Countering Dermatological Defects in Mice Protected Against Adiposity

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Two main problems were examined in this research: 1) Are Silica, Coenzyme Q10, and Alpha-Lipoic Acid able to counter or improve dermatological defects in mice protected against adiposity? 2) Is either Silica and Alpha-Lipoic Acid or Coenzyme Q10 able to improve dermatological defects in mice protected against adiposity? It was hypothesized that: 1) Silica, Coenzyme Q10, and Alpha-Lipoic Acid would improve the dermatological defects in mice protected against adiposity. 2) Silica and Alpha-Lipoic Acid or Coenzyme Q10 would not improve the dermatological defects in mice protected against adiposity. The purpose of this biomedical research and testing is to understand the living body and what occurs in certain pathophysiologic processes, and to develop safe and effective ways of preventing or treating those diseases. The scientific research of Dr. James M. Ntambi, Loss of Stearoyl-CoA Desaturase-1 Function Protects Mice Against Adiposity, inspired this research. The ultimate goal of this research was to counter the dermatological defects in mice protected against adiposity through chemical intake and by recording food consumption, oil accumulation, and weight gain for the duration of 4 weeks. Group 1 showed oil accumulation most prominent in week 4, where there was a high weight gain of 4.36 g, and the least amount of average food consumption of 17.8 g. Group 2 showed oil accumulation most prominent in week 3, where there was the lowest weight gain of 0.31 g, and the second highest average food consumption of 20.36 g. Group 3 showed oil accumulation most prominent in week 3, where there was a weight loss of 3.31 g, and the highest average food consumption of 21.84 g. Group 4 showed oil accumulation most prominent in week 3, where there was a weight loss of 0.26 g, and a high average food consumption of 24.41 g. Group 5 showed oil accumulation most prominent in week 3, where there was the second lowest weight gain of 2.51 g, and the highest average food consumption of 26.32 g. There was a 100% Oil Accumulation Improvement, with the average color rank rise being 7.3 ranks. The highest color rank rise was #1, rising 12 ranks. The lowest color rank rises were #6 & #9, rising 3 ranks. Group 1 had a higher average color rank rise than the other mice, being 9.75 color ranks. There was 100% Weight Gain. The Silica & ALA mixture produced more of a color rank rise than only the CoQ10 mixture. The hypothesis that Silica, Coenzyme Q10, and Alpha-Lipoic Acid would improve the dermatological defects in mice protected against adiposity was supported. The hypothesis that either Silica and Alpha-Lipoic Acid or Coenzyme Q10 would not improve the dermatological defects in mice protected against adiposity was rejected. All three chemicals resulted in oil accumulation; however, the Silica & ALA mixture was the most effective, followed by the combination of the Silica & ALA mixture and the CoQ10 mixture, and finally the CoQ10 mixture. Scientific research of SCD1 gene activities has the potential to advance treatments for a wide range of human diseases including diabetes, atherosclerosis, cancer, obesity, and viral infection, which are associated with high SCD1 activity. Furthermore, SCD1 research may provide a healthy alternative for anorexia and bulimia. With this potential development for humans comes the idea that one can retard weight gain, reduce body fat mass, increase oxygen consumption, increase fatty acid oxidation, and increase insulin sensitivity, while consuming a high-fat diet and without having the deleterious side effects of dry skin or hair, which was the premise of this research.
INTRODUCTION

The ultimate goal of this research was to counter the dermatological defects in mice protected against adiposity. This research could contribute to making the genetic deletion of the stearoyl-Coenzyme A desaturate 1 (SCD1) gene less harmful to mice by reducing the side effects of dry skin and hair caused by a lack of oil and fat not being synthesized. Scientific research of SCD1 gene activities has the potential to advance treatments for a wide range of human diseases including diabetes, atherosclerosis, cancer, obesity, and viral infection, which are associated with high SCD1 activity. Furthermore, SCD1 research may provide a healthy alternative for anorexia and bulimia. With this potential development for humans comes the idea that one can retard weight gain, reduce body fat mass, increase oxygen consumption, increase fatty acid oxidation, and increase insulin sensitivity, while consuming a high-fat diet and without having the deleterious side effects of dry skin or hair, which was the premise of this research.

SCD1, also known as Scd1, Scd-1, AA589638, and AI265570, is a key enzyme in fatty acid metabolism. Fatty acid desaturases are enzymes that catalyze the insertion of a double bond at the delta position of fatty acids. There are two distinct families of fatty acid desaturases which do not seem to be evolutionary related: the first contains SCD; the second includes plant stearoyl-acyl-carrier protein and cyanobacteria desA protein. SCD is a key regulatory enzyme of unsaturated fatty acid biosynthesis. In association with cytochrome b5 and NADP-dependent cytochrome b5 reductase, it constitutes part of a microsomal membrane-bound 3-component system in animals and fungi. SCD contains four putative transmembrane (TM) regions that anchor it in the microsomal membrane. SCD uses oxygen and electrons from reduced cytochrome b5 to catalyse the insertion of a
cis double bond between carbons 9 and 10 of a spectrum of fatty acids. The preferred substrates of SCD are palmitoyl-CoA and stearoyl-CoA, which are converted to palmitoleic and oleic acids respectively. These unsaturated molecules are the major storage form of fatty acids (as triacylglycerols) in adipocytes (EMBL-EBI, 2007). SCD1 acts like a switch to control fat storage. When SCD1 activity is ‘up’, the switch is flipped in the direction of storing fat, and when its activity is ‘down’, the switch is flipped in the direction of burning fat (Xenon, 2005).

Over 90 percent of patients with diabetes have type II diabetes mellitus (varying or persistent hyperglycemia, high blood sugar levels, resulting from the defective secretion or action of the hormone insulin). Patients with type II diabetes produce insulin but lose the ability to respond to insulin signaling due to down regulation of hormone receptors. This research provides a method of increasing insulin sensitivity by reducing SCD1 activity in diabetes patients. Reducing the amount of SCD1 protein, inhibiting SCD1 enzymatic activity, or both, can increase insulin sensitivity, providing a way to better prevent or treat type II diabetes. Researchers found that mice with little or no SCD activity remained sensitive to insulin when fed a regular diet and did not become obese when fed a high fat diet. This knowledge can be transposed to humans and subsequent correlation can be assumed. A test agent could be administered to the subject and if the test agent reduces SCD1 activity, it may increase insulin sensitivity in the subject. Thus, this research provides a new tool for treating and preventing type II diabetes and is useful for testing agents that may be able to increase insulin sensitivity (WARF, 2007).

