Logbook

Science Fair

Ivan

Sept. 14 On Sept. 14 I:
- decided I wanted to be a part of science fair
- bought the logbook and binder

Sept. 18 On Sept. 18 I:
- got introduced to the science fair at Webber
- met my mentor
- decided on which type of project I would work on (study)
- Narrowed my topic down to a field: Biochemistry

Sept 22 On Sept 22 I:
- Looked at interesting topics in biochemistry and found:
  → MicroRNAs in Cancer
  → CRISPR for studying the relationship between genes for allergies and the enviroment

Sept 26 On Sept 26 I:

- Decided officially what my topic would be:
  → microRNAs in Cancer
- Learned useful websites for research
  → NIH.gov
  → pubmed
  → Sci.hub... Use DOI # from pubmed

Oct. 4 On Oct. 4 I:
- Found around five papers about the topic and I printed one out

Oct. 7 On Oct. 7th I:
- Found two more papers that I can use in the future
- Decided on what question I am going to answer (Can microRNA be used to cure

Oct. 10 On Oct. 10 I:
- Looked at other CYSF projects to see what other people did
- Jessica thought my question:
  → Does microRNA have any role in cancer and how microRNA be used in the future to cure cancer
  was good

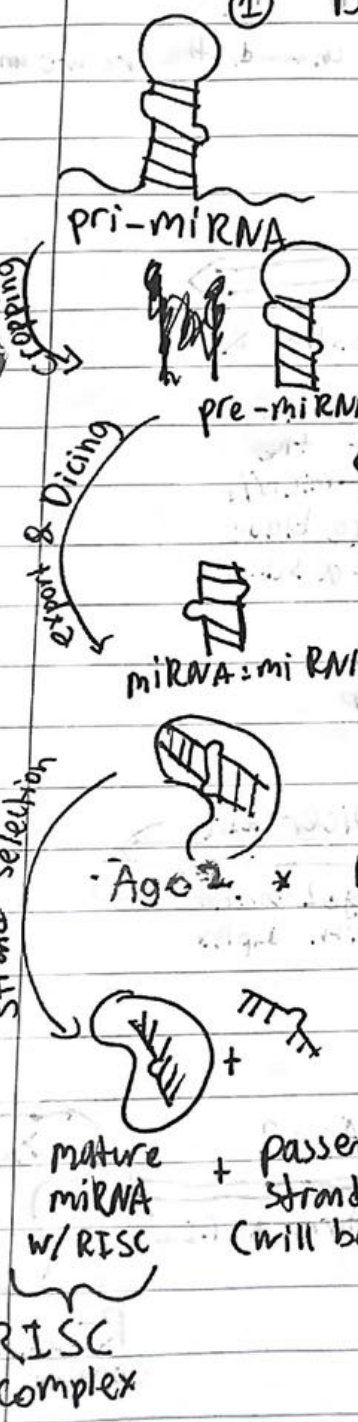
Oct 12 On Oct. 12 I:
- Printed my first paper by the NIH on microRNA in cancer

Oct 17 On Oct 17 I:

→ Read and annotated "Micro RNAs in Cancer" by the NIH.

Important Info in "Micro RNAs in Cancer"

① Biogenisis of miRNA



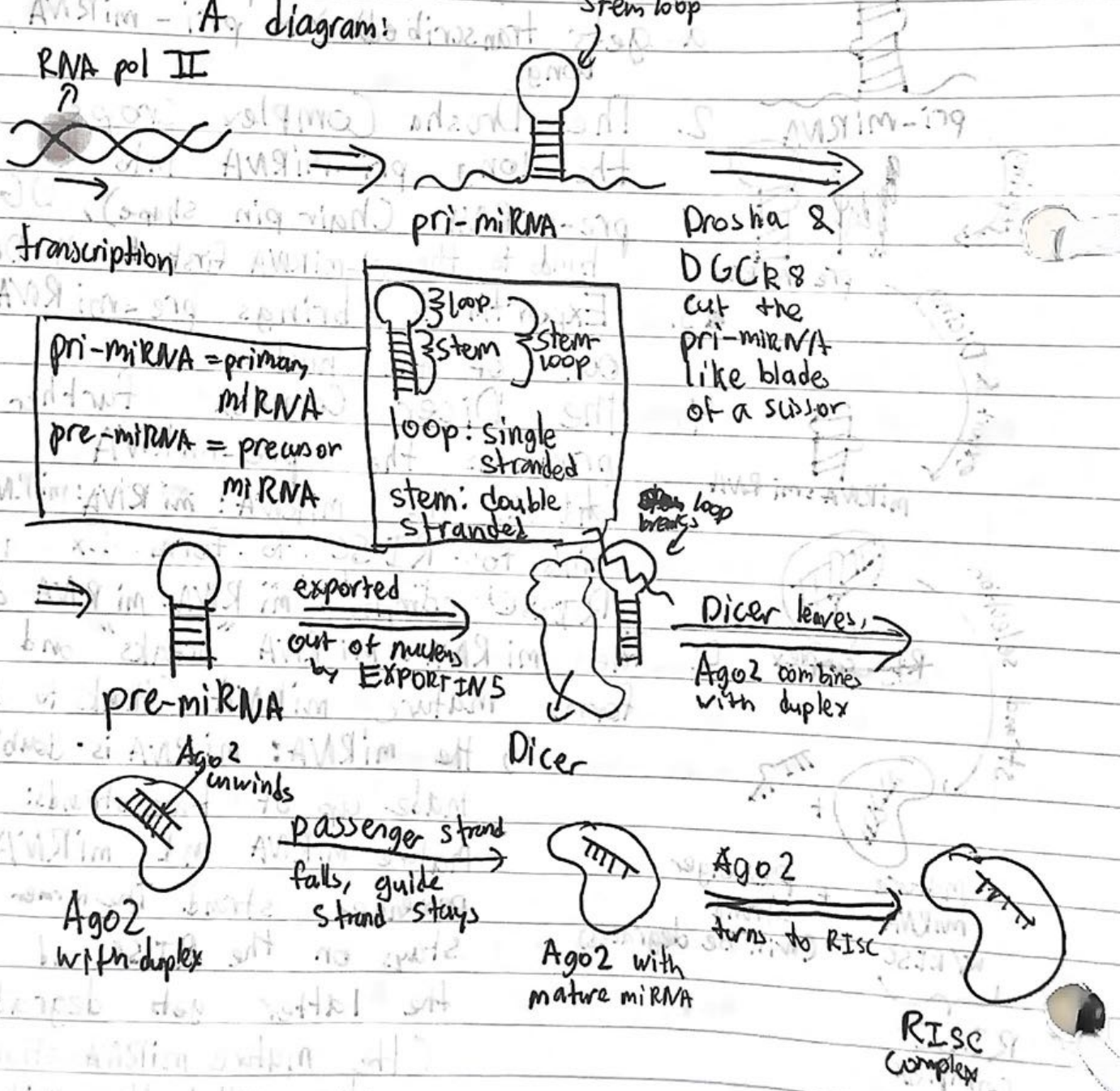
1. RNA Polymerase II (RNA pol II) transcribes a non coding gene (long) into 'pri-miRNA, also long.
2. The Drosha Complex crops the long pri-miRNA into pre-miRNA (hair pin shape), DGCR8 binds to the pri-miRNA first to call Drosha.
3. Exportin-5 brings pre-miRNA out of the nucleus.
4. The Dicer Complex further processes the pre-miRNA to form double stranded miRNA:miRNA duplex. Ago2 attatches to the duplex and unwinds the duplex.
5. The miRNA:miRNA "breaks" and forms mature miRNA (thanks to Ago 2)
   - ↳ the miRNA:miRNA is double stranded & made up of two strands: mature miRNA and miRNA passenger strand. The former stays on the Ago2 and the latter gets degraded (the mature miRNA strand is also called the guide strand

