

University of Groningen

Beyond genome wide association studies in celiac disease by exploring the non-coding genome

de Almeida, Rodrigo

IMPORTANT NOTE: You are advised to consult the publisher's version (publisher's PDF) if you wish to cite from it. Please check the document version below.

Document Version

Publisher's PDF, also known as Version of record

Publication date:

2015

[Link to publication in University of Groningen/UMCG research database](#)

Citation for published version (APA):

de Almeida, R. (2015). *Beyond genome wide association studies in celiac disease by exploring the non-coding genome*. [Thesis fully internal (DIV), University of Groningen]. [S.n.].

Copyright

Other than for strictly personal use, it is not permitted to download or to forward/distribute the text or part of it without the consent of the author(s) and/or copyright holder(s), unless the work is under an open content license (like Creative Commons).

The publication may also be distributed here under the terms of Article 25fa of the Dutch Copyright Act, indicated by the "Taverne" license. More information can be found on the University of Groningen website: <https://www.rug.nl/library/open-access/self-archiving-pure/taverne-amendment>.

Take-down policy

If you believe that this document breaches copyright please contact us providing details, and we will remove access to the work immediately and investigate your claim.

Downloaded from the University of Groningen/UMCG research database (Pure): <http://www.rug.nl/research/portal>. For technical reasons the number of authors shown on this cover page is limited to 10 maximum.

**Stellingen
behorende bij het proefschrift:**

“Beyond genome-wide association studies in celiac disease by exploring the non-coding genome”

Rodrigo Coutinho de Almeida

1. Although many closely correlated single nucleotide polymorphisms (SNPs) resulting in linkage disequilibrium aid tremendously in the detection of disease association, they seriously hamper pinpointing the casual disease SNP or SNPs. (*this thesis*)
2. Even if a SNP maps within a gene, that gene is not necessarily the disease-causing gene. (*this thesis & Smemo et al., Nature. 2014; 507(7492):371-5*)
3. The explosion of publicly available, annotated genomic data enables the design of integrative approaches to efficiently prioritize causal SNPs and genes. (*this thesis & ENCODE Project Consortium, Nature. 2012; 489(7414): 57-74*)
4. Only 5% of the celiac disease-associated SNPs are located in coding sequences, strongly suggesting that many of the celiac disease-associated variants affect gene regulation. (*this thesis*)
5. Circulating micro RNAs can be potential biomarkers for early celiac disease detection, for monitoring disease progression, and for monitoring the effectiveness of and adherence to dietary intervention in celiac disease. (*this thesis*)
6. Disease-associated SNPs can affect the binding capacity of micro RNAs. (*Sethupathy P. & Collins F.S., Trends Genet. 2008; 24:489-97*)
7. “It is increasingly apparent that the functional properties of a genetic polymorphism (usually a SNP) depend upon the cell type analysed.” *Benjamin Fairfax*
8. “The most surprising discovery about the human genome was that the majority of the functional sequence does not encode proteins.” *Eric S. Lander*
9. “Learning never exhausts the mind.” *Leonardo da Vinci*
10. “The secret of getting ahead is getting started.” *Mark Twain*
11. “If it looks like a duck, and quacks like a duck, we have at least to consider the possibility that we have a small aquatic bird of the family Anatidae on our hands.” *Douglas Adams*
12. “I am so clever that sometimes I don’t understand a single word of what I am saying.” *Oscar Wilde*