Atherosclerosis is a condition in which atheromatous plaques (fatty material) collect along the walls of arteries. These atheromatous plaques thicken, harden, and may
eventually block the arteries, leading to an insufficient blood supply to the organ it feeds. If blood flow in the arteries leading to the heart is reduced, chest pain can occur. Plaque buildups can also break apart, causing pieces of material to move through the artery, the common consequences of such being heart attack and stroke. Blood clots can form around the plaque deposits, blocking blood flow to the heart, lungs, and brain and potentially causing a stroke, heart attack, or pulmonary embolism (blockage of an artery in the lungs by fat, air, clumped tumor cells, or a blood clot). Also, if there is an excess of compensating artery enlargement for the narrow blood passage, a net aneurysm (bulge in a blood vessel) results. These complications are chronic, slowly progressing and cumulative. Most commonly, soft plaque suddenly ruptures, causing the formation of a thrombus that will rapidly slow or stop blood flow, leading to death of the tissues fed by the artery. This catastrophic event is called an infarction. One of the most common recognized scenarios is called coronary thrombosis of a coronary artery causing myocardial infarction (a heart attack). Another common scenario in very advanced disease is claudication (cramp like pains) from insufficient blood supply to the legs, typically due to a combination of both stenosis and aneurismal segments narrowed with clots. Since atherosclerosis is a body wide process, similar events also occur in the arteries to the brain, intestines, kidneys, legs, etc (Wikipedia, 2007). Recent studies have shown that SCD1, the main SCD isoform expressed in liver, is a key player in the regulation of lipid metabolism. SCD1 deficient mice have increased energy expenditure, reduced body adiposity, increased insulin sensitivity and are resistant to diet-induced obesity and liver steatosis (fatty liver). SCD1 was found to be specifically repressed during leptin-mediated weight loss and leptin-deficient ob/ob (obese) mice lacking SCD1
showed markedly reduced adiposity, despite higher food intake. In addition, SCD1 deficiency completely corrects the hypometabolic phenotype (low metabolic rate) and hepatic steatosis (fatty liver) of *ob/ob* (obese) mice. Consequently, increased SCD activity has been found in humans and animals which accumulate significant amounts of lipids in liver, whereas SCD1 deficiency ameliorates both high-fat diet induced and genetically induced hepatic steatosis. Much evidence indicates that the direct anti-steatotic effect of SCD1 deficiency stems from increased fatty acid oxidation and reduced lipid synthesis. Thus, this research may contribute to advance treatments for atherosclerosis (Dobrzyn, 2006).

The relationship between family history of selected neoplasms (tissues composed of cells that grow in an abnormal way) in first-degree relatives and the risk of pancreatic, liver, and gallbladder cancer was investigated using data from a case-control study conducted in northern Italy on 320 histologically confirmed incident cases of liver cancer, 58 of gallbladder cancer, 362 of pancreatic cancer, and 1408 controls admitted to the hospital for acute, non-neoplastic, non-digestive tract disorders. Significant associations were observed between family history of hepatocellular carcinoma (liver cancer) and primary liver cancer [relative risk (RR) = 2.4; 95% confidence interval (CI), 1.3 to 4.4], between family history of pancreatic cancer and pancreatic cancer (RR = 3.0; 95% CI, 1.4 to 6.6), and between family history of gallbladder cancer and gallbladder cancer (RR = 13.9; 95% CI, 1.2 to 163.9). The elevated risk of liver cancer associated with family history was not materially modified by adjustment for tobacco, alcohol, and personal history of cirrhosis and hepatitis (RR = 2.9; 95% CI, 1.5 to 5.3). Similarly, the risk for pancreatic cancer did not appreciably change after allowance for tobacco, alcohol, dietary...
factors, and medical history of diabetes and pancreatitis (RR = 2.8; 95% CI, 1.3 to 6.3).
This pattern of risk would support the existence of a genetic component in the familial
aggregation of liver and pancreatic cancer. In terms of population attributable to risk,
approximately 3% of the newly diagnosed liver and pancreatic cancers would be related
to this familial genetic component, the SCD1 gene. Thus, this research may potentially
contribute to advance treatments for that 3% of newly diagnosed liver and pancreatic
cancer (Fernandez et al, 1994).

Metabolic syndrome is a clustering of certain metabolic-related heart disease risk
factors including obesity, and has become one of the greatest health problems worldwide.
Effective therapies to treat obesity and metabolic syndrome are urgently needed but are
currently lacking. In a study appearing in the April 1, 2005 print edition of the Journal of
Clinical Investigation, Bei Zhang and colleagues from Merck Research Laboratories
demonstrate a new therapeutic approach to treat obesity and metabolic syndrome. These
researchers blocked the SCD1 gene, thereby suppressing enzyme expression which
regulates fat storage in the body. The SCD1 gene locus was suppressed by using specific
small molecules to interfere with the synthesis of SCD1. Inhibiting SCD1 gene
expression decreased body fat and prevented obesity and metabolic syndrome. Blocking
SCD1 also increased expression of genes involved in energy expenditure. Thus, this
research aids to further our understanding of the role of SCD1 gene expression in the
metabolism and validates the SCD1 gene as a potential target for pharmacological
intervention for obesity and related metabolic disorders (Bloom, 2005).

The autoimmune trigger of juvenile diabetes might be viral infection. Scientists
have discovered that the peculiar ability of normal insulin-producing cells to resist
infection by coxsackie virus is due to their robust response to natural antiviral compounds called interferons. Mice whose insulin-producing cells were prevented from responding normally to interferons were susceptible to damage by coxsackie virus, and developed acute diabetes similar to that developed by humans after severe viral infection. Hepatitis C has been linked to type 2 diabetes, and animal research explains this: mice carrying the hepatitis C core gene had high insulin levels, and rapidly developed insulin resistance and then diabetes. Surprisingly, infection with a virus called LMCV protects mice from developing type 1 diabetes at an early stage in the disease. When it was given in the pre-diabetic phase, LMCV stopped the destruction of insulin-producing cells. The precise time of the infection might be crucial to success, so more work is needed. SCD1 null mice have increased insulin sensitivity along with their weight loss. Thus, this research may contribute to advance treatments for viral infections (RDS1, 2007).

