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AN ANALYSIS OF THE VARIATION IN HUMAN HEIGHT BASED ON PHENOTYPIC TRAITS IN HEREDITY

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ABSTRACT

The phenotypic traits in offspring's are often dependent on that of the parents. Taking height as the phenotypic trait, we can obtain a regression model for parents and their offspring's, and also test for the significance of the overall model. As the detection of causal links between genetic and phenotypic variation is accelerating, a re-examination for our conceptual tools may help by finding unifying principles within the swarm of data. The analysis from this research reveal that Multiple Correlation Coefficient is 0.87 which implies that there is a strong positive relationship between Offspring's Growth and Parent's Phenotype with R-squared value of 0.75 which implies that 75% of Offspring's attribute will result from Parent Phenotype. A Multiple Regression model given as offspring's Growth = 58.589 + 0.535Father's Height +0.140Mother's Height.The value 0.535 implies the contribution per unit change of Father's Height in Offspring's Growth and 0.140 is the contribution per unit change of Mothers Height in Offspring's Growth. The Multiple Regression model is statistically significant with p-value of 0.003 less than 0.05. Multicolinearity test in the analysis shows that the predictor variables are moderately Collinear observing the Variance Inflation Factor (VIF). The recommendations require parents to put more efforts on preventing negative phenotypic traits from transferring from the environment to their offspring and couples should try to check their Genotype and Blood group since they highly contribute to the Offspring characteristics.

Keywords: Phenotype**,** Genotype, Offspring, