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Fresh Meats and Microbiology Discussion

M. J. Marchello, N. Dakota: Do we really have a problem with trichinae?

A. W. Kotula: Back in the early 1900's we did, but to my knowledge now there is less than ½ of one percent incidence of any trichinae occurring in any of our pork products.

I think that the last information that I saw that Zimmerman published was 0.125 percent incidence and I will let you decide whether you have a problem or not. What we were doing in our work was to try to ensure that we did not come out with any recommended cooking procedures which would not destroy the trichinae if present and we always said "if present." Now, the press looks at it one way and there is not much we can do about that. I think it is essential, however, for scientists to be aware of our concern that, if trichinae were to be present in pork, some of the methods which we had worked with and evidently some of the methods that Bill Zimmerman has worked with would not provide adequate heat to destroy the trichinae.

D. M. Kinsman, Connecticut: Gay, do you have any information about the relative merits of prebrowning as it pertains to the time element and the energy element?

Starrak: Not as specific as I would like to have. There has been some work done with that at Texas, I believe. We did find some disadvantages to prebrowning in that it does increase both the time required and the amount of energy used. Also, many cooks don't particularly like to use the browning dishes that go in a microwave oven. They are accustomed to thinking of microwave being cool cooking and there is a danger of getting burned when you find this hot dish — and a browning dish is really very hot. If you are browning meat conventionally on top of the range, you have two cooking pans to deal with rather than one. One of the main advantages of microwave ovens is that the cleanup is much easier than in conventional ovens. When you use browning dishes and skillets you wipe out the advantage that you have with microwave cooking. Perhaps somebody else knows more about the comparative energy used, but microwave does save energy in most cases. In some cases, like beef patties, it compares about the same as cooking them on a surface unit of a gas range.

Thayne Dutson, Texas: King and Harris, and King and associates weren't able to find connectin after heat treatment of muscle. There are a couple of possibilities I see and I was wondering if you would comment on them. One is that maybe their method of measurement after denaturation didn't detect the connectin. The other possibility is that gap filaments are possibly composed of something in addition to connectin. In other words, could there be a connectin component or maybe something in addition to that?

R. H. Locker: I actually had that result in my script but I took it out, being pressed for time. It is perhaps one of the nicest effects on the face of it for my theory. When you cook meat at 80 degrees, connectin disappears. Certainly this proves that gap filaments can't be of any significance in cooked meat.

M. J. Marchello, N. Dakota: I have another question in relation to the stretching of the A band and the pulling of the myosin filaments in both directions. You did show a micrograph where at times that doesn't happen and we have noticed that as well. What do you think is the reason for those

to move in one condition, but under the same conditions in another piece of muscle or at another time, they don't? Is it a different fiber type, do you think?

Locker: Well, it's a puzzle to me too. I have thought of this. I have a vague suspicion that it could be another fiber type.

M. L. Greaser, Wisconsin: How do you reconcile your gap filament idea with the antibodies staining for connectin? In the studies that have been done so far, it appears that the primary antibody staining is at the AI junction rather than throughout the whole myofibril. If we look at your model the way it is now, you would expect that you would get staining all the way through the whole thing about equally.

Locker: God made this complicated with things like N lines, a curious thing, sometimes you see them, sometimes you don't.

Greaser: Well, I guess my feeling is, if in fact there is a gap filament structure as you have proposed, that there is a whole lot more complicated structure than what you have. If titan or connectin are a part of it, then I think that there is perhaps an inner connecting transverse network somewhere in the region of the AI junction that may be contributing extra protein there. I agree that you probably wouldn't expect to see it in the center of the thick filaments when you do antibody staining, but the staining that has been done so far shows that there is some diffuse staining for connectin through the I band as well which also doesn't fit very well with your model.

Locker: I have a feeling from many years of looking at I bands that a lot of quite complex structure is yet to be found, particularly around the N line region.

Greaser: I might add that we have done some very simple playing around with different extracting solutions and just looking at what was done in the phase contrast microscope and you get quite different structural appearances depending on the kind of extractants you used, whether it be a Guba-Straub or Hasselbach-Schneider solution and some other things that are a little more exotic. All of these things lead me to be convinced that there is not just a simple structure of thick and thin filaments. There has to be some additional connecting structures present.

Kauffman, Wisconsin: You have gone to a great deal of effort to tell us how this is going to affect tenderness in the postmortem tissue. Undoubtedly, you have also thought a lot about the biological significance of this in the living animal, and so I would like to have you tell me if there is more to it than simply a structural phenomenon that the gap filament plays. Is there anything that has to do with contraction?

