According to Gregor’s 2008 Dev Bio paper, the gradient shape of larger embryos in different species is governed by [tbd]

They show that Bcd protein in some species travel up to 3 times further than in other species of *Drosophila*. They hypothesize that there exists properties within the Bcd protein which confer stability in a species specific manner. However, they showed that this is unlikely to be the case as their cloned Bcd from *Lucilia* and *Calliphora* did not generate gradients that were significantly different from endogenous Bcd from *Drosophila*. [This leads me to believe that there must be a systemic difference between the species, such that the diffusion constant must be changed. Because the length constant of the gradient is defined by two factors, the diffusion constant and the degradation rate; thus if they show the degradation rate is similar, then the only alternative explanation is a change in the diffusion rate.]

Another alternative explanation is that the region of expression is different. Many gradient models make the assumption that the molecules originate from a point source; however this is hardly a biological reality. However, the length constant only describes the rate of change (sort of, not accurate), if the origin of the gradient were to be larger or effectively further into the embryo, in a posterior-ward direction, then it may allow for the proteins of the gradient to reach further into the embryo. We may be able to apply this logic to the effects observed in the Line 2.49.3 embryos; while this may not be the primary factor, it is certainly likely that it is a contributing one. It has been shown that larger embryos contain a higher aggregate amount of maternally deposited Bcd mRNA; however the area or volume that represents has not been ascertained. Either the mRNA density is the same (or similar) and the total region of space occupied is larger or the cloud is in fact tighter; I think an argument can be made either way in this regard (subject to what the data shows). I think we have to make the assumption that the specific timepoint of these embryos can be assumed to be evolutionarily stable; while not particularly strong, they are still viable.

We observe that the Bcd protein gradient from Line 2.49.3 can be shown to be scaled; namely the profiles converge when overlayed with Line 9.31.2 embryos. This scaling property can be partially explained by the dramatically larger length constant (144 um); we reason that this indicates to us that the cause is related to either degradation or diffusion, as per the factors the equation describes. We find it difficult to make the argument that the degradation rate is the culprit, because an increased degradation rate (w) would cause the B0 to be lower, as we observe, however it would also dramatically reduce the length constant. Conversely, a decreased degradation rate would allow for a longer length constant, but then the suggested increased B0 would conflict with our findings.

It has been suggested that there may be differential degradation rates, specific to the regions of the body; namely (wa) and (wb), referring to specifically to the anterior region and the rest of the body, respectively. If this is the case, then wa would have to be necessarily high and wb would have to be lower in order to appropriately explain our findings. [How can we possibly test this? Fitting lambdas to different regions of the embryo? Perhaps break it down into segments of 10% EL]. Furthermore, this line of reasoning is problematic in that there is no observable physical barrier to help define these regions of differential degradation; the only regions which are demarcated in the syncytial blastoderm are the nuclei; however, it could be the case that if in fact the degradation of Bcd in the gradient occurs primarily in the embryo nuclei during the gradient formation stage, pre steady state. Then it could be the case that the nuclei in are programmed differently so as to allow for the adaptation of the degradation on a nuclei by nuclei scale. [It’s critical to determine the role that the nuclei play in the gradient formation phase of morphgens; there is much debate surrounding this topic].

So there are several factors in play, if the nuclei do indeed play a significant role in the formation of the Bcd morphogen gradient, then in what manner does that occur? It is apparent that they may control at least one of two factors, again degradation or diffusion. It may be the case that the nuclear trapping facilitates the diffusion in a more effective manner in larger embryos. It may also be the case that the degradation rate is affected in the embryos via the nuclei; it could be uniformly higher or lower or potentially non-uniform. A non-uniform degradation rate is a difficult position to argue in that the cells themselves do not know their position before they interact with Bicoid, so how can it be the case that they have positional information before they have positional information?