6. By now, we have Ago 2 attatched to the guide strand. Ago2, the guide strand and some proteins combine to form RISC

↳ RNA Induced Silencing Complex

* In this diagram I show the early stages of the miRNA (pri-, pre-, duplex) as unwound, they are wounded.

A diagram:



② Over view on how miRNA stops mRNA

1. miRNA have flexible base pairing
   1 miRNA can bond to tens to hundreds of mRNAs
2. Once bound either:
   → RISC cuts the mRNA which will be degraded by the cell
   → stay there to prevent RNA polymerase from binding
   Binds in 3'UTR (untranslated region)

③ Important miRNAs for my purpose

miRNAs can be oncogenes or tumor surpressors

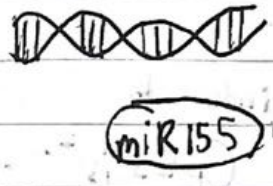
1. miR-21 (Oncogene)
   → Frequently found to be over-expressed in cancer
   → targets multiple tumor surpressors
   → increased expression of miR-21 results in increased proliferation (growth of cells by multiplying) and/or decreased apoptosis

oncogene - a gene coding for a cell cycle enhancer

tumor suppressor - anti-oncogene, discourages cell cycle

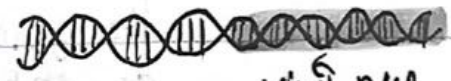
2. mi-155 (Oncogene)
   → Similar to miR21, an oncogene
   → highly expressed in tumors, promotes oncogenisis
   → Is coded for by the gene BIC, B-cell Integration cluster. (called B-cell ... since discovered in B-cells) with virus that causes lymphomas (lymph node cancer)

oncogenisis - formation of tumors



miR 155

Regular BIC gene with healthy amount of miR155



miR 155 | miR 155

miR155

miR155 miR155

too much miR155 leading to cancer

→ miR 155 targets FOXO3B, part of a family of proteins called FOXO, which controls cell cycle regulation (tumor surpressor)

→ Also targets the mismatch DNA repair (MMR) system. This corrects mistakes in base pairing. miR-155 breaks this leading to higher mutation rates called MSI (microsattellite instability)

→ high expression of miR-155 causes increased resistance to chemo therapy.

↳ Other oncogenes: miR-17-92 cluster, miR-372/miR-273 and miR-106b

↳ Some studies show miRNAs promote homeostasis

3. let-7 (tumor surpressor)

→ Frequently reduced expression in cancers

→ in a study let-7 was given to lung cancer and that reduced the growth

4. miR-43a

→ made more when p53 binds to the gene that makes it (p53 is an enhancer)

→ repressed in pancreatic carcinoma (cancer in pancreas)

→ controls cell cyle, DNA repair, apoptosis

5. miR-143, miR-145
   → both are repressed in colorectal cancer (end of intestine cancer) and prostate cancer
   → adding these back in reduces size of tumor

6. Some are either oncogenes or tumor surpressors based on context. Ex. miR-29a
   → acts as an oncogene or tumor surpressor based on context

End of Work Done on Oct. 17
pg. 4/14, 4 pages done.

Oct. 18

On oct. 18 I:
→ Continued annoting the paper "MicroRNAs in Cancer"

Sizes of: (nucleotides, nt)
→ pre-miRNA: 60-100nt
→ duplex & mature miRNA: 22 (approx) nt
→ pri-miRNA: thousands of nucleotides

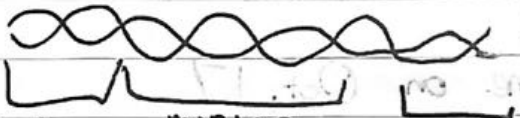
Additional notes on Synthesis of miRNAs
→ Along with Exportin 5, Ran-GTP also exports miRNAs out of the nucleus

Less is known about how the synthesis of miRNAs is regulated, but the deregulation of these systems cause cancer.

50% of miRNA: non protein coding RNA

40% of miRNA: derived from introns

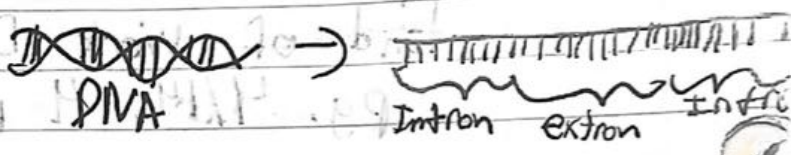
Many miRNA are coded as clusters

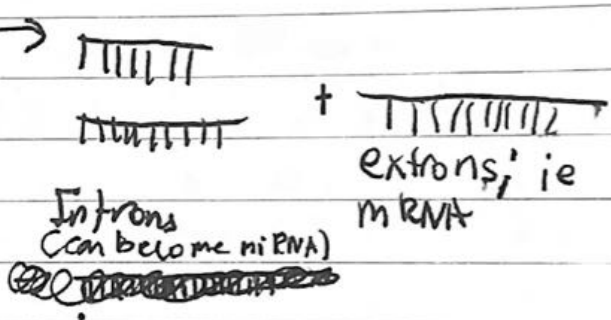


~2-9 hairpins

Clustered miRNAs can be transcribed at the same time

When DNA is ~~translated~~ transcripted the RNA produced as waste parts in it (introns). These are broken away from the RNA, the coding parts (extrons) stay to become the final mRNA





genes'

Many miRNAs are located where mutations in the DNA occur that cause cancer

④ Control and regulation of miRNAs

→ As seen in ③, miRNAs have a good part in cancer

→ according to the paper, "... it is plausible that restoration of normal miRNA expression in tumor cells could have therapeutic potential

RNA Pol II makes most miRNA, but RNA Pol III does some too

RNA Pol II adds a 7-methyl guanylate cap at 5' and poly (A) tail 3'
(bunch of adenines)

→ miRNA gene promoters are similar to mRNA gene promoters

→ The DNA binding factors that control miRNA transcription overlap with those that control mRNA transcription