8,000,000 or more people in the United States have an eating disorder, 90% of whom are women, of all financial statuses. Eating disorders usually start in the teens but may begin as early as age 8 (National Association of Anorexia Nervosa and Associated Disorders, 2006). Anorexia nervosa is an illness that affects all ages and both genders. Anorexics are obsessed with being thin and are terrified of gaining weight. They have a distorted perception of their bodies, believing they are fat, when actually they are thin. Anorexics have dry skin and thinning hair on the head and may exhibit growth of fine hair all over their body. They may feel cold all the time and are prone to common illnesses (i.e. cold and flu). People with severe anorexia may be at risk of death from starvation. Anorexics have difficulty concentrating and are constantly thinking about food. Treatment of anorexia is complex, because many people with this disorder believe
there is nothing wrong with them. Anorexia is not just a problem with food or weight; it is an attempt to use food and weight to deal with emotional problems. Bulimia is a cycle of bingeing and purging. After a binge, some bulimics fast or over-exercise to keep from gaining weight. Bulimics may also use water pills, laxatives, or diet pills to "control" their weight. Bulimics are usually close to normal weight, but their weight may go up and down. If anorexics and bulimics could overcome the psychological aspect of their disorders, they could benefit from suppressing the SCD1 gene locus (being SCD1 null), because it would eliminate the need to worry about drastic weight gain, and subsequently, result in a healthier lifestyle. Thus, this research may contribute to advance treatments for eating disorders such as anorexia nervosa and bulimia (AAFP, 2005).

Scientific research of the SCD1 gene, as noted in the previous paragraphs, has the potential to advance treatments for a wide range of disorders including diabetes, atherosclerosis, cancer, obesity, and viral infection, which are associated with high SCD1 activity. Furthermore, it may provide a healthy alternative for eating disorders such as anorexia and bulimia. The scientific research of Dr. James M. Ntambi, Loss of Stearoyl-CoA Desaturase-1 Function Protects Mice Against Adiposity, James Ntambi et al, PNAS Early Edition, August 12, 2002, inspired this research.

Expression of the SCD1 gene creates an enzyme that inserts a double bond into an unsaturated fatty acid. The result is a saturated fat, which can be stored in the rodent equivalent of spare-tire bulge. By deleting the SCD1 gene, Ntambi’s mice stayed lean and their blood sugar was lower than normal animals that ate the same high-fat diet, indicating that they did not have adult-onset of type II diabetes which affects 17 million Americans and is a major result of the increased tendency for obesity. The SCD1 gene

Haley Daus / 11th Grade
deletion activates genes that are known to increase fat metabolism, while deactivating genes that help store fat. Ntambi observed weight of wild-type, heterozygous, and homozygous mice either on a standard laboratory chow or on a high-fat diet and performed measurements of oxygen consumption and gene expression analysis to determine the effects of SCD1 null mice. As results, the SCD1 null mice exhibited reduced body weight on a high-fat diet, reduced body fat mass, increased oxygen consumption, increased expression of genes involved in fatty acid oxidization, and increased insulin sensitivity. The data suggested that a consequence of SCD1 gene deficiency is an activation of lipid oxidation as well as reduced triglyceride synthesis and storage (Ntambi, 2002).

The SCD1 null mice showed side effects; however, of dry hair and eyelids. The fat synthesized with the help of SCD1 is needed to lubricate fur and eyelids. The fur dryness was caused by a lack of oil in the hair follicles and the eyelid dryness was a result of fat not being synthesized, a process which lubricates the cornea for blinking. Thus, the SCD1 null mice used in this research exhibited the same dermatological defects as Dr. Ntambi’s mice.

The mice used in this research were obtained from The Jackson Laboratory in Bar Harbor, Maine, as suggested by Dr. Ntambi. There were 13 mice total used in this research: 6 were parental strains and 7 were SCD1 null strains. The 6 parental strains of the SCD1 null mice (C57BL/6J and DBA/1J) were suggested by Technical Information Scientist Dr. James Yeadon. The wild-type littermates from this colony were no longer available for controls. In general, the most appropriate control for a strain maintained on a mixed genetic background, for which wild-type littermates are not available, is an F2
hybrid bred from the two parental strains. Within a population of F2 hybrid mice, the full range of genetic and phenotypic variability contributed by the parental genetic backgrounds will be represented. This verified that the different genetic backgrounds of the mice did not null the data. The SCD1 null mice (B6;D1Lac-Scd1<ab-2J>/J) were homozygotes for targeted and spontaneous mutations, who exhibited alopecia (hair loss from areas of the body, especially the scalp), scaly skin, sebaceous gland hypoplasia (underdeveloped glands on the body that secrete an oily substance called sebum, which is made of lipids and the debris of dead fat-producing cells), impaired ocular lubrication and synthesis and storage of triglycerides, higher lipid oxidation, reduced growth, and lower fertility in females. Thus, the basis of this research was to correct the dermatological defects in these mice protected against adiposity.

**PROBLEMS**

Two main problems were examined in this research:

1) Are Silica, Coenzyme Q10, and Alpha-Lipoic Acid able to counter or improve dermatological defects in mice protected against adiposity?

2) Is either Silica and Alpha-Lipoic Acid or Coenzyme Q10 able to improve dermatological defects in mice protected against adiposity?
HYPOTHESES

It was hypothesized that:

1) Silica, Coenzyme Q10, and Alpha-Lipoic Acid would improve the dermatological defects in mice protected against adiposity.

2) Silica and Alpha-Lipoic Acid or Coenzyme Q10 would not improve the dermatological defects in mice protected against adiposity.

PROCEDURE

Chemical Intake through High-Fat Diet:

1) 13 mice total were used in experimentation & divided into 5 test groups

   GROUP 1: #1, #2, #3, #4= parental strains with high-fat diet only

   GROUP 2: #5, #6= parental strains with high-fat diet & all chemicals (Silica, Alpha-Lipoic Acid, Coenzyme Q10)

   GROUP 3: #7, #8, #9, #10, #13= mutant strains with high-fat diet & all chemicals

   GROUP 4: #11= mutant strain with high-fat diet & Coenzyme Q10 mixture only

      (did NOT receive Silica/Alpha-Lipoic Acid mixture)

   GROUP 5: #12= mutant strain with high-fat diet & Silica and Alpha-Lipoic Acid mixture only (did NOT receive Coenzyme Q10 mixture)

2) Silica, Coenzyme Q10 (CoQ10), and Alpha-Lipoic Acid (ALA) were obtained over-the-counter from a local pharmacy in capsule form.

