.

And here's an example of how TED effects can be seen. And they're actually quite non-local. And that's what's interesting about them too.

So here is an experiment that was done. This is a concentration as a function of depth-- so maybe a SIMS profile. And the initial profile before any annealing looked like this. So you have boron, which there's very low boron at the surface.

And then you have a boron marker layer. So it has some depth. At some depth, there's a boron layer that's doped about 1 E18. This marker layer can be put in a variety of ways-- could be put in by epitaxial growth-- whatever.

It's a region that's boron doped. We call it a marker layer. It could be the base of a bipolar transistor for all we know. And it looks like a box initially.

And then you go and you ion implant arsenic above it. Now notice the arsenic never touches the boron. So I'm not ion implanting arsenic deep into the wafer, it's just in the near surface region.

Now, this arsenic is a reasonably high dose. You can see the concentration is quite high. This arsenic does a lot of implant-- it creates a lot of implant damage. But the damage, of course, is confined to the near surface region.

A lot of the damage will anneal. Because if it's amorphous, you get SPE, right? And the fact, this little dashed line was supposed to represent the amorphous crystal and interface. So everything to the left is amorphized.

But we know that there is a certain amount of damage at the end of range. There are excess interstitials. And so these excess interstitials are then going to diffuse in. They're going to form these 311 defects. They're going to diffuse in, and then they're going to dramatically enhance the diffusion coefficient of the boron below.

So don't think necessarily of the implant damage effect as being, oh, the implant damage and the enhanced diffusion occurs only where the implant is-- just like oxidation. Remember, in OED, you can have a process happening at the surface, and you can have interstitials injected and influencing diffusion going on much deeper.

TED can be like that. The difference is that Ted is transient. It only lasts for a certain period, until the damage goes away.

One question I just had. So in this schematic illustration, see the green profile after diffusion. Look at the amount of diffusion. There was a lot of TED of the boron and not much for the arsenic.

Does anybody have any ideas, knowing what we know about the diffusion mechanisms of boron and arsenic and other things, why you would expect a lot more damage-enhanced diffusion for boron than you would for arsenic, as shown in this example? Anybody have any ideas?

AUDIENCE: [INAUDIBLE]

JUDY HOYT: What do we know about f sub i? What do we know about the diffusion mechanism of boron? Is it mostly interstitials, mostly vacancies, or both? Does anybody remember f sub i for boron?

Well, let's take a vote. How many people think it's 1, f sub i? All right. Yeah, it's 1. So what that means is f sub i-- it means boron-diffusion mechanism is almost entirely by interstitials, as opposed to diffusing with vacancies, OK? So excess interstitials-- boron needs interstitials around in order to move. That's its mechanism of moving.

How do we know that? We know that from a whole series of oxidation-enhanced diffusion experiments and nitrodation-retarded diffusion experiments that people have done-- injecting interstitials, injecting vacancies, and they see what happens to boron.

So in TED modeling what we're going to say is that this implant damage is going to inject into the silicon a lot of excess interstitials. Boron diffuses by interstitials. And its diffusion coefficient is going to be enhanced, or pumped up by these excess interstitials.

Arsenic-- what about the f sub i for arsenic, what do we think that is? Is it 1? .5? Anybody vote for a half? Yeah, it's on the order of half, roughly-- maybe 60%-- depends on who you talk to. People who do this for a living will argue their career on it. But you know, I think it's close to 60%.

Anyway, it's partly enhanced by interstitials. Rather, it's partly diffusing with interstitials, partly with vacancies. So you don't expect-- it doesn't have quite as much dependence on interstitials as does boron.

And also most of the arsenic profile here-- it's a little tricky question-- but most of it, remember, was to the left of the dashed line. So most of it occurred in the region where there was SPE. So that's going to regrow very quickly and not cause too much diffusion. So it'll turn out, as you'll see in most examples-- not all-- arsenic certainly has some TED, but it's much more prevalent for boron.

In fact, I want to show you some actual examples. That was a cartoon picture. But it's based on things that people observed in real life. Here's an example I want to show you of some data in the literature on where TED can affect a bipolar-transistor structure.

And these are epitaxially grown. I haven't talked about epitaxy yet, but it's a crystal growth technique by CVD, by which you can put in fairly arbitrary and quite abrupt doping profiles.

So let's take a look at the inset up here. And in the inset shows schematically the initial epitaxial structure of this bipolar transistor. So there's a region on the bottom in depth, which is n minus silicon. And then there's a region that is p plus. So it's got a high amount of boron-- silicon germanium. And then there's some lightly doped region. And then there's an n minus silicon cap.

