VICZIAN Kinga (CNECT)

From: RATOI Adina (CNECT)
Sent: 16 January 2014 15:10
To: VICZIAN Kinga (CNECT)
Subject: FW: [Synergy-COPD] update on submitted publications -NATURE, Genome Medicine, J.Applied Physiology- (University of Liverpool)

Follow Up Flag: Follow up
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Hi Kinga,

Is there a chance you could update PPM with this info?

Thanks in advance,

Adina

From: Mària Sánchez [mailto:msanchez@bdigital.org]
Sent: Tuesday, January 07, 2014 9:51 AM
To: RATOI Adina (CNECT)
Cc: CNECT-ICT-270086; Magí Lluch-Ariet; Felip Miralles
Subject: [Synergy-COPD] update on submitted publications -NATURE, Genome Medicine, J.Applied Physiology- (University of Liverpool)

Dear Adina, hope you have had nice holidays, receive again our best wishes for the New Year starting.

Relating to **the submitted article to Nature** ("*Insulin regulated translation drives an adaptive metabolic and transcriptional signature")*, be please informed that **the paper has been sent to referees**. This is a rather positive thing considering that the majority of submitted manuscripts are not even sent out for review.

Added to this dissemination action, our partner University of Liverpool **has recently submitted another paper to Genome Medicine** which acknowledges Synergy-COPD project. Find please the detail of the intended publication in the below notice box ("*A systems biology approach reveals a link between systemic cytokines and skeletal muscle energy metabolism in a rodent smoking model and human COPD")*.

Moreover, **an additional Synergy-COPD publication is expected to be submitted in 4 weeks,** at the latest and it will be a Nature Communications paper. Shortly after that there will be a J. Applied Physiology review. We'll keep you duly posted.

Kind regards form Barcelona,

Author/s	**DAVIDSEN P K et al. (**Corresponding author : **Dr. Francesco Falciani)**
Title	***A systems biology approach reveals a link between systemic cytokines and skeletal muscle energy metabolism in a rodent smoking model and human COPD***
Format	Article
Abstract	BACKGROUND: A relatively large percentage of patients with chronic obstructive pulmonary dis (COPD) develop systemic comorbidities that affect prognosis, among which muscle wasting is pa debilitating. Despite significant research effort, the pathophysiology of this important extrapulmor manifestation is still unclear. A key question that remains unanswered is to what extent systemic inflammatory mediators might play a role in this pathology. Cigarette smoke (CS) is the main risk factor for developing COPD and therefore animal models c exposed to CS have been proposed for mechanistic studies and biomarker discovery. Although r been successfully used as a pre-clinical in vivo model to study the pulmonary effects of acute an CS exposure, data suggest that they may be inadequate models for studying the effects of CS o peripheral muscle function. In contrast, recent findings indicate that the guinea pig model (Cavia

	may better mimic muscle wasting. METHODS: We have used a systems biology approach to compare the transcriptional profile of l skeletal muscles from two rodent models exposed to CS (guinea pigs and C57 mice) to COPD p with muscle wasting. RESULTS: We show that guinea pigs exposed to long-term CS accurately reflect the transcriptio changes observed in dysfunctional limb muscle of severe COPD patients when compared to mat controls. Using network inference, we could then show that the expression profile in whole lung c encoding for soluble inflammatory mediators is informative of the molecular state of skeletal mus guinea pig smoking model. Finally, we show that CXCL10 and CXCL9, two of the candidate syst cytokines identified using this pre-clinical model, are indeed detected at significantly higher levels of COPD patients, and that their serum protein level is inversely correlated with the expression o energy metabolismgenes in skeletal muscle. CONCLUSIONS: We conclude that CXCL10 and CXCL9 are promising candidate inflammatory s linked to the regulation of central metabolism genes in skeletal muscles. On a methodological lev work also shows that a system level analysis of animal models of diseases can be very effective generate clinically relevant hypothesis.
Event / Publication	**Genome Medicine** (http://www.genomemedicine.com/)
Expected date	TBD

Mària SANCHEZ

Project Manager
Project Management Office

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De: Adina.RATOI@ec.europa.eu [mailto:Adina.RATOI@ec.europa.eu]
Enviado el: jueves, 05 de diciembre de 2013 15:24
Para: msanchez@bdigital.org
CC: CNECT-ICT-270086@ec.europa.eu
Asunto: RE: [Synergy-COPD] submitted publication to NATURE (University of Liverpool)

Thank you, Mària. Please let me know if/when it is accepted.

Best regards,

Adina

From: Mària Sánchez [mailto:msanchez@bdigital.org]
Sent: Wednesday, December 04, 2013 8:52 AM
To: RATOI Adina (CNECT)
Cc: ROESEMS-KERREMANS Gisele (CNECT); CNECT-ICT-270086
Subject: [Synergy-COPD] submitted publication to NATURE (University of Liverpool)

Dear Adina,

Relating to Synergy-COPD project, we are pleased to inform you about the following **SUBMITTED** publication issued from the project Consortium (the University of Liverpool co-authoring):

Author/s	**BRINA D et al.**
Title	***Insulin regulated translation drives an adaptive metabolic and transcriptional s***
Format	Letter
Abstract	Insulin generates a multiply branched response whose ultimate function is to regulate pr levels in living organisms. Insulin induces a burst in bulk translation, whose significance i we show that the role of insulin-induced translation is to coordinate a long-term metaboli with reduced eIF6 (eukaryotic Initation Factor 6) fail to upregulate translational rate upon administration2, hinting at insulin resistance. Surprisingly, mice with impaired insulin-reg due to eIF6 depletion switch to a metabolic status characterized by lower glycemia. Redu glucose levels are accompanied by cell-autonomous metabolic changes. Primary hepatc deficient mice have a reduction in glycolysis, fatty acid synthesis and ATP levels, without AMPK. Acute rescue of eIF6 restores ATP levels. The metabolic switch induced by the la is accompanied by a change in steady-state levels of mRNAs involved in fatty acid and synthesis, suggesting a reorganization of transcription. Gene network analysis demonstr induced transcriptional changes are restricted to insulin-responsive tissues, and are as p induced by chromatin remodellers. Restoration of eIF6 rescues steady-state levels of Fa enzyme for fatty acid synthesis. An HTS screening for eIF6 mediators identifies transcrip involved in lipogenesis regulated at the translation level. We propose that, in vivo, transla upstream of transcription in setting a metabolic signature adapted with environmental clu resembles a metabolic learning.
Event / Publication	**Nature (**http://www.nature.com/)
Expected date	TBD

Please, do not hesitate to contact us for further details you may need.

Kind regards,

Mària SANCHEZ

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