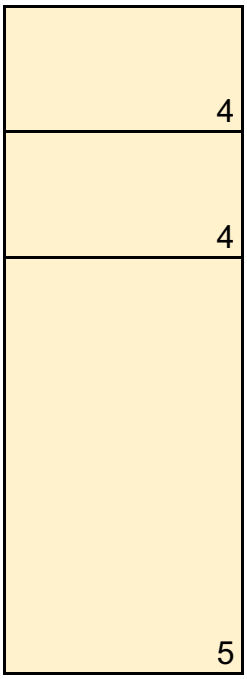
Project	Students	Teacher
Achondroplasia	V,V	JP
Anosmia in Kallmann Syndrome	K,T	KA
Adenomatous polyposis coli (APC)	E,E	SO
Hypohidrotic ectodermal dysplasia (HED)	A,M	VV,BK
Situs inversus in Kartagener's syndrome	A,U	JP

Scoliosis	K,K	YC
Muscle atrophy or hypertrophy	A,J	SK
Amyotrophic lateral sclerosis (ALS)	M,R	SG

Evaluator 1	Evaluator 2	Evaluator 1 grade	Evaluator 2 grade
JP	KA	4,5	4,5
KA	JP	4,3	4
SO	KA	5	5
VV, BK	KA	3,5	3
JP	KA	4,5	5

YC	JP	4	4,5
SK	JP	4	4
SG	JP	4,5	4,5

Presentation
5
5
5
5
5


4
4
5

Comments/ feedback to students: guiding teacher
Simple, straight-forward experiment, very relevant biological question, detailed analysis with good understanding of biology, relevant discussion. The graphical abstract could have emphasized the research questions (rather than methods)
The background and the research question are very well presented. The experiments address the research questions, but could in in some cases be explained in more detail. There is a great discussion of the expected results, impact, and the strengts and the weaknesses of the research plan.
The experiment is timely and relevant and relies on innovative and advanced methodologies. The first experiment is feasible and realistic (if the lentiviral expression works) and likely to give relevant information about the WNT activity in the M4 mutant. The second experiment is elegant and relies on state-of-art technologies. However if the M4 mutant behaves similarly to WT, it remains unclear if the mutations lead to differences in actin nucleation in intestinal epithelual cells, or if the defect is not sufficient to cause a phenotype in this assay. Finally, the in vivo experiment is very interesting but also carries the risk that ApcM4 mutant is not sufficient to cause tumorigenesis due to lack of WNT signaling even if in vivo the cytoskeletal effects could complement the WNT activation. That part is nicely addressed in the discussion. The writing shows maturity and understanding of the research topic and a variety of experimental models. Some questions: What are m1, m2 and m3 mutants (Fig 1)? Are the m4 mutation sites conserved between mice and humans? What would be the potential impact of the findings beyond the deeper mechanistic understanding?

Formulation of the hypothesis about the role of Wnt10a in ED is skilled and reasonable but written work would benefit from explicating the research question with the help of the hypothesis, keeping the disease approach in mind. The choice of target organ is justified and well described but disease phenotype especially in the chosen target organ would need more attention. Planned experiments have in most part logical succession from validation experiments to functional relevance in ED and the controls are adequately described. Some discontinuities in the experiment and planned analysis are present and justification for the use of TNF-A in explant culture would benefit the written work greatly, because it could be discussed also in discussion. The discussion is very focused on the results of individual studies. Its strong point is thorough consideration of the potential outcomes but it would benefit from synthesis to fulfill the purpose of discussion in this kind of text. Considerations about limitations and alternatives regarding the chosen experimental approaches would be useful. There are some statements of relevance in discussion but their expansion would be beneficial. Writing is good.

A clear and logical plan. You could have explained the type of DNAH5 mutations in the Kartagener's syndrome (recessive/dominant), as this has an impact to your genetic model. Graphical abstract very good!

analyses before functional studies in the Zebrafish. The caveats are discussed well. A summarizing graphical abstract is missing, but the experimental strategy is shown in the figures.

The students present a logical project with well defined goals and overall strong writing. The introduction would have benefited from a clearer explanation of the correlation between mitochondrial dysfunction and neuronal pathogenesis, which would strengthen the rationale for examining the role of this organelle in glial cells (Schwann cells). However, aside from this minor point, the text was well structured, and the research hypothesis was clearly stated.
The experimental design and methods are clearly described, and the justification for the chosen approach is logical and well supported.
The discussion is strong, addressing key potential caveats and offering a thoughtful interpretation of the potential impact of the project.