→ The important ones are by c-myc & p53

→ c-myc

a) a proto-oncogene (supports oncogenes and nullifies tumor surpressors)
b) controls 10-15% of human genes
c) controls cell growth & apoptosis by activating or repressing transcription of ~~DNA~~ mRNA
d) also controls miRNA transcription by binding to E-boxes
e) c-myc controls the activation of the miR17-92 cluster which is often highly expressed in tumors since c-myc is highly expressed in tumors
f) this suggests that c-myc & miR-17-92 cluster both work together to promote cancer

↳ over expression of c-myc and miR-17-92 is known to give more aggressive tumors

↳ the miR-17-92 contains 6 miRNAs

the cell cycle regulator F2F1

f) c-myc also promotes E2F1 transcription showing how c-myc regulates both mRNA & miRNA to fine tune the cell cycle progression

g) c-myc also represses these tumor surpressors, including miR-15a, -29, -34, & let-7 families

h) When scientists put these miRNAs in a tumor, it decreased cell growth, this shows that a down regulation of these miRNAs is an important part of c-myc cancer

↳ c-myc regulates this through direct binding to promoters on miRNA

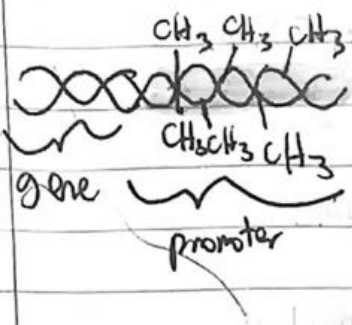
↳ c-myc increases transcription of Lin-28B, this represses the let-7 members

⑤ How We Control miRNAs

→ miRNAs are transcribed mainly by RNA Pol II, so the methods used to control mRNA transcribed by RNA Pol II also work well with miRNA

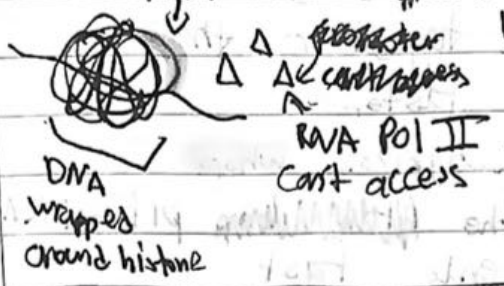
→ many tumor surpressors are under regulated since they are unaccesible since their promoter doesn't activate them

a) This happens since the promoter part of the gene is wrong. It's cytesines now have too many methyl groups making the promoter not work
↳ known as promoter hyper methylation



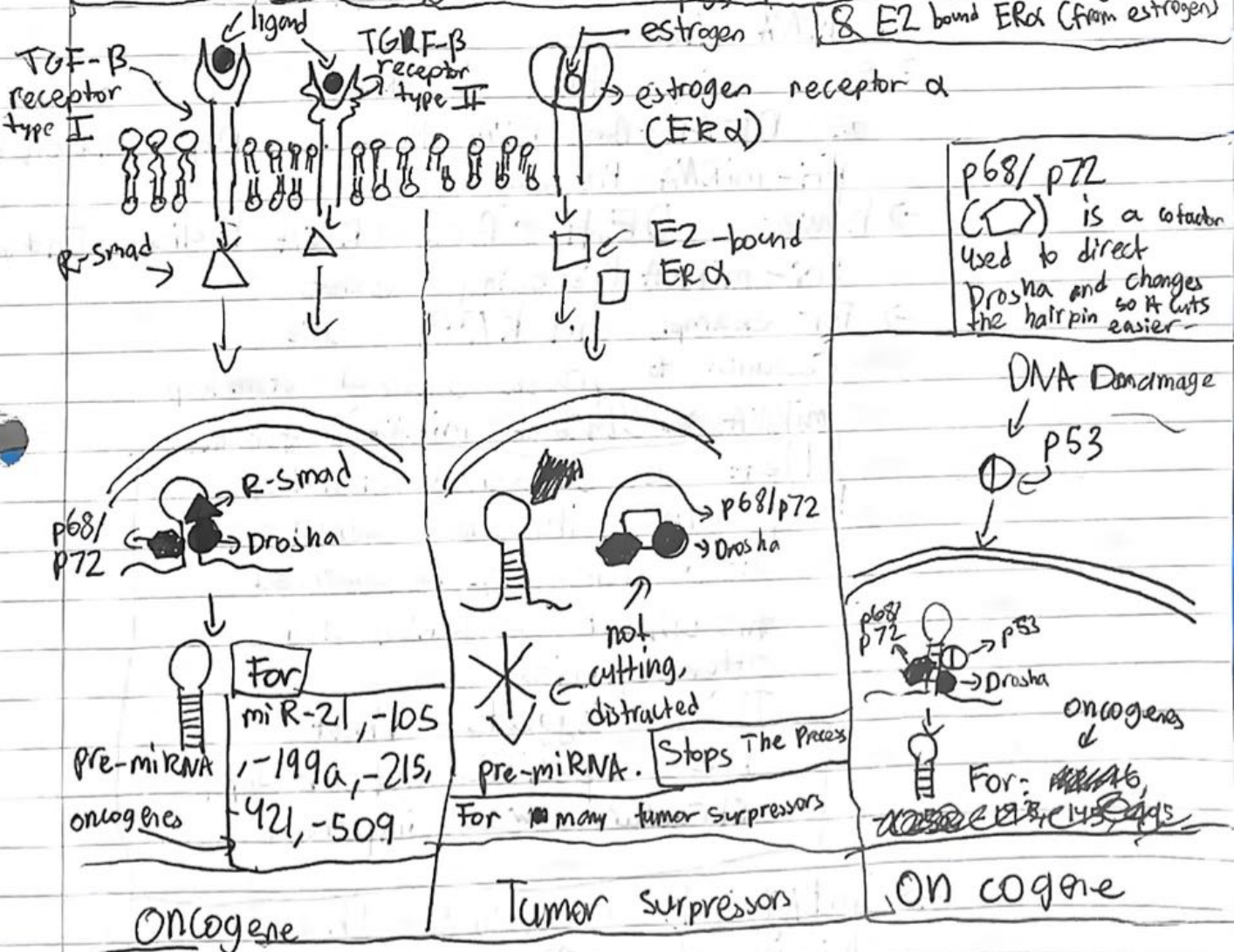
b) The miRNAs that get promoter hyper methylated are miR-127, -9-1 cluster, -193a, -137, -342, -203, -34 b/c, When these tumor surpressors get hyper methylated it can cause cancer

→ To fix this, HDACs are used (Histone deacetylase inhibitors) These unravel the DNA around the histones, making it more accesible to RNA Pol II
↳ This helps activate any miRNAs that have been silenced by histones
↳ ex. miR-1 gets silenced when the RNA Pol II can't reach the gene since the gene is wrapped too tightly around a histone. Treating it with HDAC will unravel the gene, the RNA Pol II will access the gene and miR-1 will be made



Key Point: disruption of miRNA biogenisis can alter the homeostasis of normal cellular biology

→ three important co-factors are R-Smad (from TGF-β), p53 (from DNA Damage) & E2 bound ERα (from estrogen)