So it looks like in effect, if you look at the SIMS data-- boron versus depth-- the region down here marked silicon, that's all epitaxial silicon. The region in the center is epitaxial silicon germanium. It's an alloy of silicon and germanium. It's used in a lot of high-speed bipolar transistors these days. And the cap on top is single-crystal silicon. And those are the dimensions.

And the silicon germanium region, when it was epitaxially grown, was doped with boron. And in fact here, the as grown profile is the diamonds. It's a little hard to see, but see these diamonds. It's a box. As grown it looks like a box, pretty much. And it has a height, or a doping level of about 3 times 10 to the 19th. So that's the as grown, very abrupt profile by EPI.

This little tail that you see here, this is SIMS knock on. So again, this is a SIMS profile, so it has some broadening due to the measurement technique. So that's what it looked like in the as grown.

And if you look at the crosses, if you do no ion implant-- so you do not implant the emitter-- after you anneal it, you get something that looks just like the as grown. So with the anneal-- and this particular anneal is 850 for 10 seconds. So it's an RTA. You don't expect much diffusion of the boron. And in fact, you don't get any measured by SIMS. It's still perfectly abrupt.

Now the third wafer, which is shown by these triangles, had an arsenic implant shown in the upper surface region-- just like I showed you in that cartoon. And this is actual data.

And now look at the boron profile. The open triangles for the exact same anneal, when you implanted the emitter, the open triangles have broadened. That profile is completely broadened. The P concentration is dropped, and now you have these large wings.

Well, there's a lot of enhanced boron diffusion. This is TED. And this makes the device essentially inoperable. So you cannot-- the HBT doesn't work when it looks like this. The device is ruined.

So a simple structure where you would calculate, oh, this anneal should be no problem. Here you do an arsenic implant on top. And all the interstitials injected cause this dramatic enhancement in the boron diffusion.

So it happens in real-life structures, and it's an issue. Yeah, people have found ways around. In fact, from that same article on Slide 9, here's some example of some attempts that were made in that article-- some of them successful, some not-- to get rid of TED. Well, one attempt was people thought, OK, after you implant the arsenic, just do an anneal-- 600 degrees.

Low enough temperature so the boron shouldn't diffuse, but maybe high enough to get rid of all the interstitials. OK, that seems maybe like a reasonable idea, but doesn't help at all.

In fact, if you look at these diamonds-- again, these diamonds are for-- the anneal that took place at 600 degrees followed by the usual 850 10 seconds. And it's still just as broad as it was before-- plenty of TED. So 600 degree annealing doesn't seem to be a way to get rid of the damage very effectively.

Well, the second panel down shows you another idea. How about melt the sample? That's pretty drastic. Hit it with a laser and melt it for a few nanoseconds-- so it completely melts, but it melts for so short a time that nothing moves. OK, that's melt induced-laser annealing and regrowth.

And you can see there-- and then after you do that-- you've melted the sample, you get rid of everything, then you do the 850 10-second RTA. And lo and behold, all those wings are gone, so the boron stays put.

So one way to get rid of that TED, of course, is to melt the sample, but that's kind of extreme. You need to use a laser, but it does prove that you can get rid of excess interstitials by melting, of course. And then when you do an 850 RTA, there's normal diffusion-- no more of the TED effect. So you can restore diffusion to normal.

And this was another interesting-- on the bottom panel, this was a sample where it was just annealed at 850 for 10 seconds, but there was a very high concentration of oxygen-- backround oxygen in the sample. In fact, the oxygen in the sample was about 10 to the 20.

So during the epitaxial growth, the silicon germanium layer was accidentally doped with oxygen. It was not really high purity material. And interestingly, TED is completely eliminated in the presence of a very high concentration of oxygen. In fact, you can see these plus signs are the boron profile on a sample anneal-- that, again, 850 10 seconds-- same RTA. The difference is-- and there was the same damage implant done on top-- in the presence of a high concentration of oxygen, there was no TED.

So that's kind of strange. And in fact, similar results since that time have been found for carbon. You don't need quite so much carbon. You don't need 10 to the 20th. You can get away with about 10 of a 19th carbon. So people intentionally dope carbon now in silicon, in those regions where they want to get rid of TED.

So this was kind of an accidental discovery initially, but pretty soon people started to realize this is a tremendous benefit. So towards the end of today, we'll understand a little better where these interstitials are coming from. But anything you can do to create a sink for interstitials-- be it a high oxygen-- oxygen is not the best element to use.

It also kills the lifetime-- minority carrier lifetime. So your bipolar transistor is not the best bipolar in the world. But neither is this one, where the boron base is diffused out completely.