3) Silica and ALA capsules were broken by hand, grinded by mortar and pestle, and mixed with distilled water using a 25 mg/ 1 mL concentration of ALA and Silica, creating a thin white paste.
4) 32 CC of the Silica/ALA paste was loaded into a 35 CC syringe using a small spatula, where it was distributed into 1 CC oral syringes, depending on the correct dosage per mouse. (Chemical dosages based on weight: 1 mg per 1 kg.)

5) CoQ10 capsules were broken by hand, grinded by mortar and pestle, and mixed with glycerin, ethanol, tangerine oil, and magna sweet using a 3000 mg/25 mL concentration of CoQ10, creating a thin orange paste.

6) 25 CC of the CoQ10 paste was loaded into a 60 CC syringe using a small spatula, where it was distributed into 1CC oral syringes, depending on the correct dosage per mouse. (Chemical dosages based on weight: 1 mg per 1 kg.)

7) The pastes were mixed with the high-fat diet depending on the group. (GROUP 1= no chemicals, GROUP 2= all three chemicals, GROUP 3= all three chemicals, GROUP 4= CoQ10 paste only, GROUP 5= Silica and ALA paste only)

8) Food consumption recorded daily. (Each mouse given 4 g food daily)

**Oil Accumulation through Sudan III Testing**

1) Oil samples were taken every week, including when mice first received.

2) Sudan III solution was prepared by pouring Ethyl Alcohol into a beaker and stirring in as much Sudan III was needed until the solution became deep red in color.

3) The back of each mouse was swabbed with a sterile swab.

4) That swab was submerged in the Sudan III solution and then extracted.

5) The swab was compared to a red color chart to determine the level of oil accumulation per mouse. (The deeper the red on the swab, the more oil present.)
Weight Gain Record:

1) Each mouse was weighed in a cardboard box with a manila folder cover on a triple beam balance weighing scale every week, including when first received.

2) The box & manila folder weighed 110.5 g, which was subtracted from the overall weight of the mouse, box, and folder, to get the weight of the mouse.

Precautions:

1) Mice were observed every day for 4 weeks.

2) Appropriate care for animals was provided daily, including weekends, holidays and other times when school was not in session. This care included: nutritious food, fresh water, clean housing with sufficient space, enrichment suitable for mice, and appropriate temperature and lighting.

3) These samples were non-invasive and non-intrusive and did not affect the animals’ health or well being by causing stress, discomfort, pain, or death.

4) Post experimentation, the mice were retained for further experimentation. Eventually, mice will be given to Missouri Southern State University, in Joplin, MO and their futures determined by faculty.

Red Color Chart- each integer represents one color rank
RESULTS

*S* = Silica  
*CoQ10* = Coenzyme Q10  
*ALA* = Alpha-Lipoic Acid

Data was analyzed based on progression of oil accumulation and weight gain every week and on food consumption every day.

Data from mouse #5 was disregarded due to pregnancy. This prevented obtaining a true weight for this mouse and swabbing it for the Sudan III test. A pregnant mouse needs to be left in isolation after giving birth to not become stressed and cannibalize her young. Thus, she was not disturbed by weighing or swabbing.

All mice were females except #13.

- **BLUE**= mouse number. #1- #6= parental strains
  
  #7- #13= SCD1 null strains

- **PURPLE**= time in [age (weeks) or weeks, depending on graph]. Data collection commenced when mice were 6 weeks of age and concluded when mice were 10 weeks of age. Thus, data collection persisted for a total of 4 weeks.

Mice divided into 5 groups:

1. **GROUP 1**: Parental Strains (with SCD1 gene), who were only on the high-fat diet
   
   a. **#1, #2, #3, #4**

2. **GROUP 2**: Parental Strains (with SCD1 gene), who were on the high-fat diet & all three chemicals (S, ALA, CoQ10)
   
   a. **#5, #6**
b. Data from mouse #5 was disregarded due to pregnancy

3. GROUP 3: SCD1 Null Strains (without SCD1 gene), who were on the high-fat diet & all three chemicals (S, ALA, CoQ10)
   a. #7, #8, #9, #10, #13

4. GROUP 4: SCD1 Null Strain (without SCD1 gene), who was on high-fat diet & CoQ10 mixture only (did NOT receive S or ALA)
   a. #11

5. GROUP 5: SCD1 Null Strain (without SCD1 gene), who was on high-fat diet & S and ALA mixture only (did NOT receive CoQ10)
   a. #12
### Table 1: Body Weight (g) vs. Age (weeks)

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### Graph 1: Body Weight vs. Age

[Graph showing body weight vs. age with data from Table 1 plotted.]
Table 2: Body Weight (g) vs. Age (weeks) [#2]

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Graph 2: Body Weight vs. Age [#2]
### Table 3: Oil Accumulation (color rank) vs. Age (weeks)

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<td>16</td>
<td>11</td>
<td>17</td>
<td>14</td>
<td>18</td>
<td>14</td>
</tr>
</tbody>
</table>

### Graph 3: Oil Accumulation vs. Age

![Graph 3: Oil Accumulation vs. Age](image-url)
Table 4: Oil Accumulation (color rank) vs. Age (weeks) [#2]

<table>
<thead>
<tr>
<th>Age (weeks)</th>
<th>#1</th>
<th>#2</th>
<th>#3</th>
<th>#4</th>
<th>#6</th>
<th>#7</th>
<th>#8</th>
<th>#9</th>
<th>#10</th>
<th>#11</th>
<th>#12</th>
<th>#13</th>
</tr>
</thead>
<tbody>
<tr>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
<td>8</td>
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<td>9</td>
<td>10</td>
<td>8</td>
<td>9</td>
<td>8</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>10</td>
<td>19</td>
<td>19</td>
<td>17</td>
<td>16</td>
<td>12</td>
<td>17</td>
<td>16</td>
<td>11</td>
<td>17</td>
<td>14</td>
<td>18</td>
<td>14</td>
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</tbody>
</table>

Graph 4: Oil Accumulation vs. Age [#2]
Table 5: Food Consumption (g) vs. Age (weeks)