Comments/ feedback to students: JP, KA

The choice of the experimental system could be better justified, weighing pros and cons of some alternative model systems.

In the conditinal mutagenesis experiments, neuronally expressed Dcx-Cre is used to inactivate Fezf1 and Sema3. However, it is stated that Sema3 is expressed in mesenchymal (non-neuronal) cells.

The background and the research questions are clearly presented, as well as the strengths and weaknessess of the resarch plan. The expected results are thoughtfully considered, but the description of the experiments and controls is somewhat difficult to follow. The method of Wnt target gene analysis (qPCR) is only shown in graphical abstract and not metioned in the text.

The overall understanding of the experimental system and the idea behind this research plan seems to be good and also have some novelty. However in the presented plan, the description of the HED phenotype is superficial, without describing the key features of the disease in human. The reserach hypothesis is evident from text but is formally stated only in the graphical abstract and not in the research plan text. There are some unclarities about the used samples and controls: for example what is the control in the ISH used for, or the description of LacZ reporter for in vivo monitoring of Wnt10a transcription - lacZ reporter requires fixation for the visualization making for example time lapse imaging impossible. The genetic features of the tabby mouse strain is not explained. The discussion is interesting, but it is focused only on the expected experimental results. Would have liked to see some more discussion of the strengths and weaknesses of the used experimental system, and the impact of this study.

summarizes the whole research project very nicely. The experiments address the research question, however the explanation of how the second research question is addressed remains somewhat unclear. The choice of experimental

contributes to the development of idiopathic scoliosis in ptk7 mutant zebrafish. Its goal is to clarify the molecular mechanisms involving Ptk7 and identify which Wnt pathway dysregulation is key to understanding and
a logical and clear plan, written well and easy to understand. The benefits, challenges and alternatives of using ASOs could have been described in more detail. Analysis methods well-thought. Graphical abstract very good.

Nice plan with a clear focus and logic.The graphical abstract could have emphasized the hypothesis instead of methods. Good discussion on the caveats.

Final grade
4,666666667
4,433333333
5
3,833333333
4,833333333

4,166666667
4
4,666666667

Methods in Functional Genetics and Development (GMB-304) (GMB-304)

Etunimi	Sukunimi	Opiskelijanu Laitos	Osasto	Sähköpostio
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Average of (T	Tehtävä:Test	Tehtävä:Test	Tehtävä:Test:	Tehtävä:Test L	Tehtävä:Res	Final
3,9875	3	5	4,2	3,75	4	**3,99375**
4,45	5	5	4,3	3,5	4	**4,225**
4,2375	4,5	4	4,2	4,25	4,4	**4,31875**
4,225	3	5	4,9	4	4,2	**4,2125**
4,25	4	4	4,5	4,5	4,70	**4,475**
4,675	5	5	3,7	5	5	**4,8375**
4,5	4,5	5	5	3,5	4,70	**4,6**
4,625	5	5	4,5	4	4,2	**4,4125**
4,475	4	5	4,4	4,5	5	**4,7375**
4,225	3,5	5	4,4	4	4,7	**4,4625**
4,825	5	5	4,8	4,5	4,7	**4,7625**
4,75	5	5	5	4	4,8	**4,775**
4,2125	4,5	5	3,6	3,75	4,4	**4,30625**
4,6	3,5	5	4,9	5	3,8	**4,2**
4,375	5	5	5	2,5	4,8	**4,5875**
4,55	4,5	5	4,7	4	3,8	**4,175**

Final 0-5	
4	3,99375
4	4,225
4	4,31875
4	4,2125
5	4,475
5	4,8375
5	4,6
5	4,4125
5	4,7375
5	4,4625
5	4,7625
5	4,775
4	4,30625
4	4,2
5	4,5875
4	4,175

Methods in Functional Genetics and Development (GMB-304) (GMB-304)

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Urte	Smigelskaite	015367521		urte.smigelsk	4,67
Mahsa	Vosoughifar	021565281		mahsa.vosou	4,34

Tehtävä:Test	Tehtävä:Test	Tehtävä:Test	Tehtävä:Test	Tehtävä:Res	Viimeksi ladattu tältä kurs
3	5	4,2	3,75	4	1772795438
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5	5	5	4	4,8	1772795438
4,5	5	3,6	3,75	4,4	1772795438
3,5	5	4,9	5	3,8	1772795438
5	5	5	2,5	4,8	1772795438
4,5	5	4,7	4	3,8	1772795438

ssilta