Carbon turns out to be a little better to use. You don't need as much. You only need maybe a tenth the amount of carbon. And so because it's in the lower concentration, it doesn't disturb the lifetime quite as much as the oxygen. So people have found, they can dope with carbon without inducing a lot of bad electrical effects, and then completely kill TED.

And in fact, this is a topic-- we're not going to get to talk about it too much in this course-- maybe a little-- but this is a good topic, if anyone's interested in researching for their final report-- because we won't get to talk about it. But it is being used in production today.

Bipolar transistors, the highest speed, are doped with carbon to eliminate the boron TED. Otherwise, the base width would be really wide. The device would be really slow, and it would be a problem. So it's kind of an interesting story about how that developed.

So if you're interested, please think about signing up for that topic-- although I only want one person to do one topic. So you might have to fight a fight a little bit. There's plenty of topics to go around.

OK, given that a little bit experimental introduction, now we finally get to talk about, all right, how do people model TED? Well, I showed this slide last time. I just want to review what a 311 defect looks like. Here's a high-resolution electron micrograph. So this is electron microscopy.

High resolution-- the electron microscope is looking at a very high magnification. In fact, you can see these little rows of dots, each dot corresponds to a dimer-- to two silicon atoms. And the reason they're in such nice, regular planes is because they are in nice, regular planes. It's a single-crystal material. But here you see this diffraction contrast-- this dark region, where the perfect rows are disturbed-- their symmetry, because there's a defect.

And that's what we look at in cross-section TEM. We're looking for diffraction disturbances. And we see that by this dark region. And this dark region has been analyzed to death by microscopists. And it lies along a certain plane, which is typically 311 direction. That's where it gets its name.

And what it's believed to be is a ribbon-like defect, where this direction, this arrow points along 311-- and this direction, where the length of the long section into the page is lying along 110. And it is a whole series of dimers of silicon, or imers-- little silicon clusters into this defect. And it may be 100 angstroms long-- let's say, 10 nanometers-- and maybe 30 or so in this direction-- something like that.

So it's a little cluster of interstitials, that has a particular orientation to the cluster. And these 311 defects, they form during the first few fractions of a second of annealing. They can even be formed during the implant itself.

So their formation is important. More important is, how do they dissolve? They anneal out in the time range that's on the order of seconds to minutes. It could even be longer times at moderate temperatures.

So as they anneal out-- once they form, they form very quickly, and then they gradually anneal out. As they anneal out, they give off excess interstitials. And it's these excess interstitials given off by the evaporating 311s that cause the boron TED that people observe.

So atomic-level understanding of TED. Let's go on to Slide Number 11. How did people discover this? This seems really weird, or it seems very subtle. And in some ways, you might think of it as maybe partly accidental. People were looking in the microscope for years at these kind of interesting defects that they saw.

And in fact, they looked at the kinetics of the defects. They looked at how their concentration changed over time. And they noticed that the time it took to shrink all the 311s, or get rid of them, was about of the same order of magnitude as the time the TED lasted.

So people were doing TED experiments. And they said, oh yeah, let's see, at 800 degrees TED lasts for a certain number of minutes. And other microscopists were looking at it and saying, gee, at 800 degrees these 311s hang around for about that same amount of time-- could the two phenomena be related?

And in fact, they are. People found that the time scale of TED is the same as that of the time scale of the shrinkage of these defects. And when we're talking about shrinkage, people sometimes use the word evaporation. So the little 311 is sitting there evaporating off, or giving off interstitials from the clusters.

This is some experimental data. I took this from your textbook. It's referenced. It comes out of Bell Labs. And it shows-- it's a plot of the silicon self-interstitial density, in number of interstitials per square centimeter, contained in 311 defects. So they're basically looking in the microscope and counting the density of 311 defects and then estimating from their size and density how

Many interstitials are in them, as a function of time at different temperatures. Let's take the red boxes. Here at 815 degrees, initially at the very first beginning after the implant, there's a certain density-- maybe mid times 10 to the 13th interstitials per square centimeter-- contained in these defects.

And then what you find is exponentially-- so the defect density is sort of constant, then it goes down exponentially after a certain amount of time. It just drops like a rocket. So after 200 seconds, it's gone down by several orders of magnitude. So these 311 defects, a lot of them have disappeared. And so they're dissolving, or evaporating at a certain rate-- at 800.

So let's take a look at the red circles over here. Those are at 670-- so a much lower temperature. Look at this. You get a high concentration of them-- say, 10 to the 14th, or mid 10 to the 13th.