End of work done on Oct. 18 & Oct. 19 pg. 8/15

Oct.20

On Oct. 20 I:

→ Continued annotating the paper
↳ The goal was 12/15 pages done

Cont. Annotating:

→ The methods above use p68/p72 These are known as DEAD-Box RNA Helicases

→ So, using these is known as DEAD-Box RNA Helicases - Dependent Pri-miRNA Processing

→ however, DEAD-Box RNA Helicase-Independent Pri-miRNA Processing exists

→ For example hn RNPs are required to process conserved stem loop miRNAs (~14% of miRNAs) for most

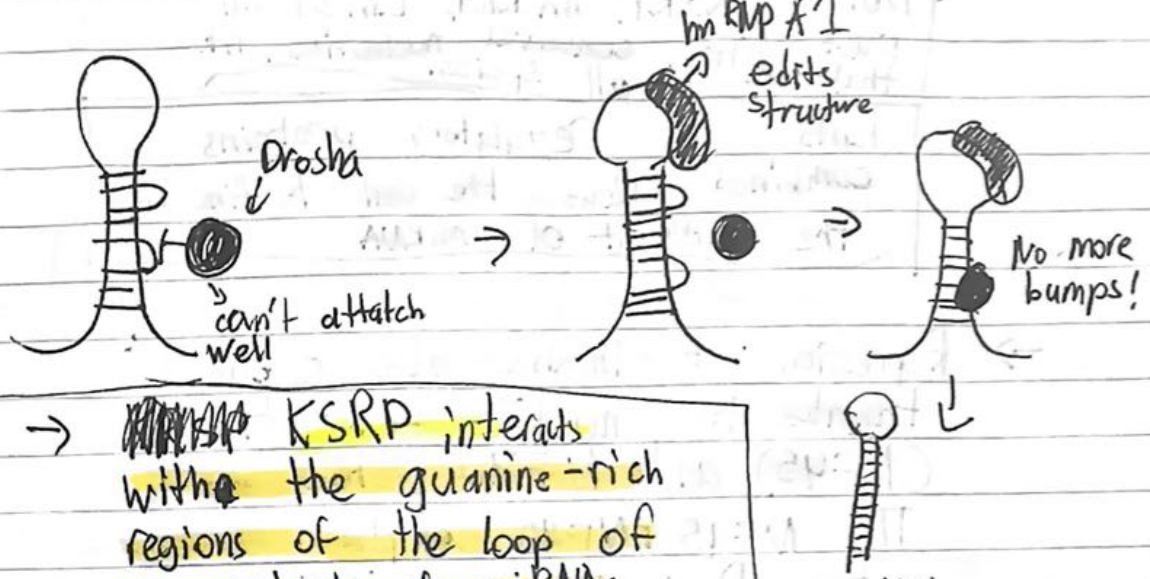
> Def: a conserved stem-loop is when the same structure of a stem loop is conserved throughout evolution and other species. This suggests that the specific stem-loop structure was important

→ hnRNP A1 re-adjusts the hairpin of miR-18a so Drosha can cut pri-miRNA-18a easier

→ The stem-loop structure of miR-18a is conserved, so the role of hn RNP must be very important for evolution to not change this

→ 14% of all miRNAs have conserved stem-loops this suggests that hn RNPs might have a very important role for these conserved

stem-loop miRNAs



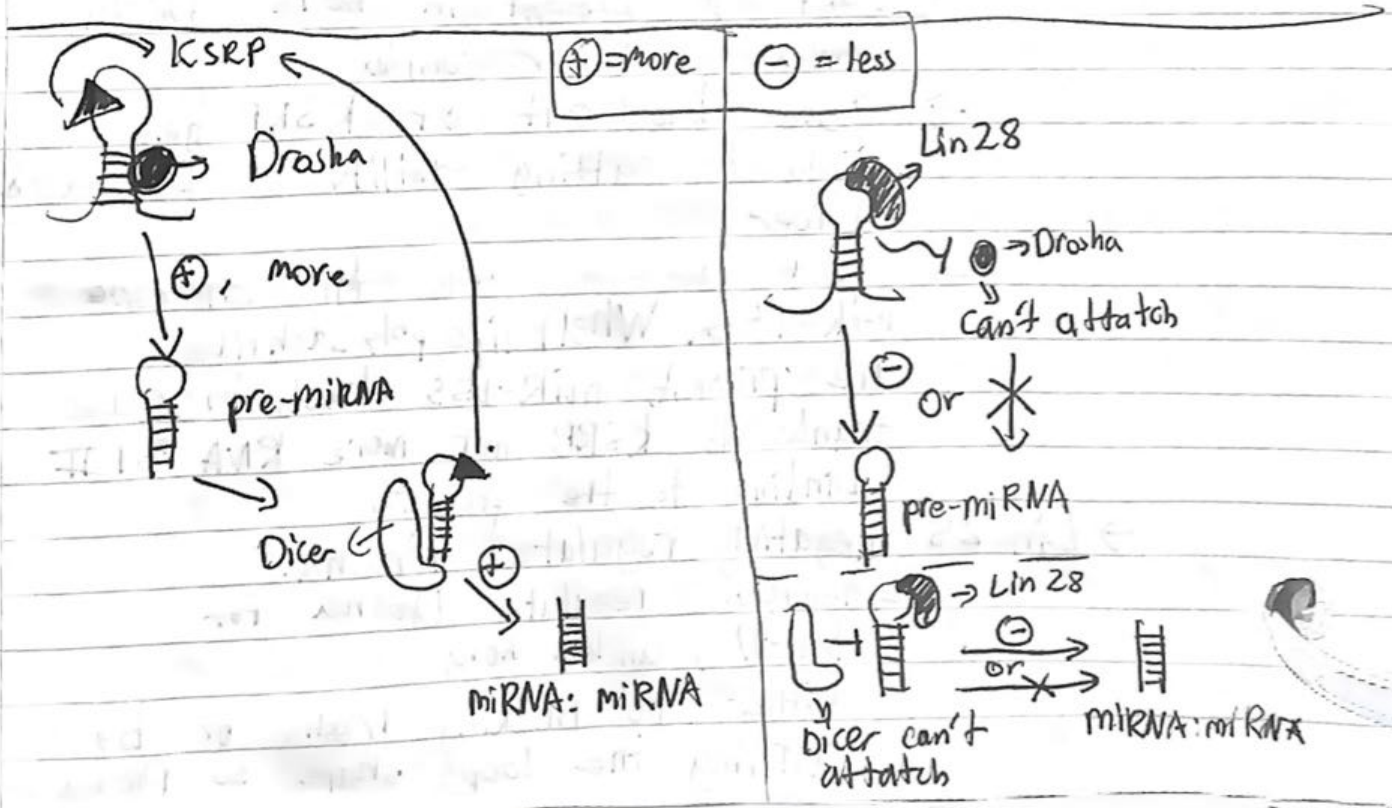
→ KSRP interacts with the guanine-rich regions of the loop of a subset of miRNAs to promote both Drosha and Dicer
  ↳ helps let-7a and miR-206 (tumor surpressors)
  ↳ without KSRP, the functions that let-7a & mir-206 have will be lost, causes increased proliferation (cells multiplying)
  ↳ KSRP is present in both Drosha complex & Dicer complex
  ↳ gene knockout of KSRP gene reduced cutting ability of Drosha & Dicer
  ↳ KSRP also controls the oncogene miR-155. When lipopolysachrides are present, miR-155 levels increase thanks to KSRP, not more RNA Pol II binding to the promoter

