clarified that the **terminator** itself was not under the operator/repressor control (but only developmentally regulated) whereas *recombinase* was the one that was controlled by the operator/repressor system. He may well be right but my intentions were: (i) to overemphasize the **regulation** of *terminator* expression by invoking the multiple mechanisms, viz. operator/repressor mediated interaction as well as developmental regulation together with the *crellox* technology, and (ii) to establish that increased expression of the gene can be achieved by providing tandem repressor-binding sites close to the pro- (because in this case the repressor was converted to function as an activator). Therefore, at this point it may be desirable to provide further clarifications to the more specialized readers regarding the molecular mechanisms of control utilized in the plant cell.

First of all, the 'oversimplified' model that I had presented was based on the prokaryotic systems (bacteria) where a simple 'repressor' protein binding to the control locus 'operator' provides a steric block to the transcription machinery and prevents the expression of the gene. Such simple mechanisms are not exactly the ones operative in eukaryotic organisms (both plants and animals). In these systems, the genomic DNA is organized as chromatin (bound to several proteins) and therefore, it stays generally in a repressed state. The expression of a gene takes place only consequent to activation. Obviously, the 'catch' is to get the activators to the promoter site where the transcription process initiates. This is generally achieved by providing appropriate binding sites for the activator on the DNA close to the promoter. In fact one can convert a 'repressor' into an 'activator' by 'fusing' the activator domain of a eukaryotic transcription activator (classic example, the HSV–VP16 activator domain fused to specific DNA binding domain). Such a fused molecule will now function as an activator rather than a repressor for a given gene, if the repressor binding sites are provided in *cis* to the promoter. Thus, for instance[1–3], if the 'operator' element of the tetracyclin resistance operon (originally derived from the bacterial transposon, Tn10) is located in proximity to the promoter of a gene, its expression can be turned on by providing the fused tetracyclin repressor–VP16 activator protein in *trans*. Once tetracyclin is added to this system, the repressor–VP16 protein falls off the operator locus and the transcription switches off. Further sophistication of this control can be achieved by converting the tetracyclin repressor (by mutations) such that it binds to the operator only in the presence of the antibiotic, i behaviour diametrically opposite to that of the parental molecule. In this case, only on addition of tetracyclin does the repressor–VP16 bind to the *cis* element and activate transcription from the target gene (see references for details). As mentioned previously, the actual methodology for controlling the expression of the *terminator* in the plant cells therefore, is likely to be more complex than the oversimplified version presented earlier.

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