And then they last a long time-- maybe 10 to the 4th, or 10 to the 5th seconds, and then it starts to drop dramatically. So we're talking at a very long time at low temperatures. So this is sort of the kinetics of the disappearance of these 311s.

And in fact, these are some micrographs that people used, I'm just showing you on Slide 12, to obtain this data. This is from Dave Eaglesham at Bell Labs, published in 1994 in *Applied Physics Letters.*

And what he's showing here in the upper diagram, these are both plan-view TEM images. This is a sample of silicon that's been implanted with an intermediate dose-- 5 times 10 to the 13th boron per square centimeter-- into the silicon substrate. And it's been annealed at 810. And this is after 5 seconds.

And you see, you're looking at a plan view. And each one of these little sort of dot areas is a 311 defect. So there's a huge density of defects. And the scale bar is a little hard to see, in the upper left from here to here, that's 40 nanometers. That's 400 angstroms. So you have a huge number of these-- a lot of these defects.

So again, if I go back one slide to Slide 11, he's starting out here after just a few seconds. That's the number of-- that's where he got this interstitial density, at 815.

Now look at the bottom-- on Slide 12, look at the bottom TEM-- bottom left, in B. This is after 100 seconds. Again, the number of defects here is now 311. It's a lot lower. I can almost count them-- 1-2-3-4-5-6-7-8-- of some order. And based on their size and density, you can calculate after 100 seconds.

Oops, sorry, let me go back one more. So here we are at 100 seconds, and it's down by several orders of magnitude. So people sat in this paper and counted all these defects-- figured out the silicon interstitial density and how it was changing with time at different temperatures.

And so in fact, I'm showing here now, also from your textbook, on Slide 13, this is a diagram that's supposed to give you an idea of the kinetics of what's going on with these defects. So this is simulated, though. What I just showed you was actual data. This is more simulation. So this is on the left axis, or the vertical axis is silicone self-interstitial density.

Now this is in terms of-- there's different ways of expressing it. This is per cubic centimeter. And this is as a function of depth. So there's a particular implant that's been done-- 40 Kev boron, 10 to the 14th. And initially this red line represents the initial plus-1 damage. So right after the implant is done, you would assume that you had this damage-- this amount-- number of interstitials.

So the interstitial density peaks here very, very high. After a microsecond, it's about the same. But look at it after a tenth of a second, or a hundredth of a second. We're talking about this flat profile right here. So after a tenth of a second at 750, we formed all these 311 defects. And the interstitial concentration is now down to about mid 10 to the 12, or 10 to the 13th per cubic centimeter, according to Supreme. And it's fairly uniform.

OK, let's think about that. What is CI star? We have equations in your textbook from a prior chapter. The equilibrium silicon interstitial density is about 10 to the 8th per cubic centimeter at 750. So what is that enhancement ratio? CI over CI star. That's 10 to the 13th over 10 to the 8th, roughly. It's still greater than 10,000.

So we've gotten rid of most of this initial damage, but now we have this sort of uniform concentration of-- we have this concentration of 311s. And the silicon self-interstitial density CI over CI star is still much larger than it would be in equilibrium, but maybe by a factor of 10,000, in this case.

So the TED is going to occur between this period-- this period here of 0.1 seconds and 1,000 seconds, depending on the temperature that you're at-- how long it takes to evaporate the 311s. During that period when the supersaturation ratio is large, it's going to be some high number-- could be a factor of 10, 100, 100,000-- something like that-- and uniform in depth.

So as the 311 defects, they've all been formed, now they start evaporating and emitting interstitials. Eventually, they're all evaporated away, and TED will be over. But for some period, they'll be evaporating, and they'll be holding CI over CI star very high, and therefore they'll be holding the boron diffusivity very high.

So let's go on to Slide 14. And this is a model that was proposed in the mid '90s or so for 311 growth. So we have the growth of these clusters and their evaporation, or shrinkage. And this kinetic model, then, is going to determine the time dependence and the magnitude of transit-enhanced diffusion.

So what people wrote down in this paper was-- and there's a reference in your text. If you want to read the original paper, I would invite you to read that. You can write down an equation that describes cluster growth. So CL sub n in this equation is just a 311 cluster that has n number of interstitials. n could be 100. It could be 1,000-- whatever. It's a cluster.

And if I add to that n number one more interstitial, I get CL to the n plus 1. So this equation could go either way. I can either add an interstitial and grow the cluster. I can evaporate away, or take away the interstitial and shrink the cluster-- either way.

So given a simple equation like this, then we can actually write down a time dependence. We can say that the time rate of change of the cluster-- number of interstitials in the cluster-- as a unit-- as a function of time-- the partial-- is equal to this. There's a growth term, on the left, minus the shrinkage term. So the growth term has some equilibrium constant, K sub f, for the forward reaction that says-- where I'm adding interstitials, and I'm growing this thing.