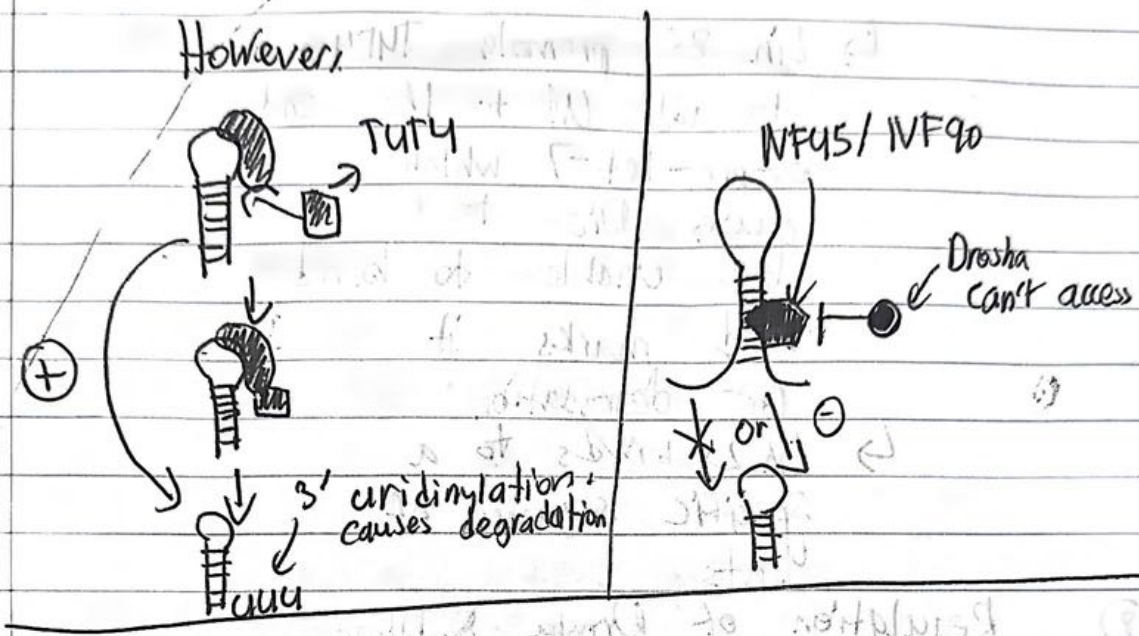
→ Lin 28 negativly regulates Drosha.
  ↳ negativly regulates Drosha for let-7, unclear how, either by blocking drosha or by altering the loop's shape so Drosha can't bind

Note: KSRP, hn RNP, Lin28 all react with conserved nucleotides, nts that are in all species.

Lots of regulatory proteins combined allows the cell to fine tune the amount of miRNA

→ Repression of Drosha also occurs thanks to nuclear factor 45 (NF45) and NF90 proteins
The NF45 / NF90 complex stops Drosha so pre-miRNA and mature miRNA isn't made
↳ Likes small miRNAs that are double stranded
↳ attatches before Drosha
↳ ~~the~~ Represses let-7 and also 3 other miRNAs





Oct. 21

On Oct. 21 I:

- continued annotating the paper
  - → goal: 13/15
  - → Exportin 5 works with GTP bound form of Ran,
  - → Dicer is an RNase type III protein

⑦ More on Dicer & RISC

- → Dicer present in nearly all eukaryotic cells (highly conserved)
- → has some helpers, TRBP & PACT
- → both help the stability of Dicer and it's speed
- → helpers with Dicer is called the RLC (RISC Loading Complex)
- → Dicer is very good at it's job, doesn't need enhancers, only ⊖ regulators
  - a) Ex. Lin28 (from DBRH-independent Pri-miRNA processing)
    - L> ceptoplasm is the primary location of Lin28-let-7 interaction

↳ Lin 28 promote TUTase to add Us to the end of pre-let-7 which causes Dicer to be unable to bind and marks it for degradation

↳ Lin 28 binds to a specific sequence of 4 nts

⑧ Regulation of Drosha & Dicer

↳ Increased Drosha's are observed in cancer

↳ 2-7 fold increase but only miR-31 seemed to increase a lot

⇒ DGCR8, when decreased minorily, also doesn't cause large decrease in miRNA

→ This happens because the two are in a feedback loop

↳ When less Drosha is present, Drosha cuts the mRNA that makes DGCR8 so that the levels can be balanced

↳ When less DGCR8 is present, it cause less Drosha to bind on pri-miRNA triggering the reduce of Drosha

↳ DGCR8 & Drosha need to be roughly equal for effectiveness

End of work done On Oct 22nd
pg. 13/15

Oct 22

On Oct. 22 I:
→ Tried to Finish annotating the paper
goal: 15/15

↳ altered expression of Dicer occur in cancer
a) Dicer is increased in prostate tumors
b) Dicer is decreased in non-small cell lung cancer
c) less Dicer → less prognosis (likelihood of normal life, symptoms becoming normal, not dieing)
d) Dicer levels also change based on cell type and stage of tumor
↳ ex. precursor of lung adenocarcinoma shows high Dicer levels, mature / advanced invasive tumor showed less Dicer levels

→ each cell has two copies of the Dicer gene Cone from each parent.
Losing one means you are hemizygous
Lose both: homozygous
a) Being hemizygous causes more cancer than being homozygous

b) The human cancer genome data supports this: more tumors happen from hemizygous (not homozygous (dramatic reduction)

c) Why? When hemizygous cell can still survive, but can't make enough miRNAs so becomes cancerous homozygous means the cell can make no Dicer so just dies (known as haploin sufficiency)

→ TBRP is also important for it (Also PACT)

ds RNA = double stranded RNA
ss RNA = single stranded RNA

a) Less TBRP & PACT decreases stability of Dicer a lot

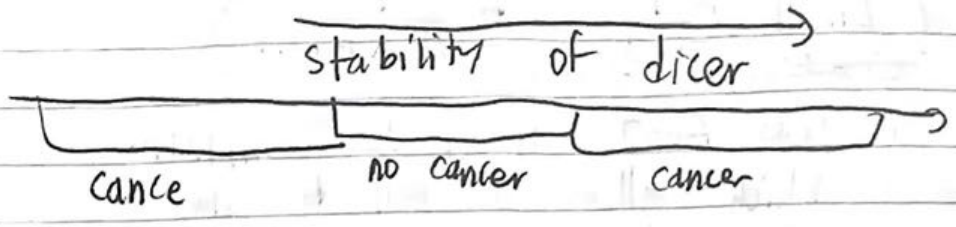
b) mutation in TBRP → less miRNA → less Dicer → Cancer

c) mutation here is a truncation mutation, shorter protein (from nonsense gene mutation, i.e premature stop codon)

d) putting full-length TBRP back in fixes Dicer

e) cellular signaling pathways also affect TBRP levels
↳ ex. MAPK / ERK promotes phosphorylation of TBRP → more stable Dicer