<table>
<thead>
<tr>
<th>Age (weeks)</th>
<th>#1</th>
<th>#2</th>
<th>#3</th>
<th>#4</th>
<th>#6</th>
<th>#7</th>
<th>#8</th>
<th>#9</th>
<th>#10</th>
<th>#11</th>
<th>#12</th>
<th>#13</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>18.47</td>
<td>17.16</td>
<td>19.21</td>
<td>20.94</td>
<td>20.3</td>
<td>18.66</td>
<td>20.28</td>
<td>21.49</td>
<td>22.48</td>
<td>22.53</td>
<td>20.71</td>
<td>20.26</td>
</tr>
</tbody>
</table>

Graph 5: Food Consumption vs. Age
Table 6: Food Consumption (g) vs. Age (weeks) [#2]

<table>
<thead>
<tr>
<th>Age (weeks)</th>
<th>#1</th>
<th>#2</th>
<th>#3</th>
<th>#4</th>
<th>#6</th>
<th>#7</th>
<th>#8</th>
<th>#9</th>
<th>#10</th>
<th>#11</th>
<th>#12</th>
<th>#13</th>
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</thead>
<tbody>
<tr>
<td>7</td>
<td>18.47</td>
<td>17.16</td>
<td>19.21</td>
<td>20.94</td>
<td>20.3</td>
<td>18.66</td>
<td>20.28</td>
<td>21.49</td>
<td>22.48</td>
<td>22.53</td>
<td>20.71</td>
<td>20.26</td>
</tr>
</tbody>
</table>

Graph 6: Food Consumption vs. Age [#2]
### Table 7: GROUP 1: Avg. Food Consumption, Avg. Weight Gain, & Avg. Oil Accumulation

<table>
<thead>
<tr>
<th>GROUP 1</th>
<th>Average Food Consumption</th>
<th>Average Weight Gain</th>
<th>Average Oil Accumulation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(weeks)</td>
<td>#1</td>
<td>#2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>19.47</td>
<td>17.16</td>
<td>20.19</td>
</tr>
<tr>
<td>2</td>
<td>16.57</td>
<td>19.13</td>
<td>17.21</td>
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<tr>
<td>3</td>
<td>16.14</td>
<td>16.66</td>
<td>20.67</td>
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<tr>
<td>4</td>
<td>16.96</td>
<td>16.48</td>
<td>16.27</td>
</tr>
<tr>
<td>Average Total:</td>
<td>74.98</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

GROUP 1: food consumption, weight gain, oil accumulation
Table 8: GROUP 2: Avg. Food Consumption, Avg. Weight Gain, & Avg. Oil Accumulation

GROUP 2
Average Food Consumption (g)
(weeks) |
---|
1 | 20.3  
2 | 19.63  
3 | 20.36  
4 | 21.21  
**Average Total:** | **81.5**

Average Weight Gain (g)
(weeks) |
---|
1 | 25.49  
2 | 2.18  
3 | 0.31  
4 | 6.48  
**Average Total:** | **34.46**

Average Oil Accumulation (ranks)
(weeks) |
---|
1 | 0  
2 | 1  
3 | 2  
4 | 0  
**Average Total:** | **3**

GROUP 2: food consumption, weight gain, oil accumulation
Table 9: GROUP 3: Avg. Food Consumption, Avg. Weight Gain, & Avg. Oil Accumulation

GROUP 3
Average Food Consumption (g)

<table>
<thead>
<tr>
<th>(weeks)</th>
<th>#7</th>
<th>#8</th>
<th>#9</th>
<th>#10</th>
<th>#13</th>
<th>Average</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>18.66</td>
<td>20.28</td>
<td>21.49</td>
<td>22.48</td>
<td>20.26</td>
<td>20.634</td>
</tr>
<tr>
<td>3</td>
<td>20.62</td>
<td>21.29</td>
<td>20.66</td>
<td>23.34</td>
<td>23.29</td>
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<td></td>
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Average Weight Gain (g)

<table>
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<th>(weeks)</th>
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<th>#9</th>
<th>#10</th>
<th>#13</th>
<th>Average</th>
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</thead>
<tbody>
<tr>
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<td>29</td>
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<td>26.536</td>
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<tr>
<td>2</td>
<td>0.55</td>
<td>0.6</td>
<td>0.21</td>
<td>0.15</td>
<td>0.11</td>
<td>0.324</td>
</tr>
<tr>
<td>3</td>
<td>-0.3</td>
<td>0.31</td>
<td>-2.54</td>
<td>-1.09</td>
<td>0.31</td>
<td>-3.31</td>
</tr>
<tr>
<td>4</td>
<td>4.79</td>
<td>4.29</td>
<td>6.78</td>
<td>5.51</td>
<td>4.78</td>
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<td>Average Total: 28.78</td>
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Average Oil Accumulation (ranks)

<table>
<thead>
<tr>
<th>(weeks)</th>
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<th>#8</th>
<th>#9</th>
<th>#10</th>
<th>#13</th>
<th>Average</th>
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<td>3</td>
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<td>6</td>
<td>1</td>
<td>1</td>
<td>3</td>
<td>3</td>
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<td>0</td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Average Total: 6.2</td>
</tr>
</tbody>
</table>

GROUP 3: food consumption, weight gain, oil accumulation

Haley Daus / 11th Grade
Table 10: GROUP 4: Avg. Food Consumption, Avg. Weight Gain, & Avg. Oil Accumulation

GROUP 4
Average Food Consumption (g)

<table>
<thead>
<tr>
<th>(weeks)</th>
<th>#11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>22.53</td>
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<tr>
<td>2</td>
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</tr>
<tr>
<td>3</td>
<td>24.41</td>
</tr>
<tr>
<td>4</td>
<td>25.5</td>
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</table>

Average Total: 96.87

Average Weight Gain (g)

<table>
<thead>
<tr>
<th>(weeks)</th>
<th>#11</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>30.64</td>
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<tr>
<td>2</td>
<td>1.46</td>
</tr>
<tr>
<td>3</td>
<td>-0.26</td>
</tr>
<tr>
<td>4</td>
<td>4.73</td>
</tr>
</tbody>
</table>

Average Total: 36.57

Average Oil Accumulation (ranks)

<table>
<thead>
<tr>
<th>(weeks)</th>
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</thead>
<tbody>
<tr>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>4</td>
</tr>
<tr>
<td>4</td>
<td>2</td>
</tr>
</tbody>
</table>

Average Total: 6

GROUP 4: food consumption, weight gain, oil accumulation

![Graph showing food consumption, weight gain, and oil accumulation over weeks]

Haley Daus / 11th Grade
### Table 11: GROUP 5: Avg. Food Consumption, Avg. Weight Gain, & Avg. Oil Accumulation

**GROUP 5**

**Average Food Consumption (g)**

<table>
<thead>
<tr>
<th>(weeks)</th>
<th>#12</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>20.71</td>
</tr>
<tr>
<td>2</td>
<td>26.08</td>
</tr>
<tr>
<td>3</td>
<td>26.32</td>
</tr>
<tr>
<td>4</td>
<td>22.45</td>
</tr>
</tbody>
</table>

Average Total: 95.56

**Average Weight Gain (g)**

<table>
<thead>
<tr>
<th>(weeks)</th>
<th>#12</th>
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</thead>
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<tr>
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<td>2.1</td>
</tr>
<tr>
<td>3</td>
<td>2.51</td>
</tr>
<tr>
<td>4</td>
<td>4.15</td>
</tr>
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</table>

Average Total: 35.75

**Average Oil Accumulation (ranks)**

<table>
<thead>
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<th>(weeks)</th>
<th>#12</th>
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</thead>
<tbody>
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<td>1</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>3</td>
<td>8</td>
</tr>
<tr>
<td>4</td>
<td>1</td>
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</tbody>
</table>

Average Total: 9

---

**GROUP 5: food consumption, weight gain, oil accumulation**

![Graph showing food consumption, weight gain, and oil accumulation over weeks](image)
The results are as follows:

1. Group 1 controls consuming only a high-fat diet showed oil accumulation most prominent in week 4, where there was a high weight gain of 4.36 g, and the least amount of average food consumption of 17.8 g.

2. Group 2 controls consuming a high-fat diet, S, ALA, and CoQ10 showed oil accumulation most prominent in week 3, where there was the lowest weight gain of 0.31 g, and the second highest average food consumption of 20.36 g.

3. Group 3 SCD1 null mice consuming a high-fat diet, S, ALA, and CoQ10 showed oil accumulation most prominent in week 3, where there was a weight loss of 3.31 g, and the highest average food consumption of 21.84 g.

4. Group 4 SCD1 null mouse consuming a high-fat diet and CoQ10 showed oil accumulation most prominent in week 3, where there was a weight loss of 0.26 g, and a high average food consumption of 24.41 g.

5. Group 5 SCD1 null mouse consuming a high-fat diet, S, and ALA showed oil accumulation most prominent in week 3, where there was the second lowest weight gain of 2.51 g, and the highest average food consumption of 26.32 g.

6. There was a 100% Oil Accumulation Improvement, with the average color rank rise being 7.3 ranks.

7. The highest color rank rise was #1, where it went from 7 to 19, rising 12 ranks. This mouse was a parental strain control that consumed the high-fat diet only.

8. The lowest color rank rises were #6 & #9, going from 9 to 12 and 8 to 11 respectively, rising 3 ranks. #6 was a parental strain control that consumed the
Countering Dermatological Defects in Mice Protected Against Adiposity 28

high-fat diet & all three chemicals. #9 was a SCD1 null strain that consumed the high-fat diet & all three chemicals.

9. Group 1 (#1, #2, #3, and #4), parental strain controls that consumed only the high-fat diet, had a higher average color rank rise than the other mice, being 9.75 color ranks.

10. 100% Weight Gain

a. The mice after week 4 were between 45.7 grams and 49.4 grams, an unusually high weight for the mice. Dr. Ntambi’s research showed female mice, whether with or without the SCD1 gene, in the range of 27.7 grams around the age of my mice.

b. SCD1 gene removal only severely retards weight gain; SCD1 gene removal does not prevent weight gain.

11. The S and ALA mixture produced more of a color rank rise than only the CoQ10 mixture. Mouse #12 consumed the high-fat diet & the S and ALA mixture, rising 9 color ranks. Mouse #11 consumed the high-fat diet & the CoQ10 mixture, rising 6 color ranks.

DISCUSSION

The mice that consumed any or all of the three chemicals showed an oil accumulation spike in week 3, high average food consumption, and low average weight gain or weight loss. The mice that consumed none of the chemicals showed an oil accumulation spike in week 4, lowest average food consumption, and high
average weight gain. This data suggests that chemical intake (S, CoQ10, ALA) allowed the mice to have increased food consumption, suppressed weight gain or weight loss, and accelerated oil accumulation, contrary to the mice receiving no chemicals. A t-test confirmed that SCD1 null mice consumed a significantly higher amount of food than the parental strain controls (p= 0.039172).

The groups that had oil accumulation spikes in week 3 had low weight gain or weight loss that week. This data requires further examination to determine a cause. These mice do not retain enough lipids to the extent that they have severe dermatological defects. The intake of extremely high-fat food (60 kcal% fat) is not enough to counter these defects. Thus, something internally is happening to these mice that is making their bodies refuse the essential lipids needed to lubricate and insulate their skin. This act of lipid disposal defies Charles Darwin’s survival of the fittest theory, where the body naturally gets rid of the components that are not of use and keeps the essential components needed to survive. The body, if there is an excess of oil, should either store or oxidize those lipids. Due to the increased lipid oxidation, these mice are accumulating oil, due to the oil spike in week 3, but are not storing it, due to the low weight gains or weight losses in that week. Thus, this data currently suggests that the mice are refusing lipid storage so severely that when lipids are presented, their bodies automatically oxidize nearly all or all of the lipids, much like a reverse survival mode.

GROUP 1, which included mouse #1, who had the highest color rank rise, had a higher average color rank rise than the other mice. GROUP 1 was comprised of parental strain controls receiving only the high-fat diet. These mice had SCD1 genes...
and were able to properly absorb oil and synthesize fat. Thus, it makes sense that these mice gained a large amount of oil because they were able to absorb and store the high amount of lipids contained in the high-fat diet.

Mouse #6 was a parental strain control; however, that had one of the lowest oil accumulations of all the mice. This mouse consumed the high-fat diet & all three chemicals (S, ALA, CoQ10). This mouse also had one of the highest weight gains, being 34.46. This data suggests that since #6 did not need extra oil, its body could have stored the excess of oil, produced from the chemicals, away as fat. An examination of the corpse could confirm or deny whether the fat was pocketed around the abdomen, heart, liver, or other organs.