So it was a constant case of f times the concentration of interstitials times the concentration of clusters. That is the forward term-- minus the reverse reaction. If I shrink it, I go the other way and I release an interstitial. So the reverse reaction has an equilibrium constant KR and some concentration of clusters CL. So we write a simple differential equation-- time dependence depends on a growth term minus a shrinkage term.

And now this is a little hand-wavy. But in your text, we talk about how this forward reaction rate is believed to be diffusion limited. So the interstitials have to diffuse into this cluster.

They're created throughout the crystal, but they have to diffuse into the 311 cluster. So we usually say that this forward reaction constant, K sub f, is somehow proportional to some nearest neighbor distance, A, and an interstitial diffusivity-- D sub i. So at least we know what kind of parameters go into that. So that's what we write for K sub f.

And the exact form doesn't have to be perfect. We just want to get the temperature dependencies in most of these cases. The reverse reaction-- so going the other way-- for the evaporation of the shrinkage is actually dominated by a diffusion mechanism, as well. So there's this term that's related to the diffusivity of the interstitials, again-- it's actually like-- this looks like a hopping frequency-- times a Boltzmann factor, E to the minus EB over KT-- now, where EB is the binding energy.

So again, if I'm going to get rid of an interstitial from the cluster, it's going to be bound by some energy. Somehow there's a lower energy to be in the cluster. I have to overcome that to get rid of it. And that's why you have this E to the minus EB over KT. So simple differential equation with 2K sub f and K sub r, that have certain temperature dependencies.

Let's go to Slide 15. So the interesting part of this differential equation-- the interesting time is the period where steady state exists between cluster growth and cluster evaporation. So in fact, to make the equation easy, we're just going to solve for the case when it's equal to 0. We're going to solve for the case where the cluster concentration isn't really changing very much. And we're going to say that's equal to 0.

So that's relatively easy. Let's just go back. I'm just setting this equation equal to 0. And now I'm going to solve now for the concentration of interstitials, because the cluster concentration drops out, right? So everything I can solve, then, for the maximum interstitial concentration just in terms of the ratio of KF and KR.

So going back to your Slide 15, I can then figure out a maximum concentration of interstitials that are trapped in these 311s. It's just KR over KF, and it looks like this-- depends exponentially on the binding energy over KT. So that is an estimate of the maximum number of excess interstitials.

If I divide that by CI star, that gives me the supersaturation ratio. Again, why do I want that? Remember in our discussion of diffusion, diffusion coefficient goes diffusivity times F sub i times CI over CS star. So whenever you enhance CI over CI star, you enhance the diffusivity-- say, of boron.

So I write down this ratio. And we have to use the CI star. We use the formula for it coming from Chapter 3. And CI star brings in with it a formation energy of the silicon interstitial, that we're calling E sub f. That's not the Fermi energy, that's the formation energy.

So this is the formula that was used for CI star. It's got a pre factor-- CI 0-- and an exponential-- E to the minus EF over KT. So the formation energy is about 3 electron volts, roughly, for silicon interstitial.

The binding energy-- people have fit data to show that the binding energy of a silicon to this 311 cluster is about 1.8 electron volts. So people have estimates for this term here and this term here. You can put in estimates for, you know, the nearest neighbor distance, and CI 0 can be estimated. So you can plot this equation-- CI max over CI star-- as a function of temperature.

And in fact, that's what Slide Number 16 shows. This is a plot of a simple equation as a function of temperature-- the interstitial supersaturation ratio as a function of temperature. So what does this tell you?

Well, it gives you an idea of the maximum enhancement in the diffusivity, right? Because if I'm going to say that boron's diffusivity depends on F sub i times this ratio-- and let's say, I'm at 800, and I'm in a period where I have TED at 800, CI over CI star can be as large as almost 10 to the 4th-- maybe mid 10 to the 3rd. So it gives you an idea of the magnitude of the enhancement in the boron diffusivity.

Relatively simple derivation-- you can get an idea right off the bat of how much CI over CI star can be enhanced during the steady state period. Now, eventually, it's going to end. Eventually, all the silicon will evaporate-- the 311s. There'll be no more excess interstitials, and TED is over.

So the question on Slide 17 is, how long is that? So now I know how much CI over CI star is boosted up-- simply a function of temperature. You can pull it right off that plot.

Now the question you ask me-- well, I know the temperature. How long does this event last? How long does it take to evaporate all these 311s?