→ more growth miRNAs, less tumor surpressor miRNAs → cancer



The MAPK/ERK pathway when ON inhibits apoptosis, promote proliferation. If it is always ON it can cause cancer

Ago2 regulation
- ↳ humans contain 8 ago proteins: Ago 1→4, Piwi 1→4
- ↳ Piwi are structurally similar to Agos but interact with piRNAs

→ Levels of Ago2 contribute to miRNA regulation
(Ago2 ∝ miRNA levels
no Ago2, no MiRNa
more Ago2, more miRNA)

→ Ago proteins are to stabilize miRNA levels

→ Ago2 found to be increased in breast cancer

→ EGF (Epidermal Growth Factor, ligand to ⟶) increases Ago2 stability causing more Ago2

→ Ago2 targeted for degradation by Lin 41 (expressed in stem cells & undifferentiated cells)

(Lin41 $\propto \frac{1}{\#\ Ago2}$ ~~a) less~~ $\propto \frac{1}{\#\ of\ miRNA}$)

a) less Ago2 means less miRNA which allows the cell to stay stem-like

b) Lin 41 is the target of let-7

$\#\ Lin\ 41 \propto \frac{1}{\#\ of\ let\text{-}7}$

Similar to:

$\#\ Lin\ 28 \propto \frac{1}{let\text{-}7}$

c) In lung cancer # of let-7 is less, so Lin-48 is more so Ago2 is less (might be a characteristic of poorly differentiated tumors)

⑨ Mutations in miRNA processing
↳ single nucleotide polymorphisms (SNPs)
↳ point mutations

→ These mutations in pri-miRNA can change it's physical structure and cause it to not be able to bond to Drosha (mutations in pri- cause less pre-)

→ mutations in pre-miRNA reduce mature miRNA levels
↳ ex. when one nt changes from C→T in pre-miR-16-1 causes less miR-16-1

→ SNPs also alter levels
↳ Ex. a common G→C mutation in pre-miR-146 causes more risk of papillary thyroid cancer. When the C is there less mature miR-146 gets formed since it alters the 2nd structure (physical structure) of the pri-miR-146 and stops Drosha
↳ a mutation (poly morphism) of miR-125a makes the loop bigger blocking Drosha

→ Additionaly, SNP of miRNAs can cause the miRNA not be able to bind to the target
↳ ex. SNP in let-7 makes it unable to bind to the 3'UTR region of K-Ras which increase the risk of non-small cell lung cancer.

→ Therefore, loss or gain of miRNA binding sites in 3'UTR can be an important determinant in cancer

→ Length of 3'UTR also plays a role
a) in cancer 3'UTR is smaller so it is harder for tumor surpressor miRNAs to bind
b) the precise 3' end of some mRNAs could distinguish different tumors

→ The combined effect of reduced expression of miRNAs (tumor suppressors) and shorter 3'UTR might contribute to development of cancer

⑩ Conclusion

→ miRNAs regulate cell differentiation, proliferation and survival

→ alteration of miRNA expression cause cancer

→ regulation of miRNAs is diverse

→ an understanding on how miRNAs are regulated under normal conditions will allow us to better understand what gets de-regulated in cancer

End of work done on Oct, 22
pg 15/15

Done Annotating and writing notes on: "MiRNAs in Cancer"

Oct.23 On Oct, 23, I:

- had 3rd Science Fair meeting

Homework:

- Find what do you want to find out
- no book report
- Look at other studies to say how they

Structure it
↳ Literature review?
↳ Analysis?
↳ see how much data you want to incorporate
- Email Ms. Garcia on this?

Oct. 24 - Nov. 9

From Oct. 24 — Nov. 9 I:

→ Decided where I was going to get my data (GDC)

→ Learned how to use the GDC

→ Decided which specific miRNa family I am going to look at (let-7)

→ Decided which cancer region I am going to look at (Kidney)

→ Decided on which specific type of Kidney cancer I am going to look at:
Kidney Clear Cell adenocarcinoma
Based on # of cases present on type in GDC

→ Downloaded Data
1. Categorized by Stage or if it is Normal!
   - Stage 1
   - Stage 2
   - Stage 3
   - Stage 4
   - Normal

2. I grouped all these folders under the folder "Stage" for simplicity when searching.

3. Then, I went into my terminal and "cd" into Stage

4. Then I ran "grep -r" ... which means look in all the ... until you find ...

5. I ran this for all the let-7 miRNAs available and tabulated the results

Let-7 available:

hsa = homo sapien

hsa-let-7a-1
hsa-let-7b
hsa-let-7c
-7d
-7e
-7f-1
-7g
-7i

→ the -1, -2... means isoform. Isoforms of miRNA code for basically the same result, very ...

Nov. 10

Cont. on Nov. 10:

The reason only 1 isoform for hsa-let-7a & -7f is because the isoforms are expressed basically the same in the cell, so creating a new graph and table for them is pointless

→ Extract & Display Results

1. On each sheet there are:

table:

the columns are:

| UUID | File name | read count | RPM | Cross mapped |

Explanation of each column:

UUID: This is the ID of the data file

Case UUID: ID for the person from which the data was made

File name: name of the file

read count: how many times a specific miRNA sequence was observed in the data

Note: Read count isn't very accurate since different samples have different sequencing depths (reads)

RPM (Reads per million):

This way is much better and more accurate since it takes reads into account:

Calculated as:

$$\left(\frac{\text{read count}}{\text{total miRNA reads}}\right) \cdot 10^6$$

Note: Later, I added another column, file URL. This tells the URL for the data from which we got it. The form of the url is

"https://portal.gdc.cancer.gov/files/"

and then we [illegible] the UUID.

