

ONTH, ONE PROTEIN <

Issue 137, April 2012 www.proteinspotlight.org

kiss of life

Vivienne Baillie Gerritsen

We all take Spring for granted. The moment the first bouts of warmth hit the air, we fully expect to see the lawn duly mottled with daisies, leaves pushing their way into the nascent season and flowers blossoming wherever we care to look. And quite rightly so. We all know it's going to happen since it does every year. And we do realise that Nature needs to renew itself every once in a while. The process is – you could say – automatic. But it is only automatic because there are hordes of molecules that are able to recognise, in many different ways, the environmental cues – such as warmth and humidity for instance – and translate them into growth. An amazing state of affairs, if you give it a little thought. One such molecule, known as DELLA protein RGL2, has been the centre of attention amongst plant molecular biologists for some time now. Indeed, RGL2 is proving to be at the very heart of seed germination.



Ana Duncan, *Germination* Medium: Bronze, 53 x 36 x 29cm Courtesy of the artist

Over time, Nature has developed a cunning way to keep seeds from germinating unless the environmental conditions are favourable. Seeds are able to remain in a dormant state for a long time – as many as thousands of years actually. The oldest seed, known to date, still had all it needed to germinate two thousand years after it fell upon the tiles of Herod the Great's palace (73-4BC) in Israel! Besides being a very clever system, it is mind-boggling. Seeds that are able to survive so long are seeds that are in what has been a coined a "dormant" state – a little like Perrault's Sleeping Beauty awaiting the kiss that will free her from lethargy.

A dormant seed is surrounded by a coat, itself made out of two layers – a first layer which is "dead" and rigid and a second, known as the endosperm, which is still "alive", and in immediate contact with the embryo. Scientists discovered that when you remove the coat as a whole, germination is triggered off. This means that there must be something in the coat which represses germination. In other words, there must be something that belongs to the coat and is diffused into the embryo to keep it dormant.

The system is complex, and no doubt demands many more years of research. But the rudiments of germination are beginning to emerge. There is a phytohormone, known as ABA – or abscisic acid – which actively represses seed germination. ABA is found in the endosperm and diffuses into the embryo's environment. How, exactly, it represses seed germination remains unclear but it has been demonstrated that its presence in sufficient quantities is necessary. The expression of this particular hormone is dependent on a protein known as DELLA protein RGL2, also found in the seed's coat. RGL2 favours the production of ABA, thus keeping the seed in a dormant state.

The expression of RGL2 itself is dependent on the presence of another plant hormone known as gibberellin - of which there are many types. Gibberellin was first discovered in the 1920s in Japan and isolated in the 1930s from the fungal plant pathogen Gibberella fujikuroi - hence its name. This particular hormone is central in triggering off the germination process in that upon imbibition of the seed and in the presence of other environmental cues such as favourable light conditions - gibberellin (GA) has the power to bring about the degradation of RGL2. Hence the arrest of ABA production, and the resulting capacity for the embryo to blossom. So to speak... Seed germination is therefore governed by a subtle balance between the presence of GA and ABA. With RGL2 in the midst of the two.

RGL2 is, in fact, a negative regulator of seed germination. The exact mechanism involved, however, is unknown. Perhaps it is able to repress the expression of hydrolysing enzymes, whose activity is released thanks to gibberellin. But it may also be able to prevent germination via other pathways. Only time and research will tell. One interesting fact is that it has become obvious that plant growth is not simply a case of endogenous factors responding to environmental cues, but that signalling systems such as the GA/RGL2/ABA system also interact with outside signals – giving a sense of "dialogue" to the notion of seed germination.

As always, no pathway can be narrowed down to one, two or even only three actors. Any developmental pathway is complex and involves many variables. What is certain is that DELLA protein RGL2 does seem to have more than just a passive role in the dosage of GA and ABA levels, and hence seed germination or dormancy. We are miles away from the former theory whereby it was thought that the outer "dead" layer of the coat simply formed a barrier to water and/or oxygen, or exerted a mechanical restraint on a growing embryo. Needless to say, such research is of great interest to farmers who seek to modify the time of germination for instance. Many questions remain, least of which the control of RGL2 expression. How is it abolished when germination begins? So many questions, which are not so far removed from those that surround the mystery of childbirth... What is the nature of the molecular dialogues that cause a foetus to decide it is time to move on?

Cross-references to UniProt

DELLA protein RGL2, Arabidopsis thaliana (Mouse-ear cress) : Q8GXW1

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