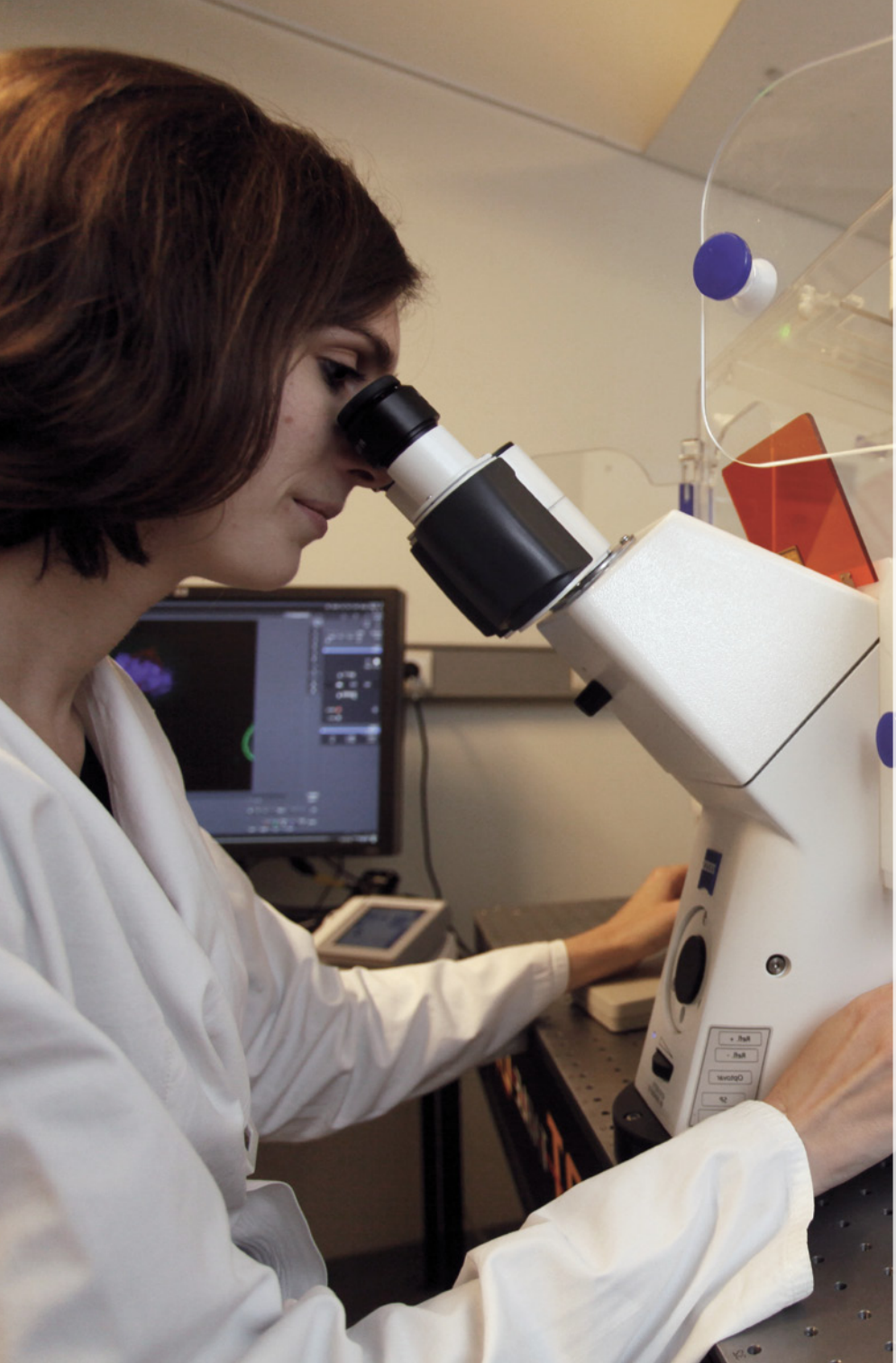

BIOPARK
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news

The Biopark Charleroi Brussels South Newsletter

n°19 — fall 2013

Regional development

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In brief

DISCOVERY OF NEW RIBOSOME ASSEMBLY FACTORS

Ribosomes are the cell's *workers*: these sophisticated molecular machines *read* the genetic information encoded on RNA, and puts amino acids together to build functional proteins. Because ribosomes are absolutely essential for gene expression, disruption of their synthesis irreparably leads to severe diseases. In fact, a new class of human diseases has recently been discovered and designated as *ribosome diseases* or *ribosomopathies*.

In a recent issue of *Molecular Cell,* the **RNA Metabolism Laboratory (IBMM)** has tested directly in human cells the involvement in ribosome biogenesis of 625 nucleolar proteins. The team led by **Prof Denis Lafontaine** also identifies 286 novel ribosome assembly factors, including 74 without yeast counterparts, yeast being the historical model used in ribosome biogenesis studies. The study also reveals that 38% of the ribosome assembly factors identified have been connected to severe human diseases, mainly cancer and genetic disorders. The study also showed that nearly one third of the human factors identified perform additional or even entirely different functions than their yeast homologs.

This conclusion is very important as it poses fundamental implications for understanding ribosomopathies at the molecular level and for developing effective therapeutic agents. This work provides a key reference dataset and fundamental resource to the scientific community and is available on the website www.ribogenesis.com.

N. J.

IMPROVED UNDERSTANDING OF CARDIAC HYPERTROPHY THANKS TO HIV

Understanding how latent reservoirs of the HIV-1 virus which causes AIDS are reactivated is a major step in improving how the virus is treated. Teams under **Carine Van Lint (IBMM)** and Olivier Rohr *(Université de Strasbourg)* have already shown that the cell factor CTIP2 played a role in forming these reservoirs. Studies have also shed light on the essential role played by another factor, the transcription elongation factor P-TEFb, in reactivating HIV from latent reservoirs. When its activity is inhibited, P-TEFb also contributes to cardiac hypertrophy.

In a paper recently published in *PNAS*, both teams have revealed a link between these factors: CTIP2 inhibits the activity of the P-TEFb complex by recruiting it, via the cell RNA (7SKsnRNA), to P-TEFb dependent gene promoters. A look at how these genes are expressed in the hypertrophied hearts of mice revealed how CTIP2 controls deregulated genes in the wake of disrupted P-TEFb activity.

This discovery unveiled a new mechanism for controlling this factor's activity. A better understanding of P-TEFb regulation is essential to developing new treatment strategies targeting diseases like AIDS and cardiac hypertrophy.

N. J.

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L'UNION EUROPEENNE ET LA WALLONIE INVESTISSENT DANS VOTRE AVENIR.

Quartely publication

Editor : Nathalie Gobbe • Editorial Staff: Bruno André, Christelle De Beys, Dominique Demonté, Damiano Di Stazio, Natacha Jordens, Véronique Kruys, Frédérique Margraff, Arnaud Termonia

Sub-editor : Nancy Dath • Photos : Bruno FAHY (partim) • Lay-out : Céline Kerpelt I Curlie • Printing : Paragraph

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