Well, eventually over time, the 311s all evaporate and CI over CI star goes back-- actually, it should go back to 1, basically. So here's an example, on Slide 17. This is from a Supreme simulation. This was for boron TED. And we're annealing at a certain temperature. This is the implant that was done-- 10 Kev boron, 10 to the 14th atoms per square centimeter.

And here's the as-implanted concentration profile here, shown in blue. So that's the boron, just to give you an idea. And then after 1 minute at 750, this is how the boron has diffused-- the black line.

And look, at 1 minute at 750, the dashed line refers to the right hand axis. Sorry, you should write that in. So this dashed line is to the right-hand axis. That's the interstitial supersaturation ratio. At 750 it's about 10 to the 4th. And it's pretty uniform throughout the sample.

In fact, I could probably have put that off from the last plot. If you go back to your last slide-- 16-- 750, look it up-- CI over CI star is about 10 to the 4th. So you can use this as a simple back-of-the-envelope way of calculating the amount of enhancement-- CI over CI star.

And then after 10 minutes, look how far the boron has gone. The boron profile has gone quite a ways. The junction depth now here is about 0.4 microns. So that's the red profile-- solid line.

And the dashed line here, you should also reference that, to the right-hand axis. The interstitial supersaturation ratio is now down. Instead of 10 to the 4th, it's 10 to the 2. It's about 100 after 10 minutes, because we're getting rid of more 311s. And eventually the number of them is going down.

So this thing only lasts so long. And so the question is, how do I figure out-- a back-of-the-envelope calculation-- how long I need to know that this enhancement is going to last as a function of temperature? So let's go to Slide Number 18. And here's a way-- a simple quick and dirty way of estimating it.

What we say is that all these excess interstitials, how do we get rid of them? They are emitted by a 311. They don't just sit there. They're going to diffuse, OK? Where are they going to diffuse? Well, they're going to diffuse into the bulk and recombine in the bulk. And they're going to diffuse to the surface.

And the surface, you know, is a perfect place to have recombination, because there's lots of vacancies at the surface. So an extra silicon atom can always be accommodated at the surface. So the diffusion, you have this-- let's say, this initial profile of excess interstitials. This is a very crude profile, as a function of depth. So this red line is meant to represent the surface.

Moving from left to right, we're going in deeper in the sample. I have some dose Q of interstitials, that I've implanted. How did they get there? Well, they got there because they were recoiled.

So here's my dose Q. And it peaks at some projected range RP. OK. And the surface being the dominant sink, especially if it's a low-energy implant, I'm going to have a certain flux of interstitials towards the surface.

Well, how can I write that flux? Well, we can write it as the diffusivity, D sub i, times the peak concentration, CI max over RP, where RP is the distance-- where the peak is the range of the implant. How do we get that? Well, remember Fick's law. Flux is a diffusion coefficient times a concentration gradient.

Well, here's a very crude way of estimating the concentration gradient. It's just the peak concentration divided by the distance over which it recombines, assuming they all recombine at the surface, or it recombines to 0. So here's a very quick estimate of the flux towards the surface-- DCI max over RP.

Now, I need to know, how much time does it take to dissolve all those? It's going to be the dose Q, which is whatever I implanted, divided by that flux. Here's my flux. So at the bottom of Slide 18, I've just taken the dose, and I multiply it by 1 over the flux.

So the time to dissolve these clusters is going to be Q times RP divided by the diffusivity of the interstitials times the maximum concentration-- CI max-- the maximum concentration. That gives me an idea. I have an idea-- how long will TED last? Well, it lasts until all the clusters have been dissolved, or evaporated.

And you can see, if I implant a higher dose Q, I expect TED to last longer. If CI is larger than the concentration, then you expect the time to last a little shorter. So this gives us a rough idea.

In fact, we can simplify this and put it in terms of a temperature dependence, if we go to Slide 19. So here is the time that TED lasts-- tau-- as I just showed it from the last slide. Now, I'm going to put in what CI max. We just calculated the maximum interstitial-- excess interstitial concentration induced by the implant. Remember, we said it was KR over KF.

So we have that in terms of a Boltzmann factor on the binding energy. And so I can just substitute that in. And then in the middle of the slide, we can write down the time to resolve with the clusters, showing here.

Well, it depends on Q, the dose implanted, RP, the diffusion rate, and an exponential of the binding energy of the silicon atom to the cluster of the silicon interstitials. We can put in what the diffusivity looks like for silicon interstitials. It's a cost prefactor times E to the minus E migration over KT, where people have estimated what that migration energy is.