Locker: The most interesting thing about gap filaments is their real biological function. Whether they are involved in contraction, I wouldn't have a clue. The only thing that I can put my finger on at the present time is the fact that the N line seems to be suspended on this. We have two pieces of evidence, one of which I have showed you. I think that biological function of the gap filaments is tied up with the biological function of the N line. I know it is going to be a profitable and worthwhile field for somebody to give some serious attention to.

Dutson: We have seen gap filaments show up in sternal muscle on an animal which still had the physiological attachment. If you hang an animal up and let the head come down, there is enough stretch applied to that muscle to pull it out to the point where you can see the gap filaments. Now, this muscle is still in that condition where it can contract and, if you let the head come back and the muscle is stimulated it will come back and still show a normal type structure. I am wondering if you have seen this type of thing and also if you feel that maybe the gap filaments are connected with the other material in the N line region — sort of an orientation type of thing for some kind of extra physiological stretch in order to get the system back into functioning again?

Locker: I mentioned before that the fact that you can stretch a muscle beyond the N line point and it comes back without apparent damage may well be a result of the presence of the gap filaments. I am not quite clear whether this is likely to be a real physiological function. I suppose if you can see it in a hanging carcass, then perhaps it is the sort of situation that may actually occur in life. I am not a physiologist or anatomist and possibly the situation does occur in normal muscle functions sufficiently often to require some such recovery mechanism. I would personally like to think that there is some other reason for that, but perhaps that isn't itself a very good reason. I don't know.

Kauffman: One more thought. Have you ever looked at diseased muscle and been able to identify your same gap filaments? Is there a possibility that this is related to syndromes of muscle disease?

Locker: It is an interesting question. Certainly I think there is more activity around in those muscles so they might be more vulnerable. That could be an interesting thing to look at, but I can't answer it.

Greaser: What evidence do you have that in fact the gap filaments are not partially unraveled thick filaments or thin filaments that have been pulled loose from the I band and are now in that position? It seems to me that clearly from the diameter they are not the same size as a thick filament, but it is very difficult to distinguish their size from a thin filament. If one wanted to argue the point that these are really just thin filaments that have been pulled loose, what sort of evidence would you have that that is not the case?

Locker: For myself, I find it fairly convincing that Hasselbach-Schneider solution sort of leaves them behind. I can't really see if they were dragged out, they shouldn't still dissolve. They seem to be quite immune to this very powerful solvent. I just find it very hard to accept that they were simply stretched out.

Greaser: Hasselbach-Schneider solution will dissolve myosin, but if, in fact, by virtue of your stretching you have pulled loose some thin filaments from the Z line attachments, for instance, they are now in the gap between. Whether or not you dissolve the myosin, they would still be found in that location.

Locker: I think my pictures indicate that the I filaments are quite independent of them. Even in those wildly stretched sacomeres that are up to about 11 or 12 microns, you still see the I filaments sitting intact.

Greaser: I am not trying to tear down the model, but I am just saying I am sure that there are some people who have viewed the work and said that gap filaments are just I filaments pulled loose. From what I have seen, you don't yet have convincing evidence that those thin filaments are not, in fact, actin.

Locker: Actin filaments?

Greaser: Actin filaments!

Trout: As you are aware, Macfarlane did work on pressure treatment of prerigor meat and is supporting what Marian Greaser was saying. It has shown that the temperature and pressure will depolymerize the actin. If that is the case, it would support further the direction to which Marian Greaser was pointing. Any comment?

Locker: There is an old saying that pressure treatment very rapidly depolymerizes thick filaments too. It will do this in a matter of miliseconds virtually at ordinary kinds of temperatures. The mechanism of pressure heat tenderizing is a pretty complex thing. I have done quite a bit of work on it lately from the point of view of seeing whether it was the gap filaments involved. Having done it and satisfied myself at least that gap filaments are involved in this, I am still not clear just what the mechanism is. The strange thing is that a lot of thick filaments, for example, are supposed to depolymerize in milliseconds under these sort of pressures as well as I filaments. They must put themselves together again remarkably well when you let the pressure off. There is a lot of very curious things about them. I have quite a lot of results. We certainly don't have time to talk about it today. I am not sure whether this mechanism is entirely a depolymerization or whether a question of accelerated aging is involved as well. I have some suspicions that this might be the case, but I am not sure.