This is done [illegible] the formula button:

f_x = CONCAT("https://portal.gdc.cancer.gov/files/", C2)

Where C is the column for file UUID and 2 is the row

An example for one is:

f_2 = CONCAT("https://portal.gdc.gov/files/", C4)

Graph: Using the "Stage" and RPM columns I created 2 graphs, one a line graph the other a scatter plot

In the graph:

x-axis: Stage
y-axis: RPM

Cross mapped: If any miRNAs have similar sequences to the target miRNA. If so it can make it hard to get more values

Note: I've also added the means of each stage and cold

This is done by doing:

$\bar{x} = AVERAGE(C_{n_1} : C_{n_2})$

where C is the column where the RPM is
and,
n_1 = row of start of stage
n_2 = row of end of stage

Also, In addition to the RPM graphs, there is also the mean graph which is a bar graph graphing the
Stage: x axis
RPM: y axis

Nov. 19 On this day I:

- Started by background research
- wrote on miRNA and Cancer

Nov. 23 On this day I:

- Wrote on Kindey Clear Cell adenocarcinoma

Nov 27 On Nov 27 I:

- Edited the document
- added more to the para. on miRNAs
-

Nov. 29 On this day I:

- Started research paper

Nov. 30 On this day I:

- Read and made notes on pages 1-4 of "The Roles of the Let-7 Family of miRNAs in the Regulation of Cancer Stemness"

On Let-7 Family

Discovery

① Let-7 gene was discovered in C. elegans, a species of round worm (Nematoda)

② It was the second gene discovered in that species and is highly conserved

③ It's expression determines the fate of adult cells in the worm

④ highly conserved

Dec 2 On this day I:
→ Read on the paper and summarized info on Background Info

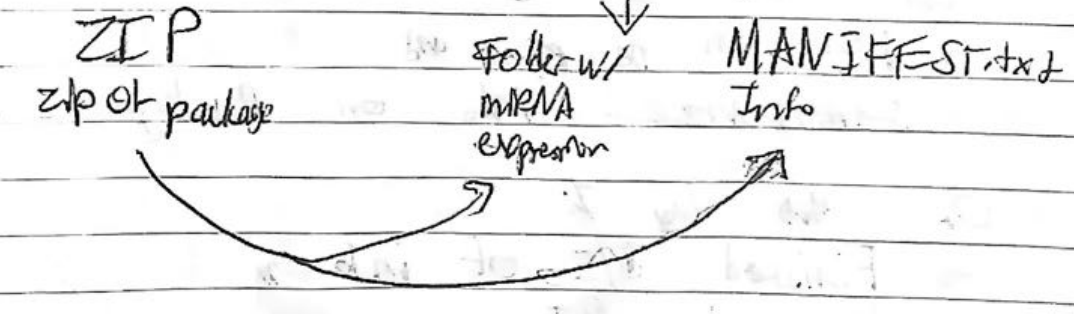
Dec 3 On this day I:
→ Finished 3/5 of info on paper in BI

Dec 4 On this day I:
→ Finished the entire BI 5/5

Dec6. On this day I:
- Started downloading the files for Trial 2 of Project_Data as the first trial seemed incorrect with huge outliers.
did Stage 1-3

Dec7 On this day I:
- Finished downloading Stage 4 & Normal data

SCIENCE_FAIR → Trial 1/2 Project_Data
↓
Stage 1-4, Normal
↓
Case ID of person
↓



Dec8 I graphed the same way I did Trial 1, just that I am including isoforms.
→ finished graphing hsa-let-7a-1
-3

Dec. 16 Finished graphing hsa-let-7b-e

Dec. 12 On this day I:
- Decided to increase my sample size by downloading all the available files for cancer ~500 sample size
- Started downloading files from GDC

Dec 14 On this day I:
- Finish downloading all GDC Files and finish Uploading files
- Groached w/ all data done

Dec 5 On this day I:
- Started to code Python model demonstrating the validity and high accuracy of a system for let-7b detection.
- p-value: $1.18 \times 10^{-7} = 0.000000118$
- Since a good p-value for scientific analysis

Dec. 17 Continued coding model

Dec 18 Finished coding model

Dec 19 Fixed bug for class seperation

from making A binary and using the new sheet, Python-Data.xlsx

Dec 21 Made box plot for Results
added progress to Science Fair Info so far

Dec 22
- Added more info for testing Code
- Ex: Screenshots of code, table displaying results, etc.

- Added Info from talk with other PhD, added more papers to study
  - → Found paper saying IL-1b should be down regulated in ccRCC, showing that there is a disagreement!

Jan 10 On this day I:

- Reviewed Code
- Reviewed SVMs and SMOTE

Jan 12 On this day I:
- Recorded Info on model
  Create page

Accuracy: 90.70%
Precision: 88.70%
Recall: 93.58%
ROC-AUC: 93.09%
F1 score: Normal: 0.90; Cancer: 0.91
# of False Positives: 13 (FP)
# of False Negatives: 7 (FN)
# of True Positives: 102 (TP)
# of True Negatives: 93 (TN)

Definitions:
Accuracy %: $\frac{TP+TN}{Total}$ Percent of guesses correct (% of the time model was correct)
Precision %: $\frac{TP}{TP+FP}$ Percent of time model predicted cancer correctly $= \frac{\text{correctly predicted } \oplus}{\text{Total predicted } \oplus}$

Recall %: How many ⊕ did the model get out of all ⊕?
$\frac{TP}{TP+FN}$

F1 score: by getting the harmonic mean of precision and recall, it can display how well the model can identify cancer
$= 2 \cdot \frac{\text{Precision} \times \text{Recall}}{\text{Precision} + \text{Recall}}$

ROC-AUC: A general score on how well it can classify, based on the effectiveness of its threshold

Jan 14

On this day I:
- Started writing analysis of results
  1. Let 7b III ?
  2. Let 7b = best biomarker
  3. ~~Let 7b~~
- Decided to do a paper

Jan 17

On this day I:
- Finished Analysis (Rough)
- Finished Abstract / Summary of Paper → JEI

Jan 19

On this day I:
- Started & Finished Introduction
- Refined Analysis
- Edited w/ Grammarly

Jan 20

On this day I:
- Finished Results of Paper
- Added Concluding para. for Discussion
- Added Acknowledgements
- Added 1-6 of References w/ QuillBot
- Added Title

Jan 21

On this day I:
- Finished Rough draft of paper
  ↳ Discussion & Materials & Methods ✓
- Added References 7-15 or 16
- Started reading (not annotating) paper by Wiley of Let-7 in lung cancer.

Feb

Jan31 On this day I:
- Reviewed BI
- Finished Introd on paper

Feb2 On this day I:
- reviewed analysis

Feb4 OTDI:
- reviewed Discussions & Methodology

Feb5 OTDI:
- added Bib to BI

AMA → Quantitat → Ivan Su Info
Ubat

Feb8 OTDI:
- Checked over Results
- dh meeting w/ professor.

Key: Do Literature Review for Research Gap

⇒ (Todoist) ⇐

Once done, meet hhn
also, do comments

Feb 10-12 OTDI:
- Used Grammarly (No AI) to review my work grammatically

Feb 15 OTDI:
- Reviewed 2 of 3 of his comments

Feb 17 OTDI:
- Started planning thesis

Canva?