Mouse #9 was a SCD1 null strain that had one of the lowest oil accumulations of all the mice. This mouse had decreasing food consumption throughout the week [(weeks): 1=21.41 g, 2=20.57 g, 3=20.66 g, 4=18.36 g], possibly suggesting that it caused the decreasing oil accumulation [(weeks): 1=2 color ranks, 2=0 color ranks, 3=1 color ranks, 4=0 color ranks].

The unusually high weight gain could be explained partially by the growth curve for mice. This is noticeable in humans as well; the growth curve for humans is attributed to pubescent increase in body mass. Also, Ntambi’s research showed that the results of SCD1 gene removal are more dramatic in males than in females; therefore, gender could explain the weight gain, since all the mice were female except #13. Another explanation could be duration of experimentation. This data was collected over a 4 week period, showing minimal signs of weight retardation concurrent with chemical intake; however, Ntambi’s data was collected over a 23
week period, showing minimal difference in weight retardation in females until week 20, and not a statistically significant difference until week 23. The largest amount of weight gain being in the first week accentuates the possibility that the unusually high weight gain can be attributed to the growth curve. To eliminate this variable, data will be collected when mice are older, eliminating the growth curve factor. The parental strain controls gained a larger average amount of weight than the SCD1 null strains; however, a t-test revealed that the parental strain controls did not gain a significantly larger amount of weight than the SCD1 null strains (p = 0.144783).

The SCD1 null strains who consumed the high-fat diet & all three chemicals (S, ALA, CoQ10) had a lesser color rank rise than the SCD1 null strain who consumed the high-fat diet & only the S and ALA mixture. On the contrary, the SCD1 null strains who consumed the high-fat diet & all three chemicals had a higher color rank rise than the SCD1 null strain who consumed the high-fat diet & only the CoQ10 mixture. This data suggests that the CoQ10 mixture was not as effective as the S and ALA mixture in improving the dermatology defects in these SCD1 null mice that refuse adiposity. This data also suggests that the combination of both mixtures was responsible for dragging the average oil accumulation down for the SCD1 null strains who consumed the high-fat diet & all three chemicals. [(average oil accumulation): SCD1 null on high-fat diet & only S and ALA mixture = 9 color rank raise, SCD1 null on high-fat diet & only CoQ10 mixture = 6 color rank raise, SCD1 null on high-fat diet & all three chemicals = 6.2 color rank raise] Thus, the Silica and ALA mixture was the most effective, followed by the combination of the Silica & ALA mixture and the CoQ10 mixture, and finally the CoQ10 mixture. A t-test confirmed that mice
receiving either no or some chemicals had significantly higher color rank rises than mice receiving all three chemicals (p= 0).

There were interesting observations during experimentation: 1) The parental strain controls were calm during swabbing for the Sudan III test, while the SCD1 null strains were extremely agitated or nervous. This could be attributed to the severe dermatology defects, similar to the way a human with sunburn (irritated skin) would be more sensitive when touched, or this could be a result of increased mobility and speed, secondary to increased oxygen consumption. This proposes the question: would SCD1 null mice exhibit increased mobility and speed as a side effect of increased oxygen consumption? 2) #3 and #13 were the most active mice, but the parental strain controls were much more active than the SCD1 null strains until week 3. The SCD1 null strains spent most of the monitored time resting under the wheel during weeks 1 and 2. The behavioral signs between the SCD1 null mice and their parental strain controls were significantly different, and when compared to humans, the SCD1 null mice could be phenotypically expressing the equivalent of antisocialism or even depression. Every SCD1 null strain slept in their food bowls and became more interactive from week 3 on, the same week as their oil accumulation spikes. This proposes the question: would oil accumulation favorably impact the anterior frontal lobe, the part of the brain heavily involved in personality, behavioral control, inhibition, motivation, and social understanding? 3) SCD1 null mice exhibited the most severe defects in their neck and upper back regions. Their lower dorsal area, near the lumbosacral (lower spine and surrounding areas), exhibited significantly less defects, but still exhibited dryness. This presents the
possibility that whatever process is blocking lipid absorption is exhibited near the rostral area (head end) of the mouse as opposed to the caudal area (tail end). This is probably related to an unequal distribution of sebaceous (oil) glands in the skin.

The hypothesis that Silica, Coenzyme Q10, and Alpha-Lipoic Acid would improve the dermatology defects in mice protected against adiposity was supported. The hypothesis that either Silica and Alpha-Lipoic Acid or Coenzyme Q10 would not improve the dermatology defects in mice protected against adiposity was rejected. All three chemicals resulted in oil accumulation; however, the Silica and ALA mixture was the most effective, followed by the combination of the Silica & ALA mixture and the CoQ10 mixture, and finally the CoQ10 mixture.

The three chemicals used in this research were chosen based on their properties that improve hair and skin. Silica stimulates collagen synthesis, which improves the condition of hair and nails. Certain skin and bone complaints can be treated with Silica, including fractures that are slow to heal, rough or peeling lips, acne, weak nails, and ingrown toenails. Coenzyme Q10 is a biologically active quinone (a common component of biologically relevant molecules) with an isoprenoid (generally the most common hydrocarbon found in the human body) side chain, similar in structure to vitamin K and vitamin E. It transfers electrons easily, acting as an antioxidant, which improves skin health and appearance and promotes energy production, especially in the heart. It is widely used for the treatment of heart disease (especially heart failure), gum diseases, and breast cancer. Alpha-Lipoic Acid is a natural antioxidant that helps regenerate glutathione, giving skin cells extra protection. It is a vitamin-like antioxidant that helps neutralize cell-damaging free
radicals, which can contribute to oxidative stress, which plays a role in the premature aging of cells. Also, it helps metabolize sugar in the body, especially in muscles, where it promotes energy (Wikipedia, 2007).

The Coenzyme Q10 paste included glycerin and ethanol because CoQ10 is lipid based; thus, it has very poor solubility in water. Consequently, an organic base was used to convert the CoQ10 powder into liquid form. Tangerine oil and magna sweet were added to give the taste of an orange rind. The results of this project depended on the mice consuming the chemicals, which were mixed into the diet, so the physiological measures of taste and smell were added with the prospect of increasing food consumption; thereby, increasing chemical ingestion.