And here we have a relatively simple expression for the time constant-- the length of time that TED lasts as a function of temperature. It depends exponentially on the temperature. And inside the exponential over KT, I have the binding energy plus EM, the migration energy. So I have a nice little formula for how long TED lasts. It depends on the dose too, in RP.

So let's go to Slide 20. Here's an actual example, using that simple formula. We're asked to calculate and plot how long TED lasts and how it depends on temperature for an implant of 40 Kev phosphorus at 10 to the 14th. Well, from the figure-- you can go back to Figure 8.3. It's just RP as a function of energy. You can figure out the projected range. It has an RP of 60 nanometers.

And you have everything you need to in this formula. You have the dose. You have the RP estimate. We know EB-- binding energy is 1.7. The migration energy is 1.8. We add all that up. And here is the equation.

So this gives us the time dependence for this particular energy and dose. And there it is on Slide Number 21. So this is the time-- the duration in seconds-- how long TED is going to last for this particular dose and energy of phosphorus as a function of temperature.

And lo and behold, look what it shows. At 1,000 degrees, or maybe 950, TED only lasts about a second. OK, so it's very short. Because you form the 311s, and they have a pretty high evaporation rate. If you're down here at 800, TED lasts, maybe for this implant, about 100 seconds. OK, so it lasts a lot longer-- minutes. And the temperature dependence of this time is given by this equation.

Now, if I have a higher dose-- let's say, I do 10 times the dose-- what happens to this line? The red line, does it stay the same? Does it move up by a factor of 10 times longer, or 10 times shorter? Based on-- let's just go back one slide. What is the dose dependence of that time?

Depends directly on the dose, right? So if I implant 10 times the dose, TED is going to last 10 times as long. See, the Q dependence. So you can use this in a lot of your homework problems, or anytime you have a problem. Assuming the RP is about the same, you can use this nice curve, because it just scales linearly with the dose.

So if I implant 10 to the 15th, then at 1,000 degrees, instead of lasting a fraction of a second, it could last a full second. At 800, instead of lasting 100 seconds, it could be 1,000 seconds. So that's the interesting thing about the dose of the implant, it tells you how long TED lasts. Because the dose injects a certain amount of these interstitials. They come together to form 311s.

Depending on how many you originally form, it'll tell you how long it takes to get rid of them. And how long it takes to get rid of them is a strong function of temperature, as well.

So Slide 22, I took this equation from your text. It looks a little mysterious. It shouldn't be. What this is saying is the overall amount of profile motion-- remember, we said, we could multiply-- we had something called the DT product. It gives you the idea of the thermal budget. The DT product effective of a dopant, remember, it depends on its equilibrium diffusivity, D, times CI over CI star.

Now, instead of putting down time-- the total time of the implant of the anneal-- I'm going to put down tau enhance. Tau enhance is the time that TED lasts.

So you can plug-in for those things-- CI over CI star and tau enhance-- and you get something that looks like this. OK, so it depends on the diffusivity of the dopant, the self-diffusion coefficient of silicon interstitials, the concentration of silicon in the dose, and RP.

So the purpose of showing this is it explains the backwards, or anomalous temperature dependence of TED. Even though the dopant diffusivity has an activation energy of about 3.5-- so that's what you would expect for this thing-- this is being overwhelmed by the activation energy of the self-diffusion. So here we have DA-- the activation energy of the dopant diffusion-- might be E to the minus 3.5 over KT.

But in the denominator is the diffusion of silicon itself, which has a much higher activation energy-- E to the minus 4.8 over KT. So this is how we can-- when we ratio these two, we end up with a positive in the exponential. So this is how it's possible to get more broadening at a lower temperature. That's just from looking at the equation.

The thinking behind this-- the rationale is you have a fixed amount of damage introduced by the implant, but the background point defect concentration-- CI star-- and the interstitial self-diffusion coefficient, they go down rapidly with temperature. OK. So again, the amount of profile motion depends on the ratio-- CI over CI star.

So CI star is going down very rapidly with temperature. So the supersaturation is then going up as temperature is lowered. So it's partly because CI star goes down that this ratio is lowered, and so you get more TED at low temperatures.

And also the duration of the excess interstitials is longer at low T, due to the lower diffusion coefficient. We just saw that. Look at this plot-- Slide 21. The lower I go in temperature, the longer it's going to take to evaporate all those interstitials and get rid of them. So TED lasts longer, and it can have a stronger effect at low temperatures.

In fact, Slide 23 is just an example of that taken from Supreme simulation. This is a boron profile annealed at different temperatures. So its concentration versus depth. The solid lines here represent the boron, and they refer to the left axis. The dashed lines represent CI over CI star. They refer to the right axis.