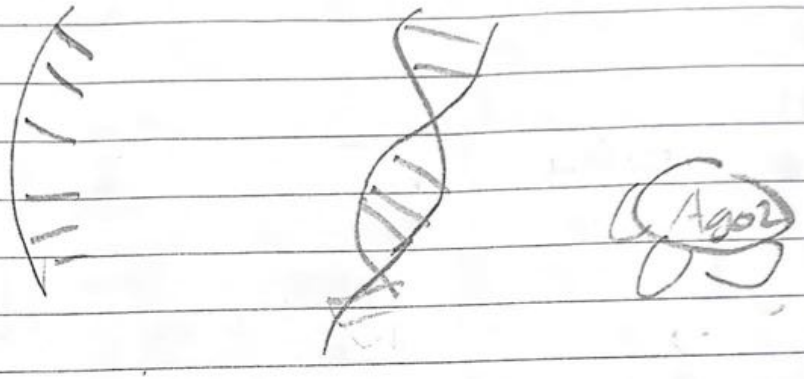
Slide Show

dimensions?

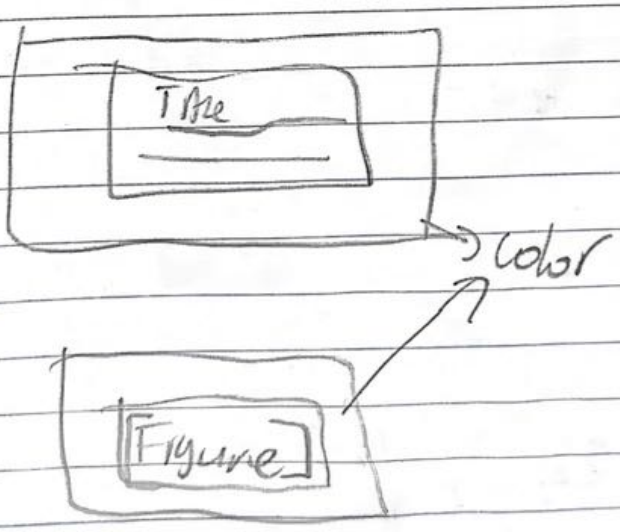
Feb 18 OTDI
- submitted everything to Turn It In Google

Feb 23 OTDI:
Canva
Color palette:
red, pink, teal, dark blue, yellow?

Feb 25: OTDT
- Got all graphs / tables
- looking for icons



Mar 2 OTDT:
- Extracted & summarized the texts

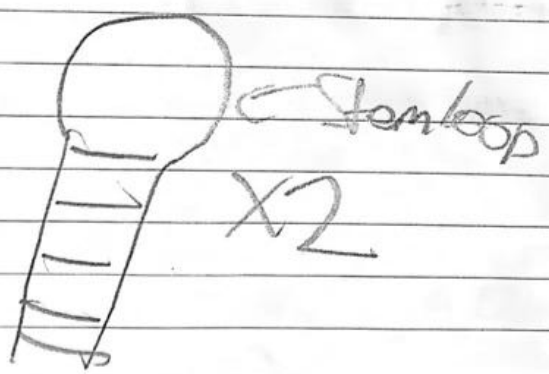


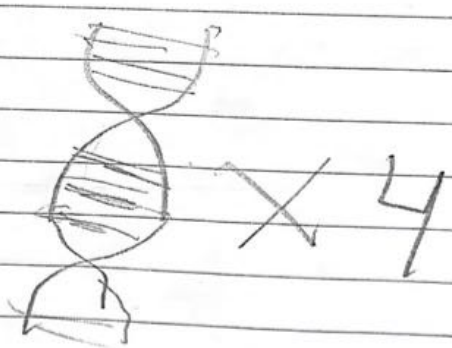
Mar4 OTOT

- did 1-8 for Lit review → JEL
- Reviewed all summarized texts

Mar6 OTOT

- found the terms



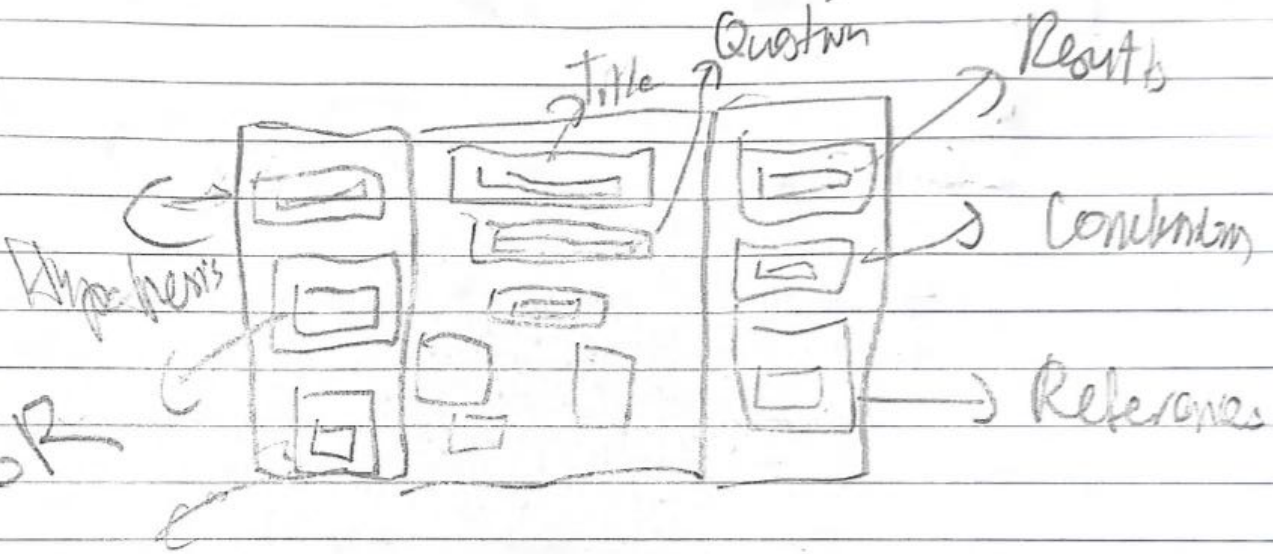


[Other diagrams] x4

Mar 7

OTDI:

Finished fully canva layout



procedure

(flowchart)

Mar 8

OTDI:

- In layout, switched BR & hypothesis
- Started building trifold
  - Finished middle fold

Mar 9

OTDI Locked in

- Finished entire trifold !!!
  Now ppl reparing

Mar 10 OTD I had a GH test:
- Prepared (reviewed)

Mar 12 OTD I got TODO List:

Science Fair TODO

- Complete CYSF portal
  - → Finish declaration form (Finished Mar 13)
  - → Finish paper things to portal
    - → Methodology, Discussion, BI, etc.
  - → Attachments
    - → banner
    - → 4:3 photo of me
    - → 4:3 photo of my project
    - → 10mm video
    - ~~→ Video on poster~~

→ Logbook

Mar 15 OTD I
→ Finished Method & BI of CYST
(just HC → HCV of the [FINAL] version of the doc)
(ran through Grammarly)

Mar 16

ODI

- Did the same as yesterday, but for Discussion & results
- need to add images & citations for

Mar 19

ODI locked in (kinda)

- Added citations & images
- added banner, added 4:3 image of representation of project
- pertinent image of me in a suit
- started to remove a piece of tape on it with pixlr.

Mar 20

- need to add image citations → Mar 20
- need to edit poster for video
- need to scan logbook → mar. 20