

## Method for enhancement of protein production by *Trichoderma reesei*

We have developed a new method for improvement of cellulase and hemicellulase production in *Trichoderma reesei* by overexpression of a developmental regulator.

### BACKGROUND

The plant cell wall consists of the  $\beta$ -(1,4)-linked glucose polymer cellulose, hemicellulose polysaccharides of varying composition, and lignin. The former two are formed at annual production rates of 7.2 and  $6 \times 10^{10}$  tons, respectively, and thus represent the largest reservoir of renewable carbon sources on earth which could be used for the production of biofuels and other biorefinery products, thereby replacing products derived from fossil carbon components. While chemical and enzymatic processes for the hydrolysis of these polymers are known, enzymatic hydrolysis is preferred because it produces no inhibitory by-products and is thus environmentally compatible. However, enzyme production is still one of the cost-intensive steps in plant biomass conversion, thus warranting novel approaches for further improvement.

### TECHNOLOGY

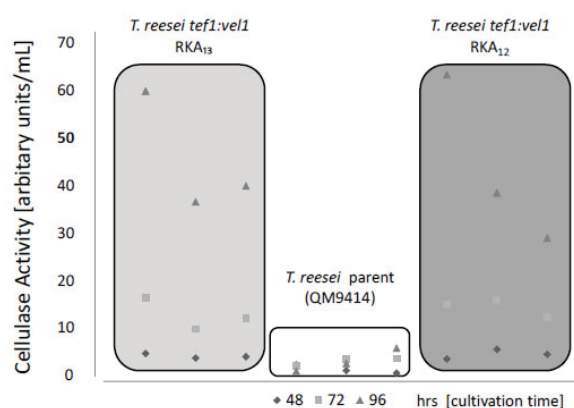
We have overexpressed the central regulator of fungal developmental-specific gene expression - VELVET (VEL1) - in *Trichoderma reesei*, the industrial producer of cellulases and hemicellulases to increase their production.

Table 1. Increase in xylanase and  $\beta$ -xylosidase production by *vel1* overexpression

	substrate	QM 9414	<i>vel1OE</i>
$\beta$ -xylosidase	cellulose	0.145 [ $\pm$ 0.07]	0.334 [ $\pm$ 0.08]
	xylan	0.580 [ $\pm$ 0.09]	1.30 [ $\pm$ 0.10]
xylanase	cellulose	0.36 [ $\pm$ 0.03]	0.487 [ $\pm$ 0.06]
	xylan	0.33 [ $\pm$ 0.03]	0.430 [ $\pm$ 0.07]

Cultivation time 96 hours

Figure 1. Increase in cellulase formation by *vel1* overexpression



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### ADVANTAGES

VEL1 occupies a central position of regulation and thus increases the formation of all cellulases and hemicellulases. In addition, it does not interfere with induction of these enzymes and further improvement can therefore be reached by simultaneous overexpression of transcription factors such as XYR1.