So let's see what happens to boron at different temperatures for the same amount of time-- 900 degrees, 800 degrees, 700 degrees. Look at CI over CI star at 700-- much higher than at 800 and also much higher than at 900. And it lasts a lot longer at 700.

So we do a 30-minute anneal at 700, the entire period is transient-enhanced. Whereas if I do an anneal at 900, how long does it last? Well, of this order of magnitude of a second-- so only the first second is enhanced, and then TED goes away.

So that's how you can explain a low temperature anneal ending up with so much more motion. It's got a higher CI over CI star, and it lasts longer.

Slide 24-- just going through a little more of the subtle effects. And you can read through this in your text. We have this sort of equation that we just wrote down for the DT product, which is a measure of the amount of broadening for transient-enhanced diffusion.

And the question is, how does it depend on energy and dose? Well, on the left-hand plot, this is some actual data that was taken. People measured the square root of DT-- how much does the does the dopant move as a function of the energy of the implant that was inducing the damage?

And as you can see at high energies, you actually get a saturating effect. You see, this equation predicts that the amount of motion should depend on RP, linearly. So if you go to a higher energy, you expect a higher amount-- a larger amount of broadening.

And that's true in the beginning. So here we go low energy here-- 10 Kev, 60. We do see this increase. And it starts to saturate out. And that's because high enough energy, the surface is no longer the dominant sink. In fact, you get bulk recombination as the profile gets really deep.

And remember back here when we were doing this simple derivation-- back-of-the-envelope derivation on page 18-- remember, we were saying-- we ignored the diffusion flux that went into the bulk and recombined. For a simple back-of-the-envelope calculation, we said, all the interstitials diffuse towards the surface and recombine there.

When you get them deep enough in the bulk, that's not necessarily the right model. Again, just to show, this is a little back of the envelope. So in real case, the motion-- the amount of TED will actually saturate at some energy. So take this RP dependence with a grain of salt.

Same thing for the dose dependence. This is again the square root of DT-- the diffusion distance. Now this time versus anneal time, it turns out, if you look at different doses-- indeed, if you do a long enough time here, which I'm showing here-- say, close to 1,000 seconds, or close to 10,000 seconds-- you can see a dose dependence.

And in fact, this open bullet is for 10 to the 13th. The closed bullet is 4 times that. And indeed, you do see about 4 times the dopant motion.

So it does scale with the dose, but only at long enough times. At short times the dose dependent is not so evident. And that's because, again, we did a very simple calculation of that differential equation. We solved only for the steady state period, which takes a certain time to develop.

So this is more of a second-order effect. So this equation, again, you take it with a grain of salt. It's meant to give you a back-of-the-envelope estimate.

So on Slide 25-- when you walk away today, this is the general picture you should take in your head of TED. And what is, it's a plot of the enhancement in CI over CS star in a log scale as a function of time.

And so what we say is we have some steady state period, and its length is called tau enhancement. It's the time during which the 311 clusters are decaying. It lasts a certain period-- tau. And that period depends on-- the duration depends on the dose, of how many interstitials I put in. And then after that period, it just rapidly exponentials-- exponentially decays.

So the critical parameters are, what is the supersaturation height, or the level, which depends on the temperature-- as we know, it goes up as temperature goes down. And what is the duration of that steady state condition, tau enhanced? So with those two pieces of information, how high is it enhanced, and how long does it last, you can do a lot of predicting, roughly, of the amount of profile motion you're going to get due to the TED effect.

OK, so let me just summarize what we've said about ion-implanted TED. Ion implanted is the dominant method for putting dopants in. We talked about [INAUDIBLE] statistics, that we can calculate using simple distributions. We talked about Pearson IV in amorphous targets-- how it works very well.

Ion channeling has to be modeled by something more complex and complete, like Monte Carlo. Next time, I'll show you some ion channeling profiles and how they're fit by dual Pearson. We spent some time today talking about the plus-n model from for residual implant damage. It tells you that you get roughly n excess interstitials per primary ion.

These excess interstitials all get together. They cluster into these 311s very quickly-- in less than a tenth of a second. And they dissolve very slowly. And their dissolving process, that gives rise to TED. And we have a simple model for TED. They can explain the time, the temperature, the dose, and, roughly, the energy dependence of TED.

And so next time in class, we'll do a few calculation examples. If you can bring a calculator, we'll do some calculations together. If you have a calculator, it'll be easier. We'll actually do some calculations-- back of the envelope calculations on TED kinetics.

And that clipboard is going around. Does everybody have that? Please sign up for your project, and at least sign up for whether you want to do written report or oral report. OK, thanks.