The Sudan III test was used in this research because Sudan III stains neutral fat a red color, indicating how much oil is present depending on the shade red of the sample. Sudan III is prepared by mixing Sudan III powder into 95% ethanol until the solution is saturated red in color. This test serves as an oil indicator.

The high-fat diet used in this research was obtained from Research Diets in New Brunswick, NJ, as suggested by Dr. Ntambi. The diet was 60 kcal % fat, as was used in Ntambi’s research, and was distributed to each mouse in increments of 4 g per day, as suggested by Research Diets.

An important detail to note about the mice used in this research is that they were naturally mutated, meaning their mutation was produced through breeding to exhibit the same characteristics as Dr. Ntambi’s mice exhibited. Dr. Ntambi, on the other hand, specifically removed the SCD1 gene from these mice. This proposes the question; do the effects of SCD1 removal decrease through each generation of mice?
Post experimentation it was observed that the severity of the dermatological defects of the SCD1 null mice increased. [In order from most to least severe: #11, #10, #8, #12, #13, #7, #9]. During experimentation, there was considerable dryness, flakey skin, and redness; however, post experimentation, the hair became even dryer, the flakey skin remained, and the redness turned into bloody areas where patches of hair and skin were missing. The dermatology defects were not this severe prior to or during experimentation, proposing the question, do the clinical signs associated with being SCD1 null increase in severity over time? The defects were improving with chemicals (S, ALA, CoQ10) and only started regressing when chemical distribution ceased. This observation proposes the question, is oil accumulation through chemical intake in SCD1 null mice significantly altered when chemical contributors are continuous and revoked? This could be similar to how sugar affects the blood sugar in a sine curve method. The natural sugars in an apple will initially raise blood sugar above the original level; however, if sugar does not keep getting added into the blood stream, the blood sugar will plunge below the original level, but eventually will return to the original level. These chemicals could have raised the lipid level higher, giving the mice’s bodies more lipids to oxidize. If their bodies oxidize only a certain amount of lipids in a given time, this increased amount of lipids being retained, with the same amount of lipids being oxidized, may have allowed their bodies to accumulate more oil. When the experiment concluded and the mice began consuming normal food again, the decreased amount of lipid input, with the same amount of lipid oxidization, caused a regression and resulted in oil expenditure. Thus, although the defects improved with these chemicals, the defects worsened past the point of the original
defects when chemical consumption stopped. To confirm or deny this claim, mice will be monitored over a longer period of time, while consuming either a high-fat diet or a high-fat diet with chemicals. During Experimentation, oil accumulation will be tracked while increments of chemicals are both added into the diet and revoked from the diet. This will determine whether SCD1 null mice need a continuous supply of chemicals to benefit from increased oil accumulation through chemical intake.

The purpose of biomedical research and testing is to understand the living body and what goes wrong in disease, and to develop safe and effective ways of preventing or treating those diseases. It has been estimated that animal research and testing accounts for about 10% of all biomedical research. Animals are vital in all stages of this undertaking, not just in safety testing. Although vital, the use of living animals is just one of three main research methods in medicine and biology. These methods are not alternatives to each other; they are complementary methods that are all equally valid and all contribute vital pieces to the overall picture. The non-animal techniques are: 1) *in vitro* techniques, involving the study of isolated molecules, cells and tissues (which may come from humans, animals, micro-organisms or even plants). This gives useful information about interactions between molecules, within or between cells, or about organ function. 2) study of human beings and populations, which can give very useful information about the body in health and disease and about the distribution of diseases in society, but is limited by what is considered ethical. Animals are only used when the answers to scientific questions cannot be obtained in any other way or when it is necessary to see what happens in the whole living body,
but the use of human subjects would not be ethically acceptable, as in this research (RDS2, 2007).

APPLICATION

The ultimate goal of this research was to counter the dermatological defects in mice protected against adiposity. This research could contribute to making the genetic deletion of the stearoyl-Coenzyme A desaturate 1 (SCD1) gene less harmful to mice by reducing the side effects of dry skin and hair caused by a lack of oil and fat not being synthesized. **Scientific research of SCD1 gene activities has the potential to advance treatments for a wide range of human diseases including diabetes, atherosclerosis, cancer, obesity, and viral infection, which are associated with high SCD1 activity. Furthermore, SCD1 research may provide a healthy alternative for anorexia and bulimia**. With this potential development for humans comes the idea that one can retard weight gain, reduce body fat mass, increase oxygen consumption, increase fatty acid oxidation, and increase insulin sensitivity, while consuming a high-fat diet and without having the deleterious side effects of dry skin or hair, which was the premise of this research.

A supplementary approach was taken in this research with the prospect of developing a temporary solution, a molecule inhibitor for humans, which suppresses the SCD1 gene while delivering the necessary chemical dosages. This temporary solution would facilitate the benefits, while preventing the detriments, of being SCD1 null, until the genetic aspect of when SCD1 gene removal transitions from being beneficial to harmful is discovered and applied.

Haley Daus / 11th Grade
FURTHER RESEARCH

1. The effects of continuous and revoked chemicals on oil accumulation in mice protected against adiposity will be examined. This will determine whether SCD1 null mice require a continuous supply of chemicals in order to benefit from SCD1 gene removal.

2. Behavior versus oil accumulation will be examined while mice are placed on different increments of chemical consumption. This may confirm or deny my previous claim that oil accumulation positively impacts the anterior frontal lobe of the brain.

3. A longer duration of experimentation will be allowed to determine an extended pattern of oil accumulation and weight gain.

4. The cardiac and metabolic stability of the mice could be monitored without Silica, Alpha-Lipoic Acid, and Coenzyme Q10, and with those three chemicals, to determine the long-term effects of SCD1 gene removal. This would include examining the amount of fat build up around the heart and other organs and measurement of oxygen consumption.

5. Research could be repeated with brain function test to determine whether severe oil deficiency effects the brain’s memory or logic.

6. Acid samples could be taken weekly from blood or urine to determine if there is an acid deficiency.

7. A range of chemical dosages could be distributed to determine a safe minimum and maximum chemical intake.
8. The mice could be placed on a high-sugar diet, instead of a high-fat diet, in order to determine whether the mice are still able to refuse adiposity.

9. A drug could be developed that suppresses the SCD1 gene in humans while replenishing essential nutrients that prevent dry skin and hair while promoting brain and heart health.

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Haley Daus / 11th Grade


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Haley Daus / 11th Grade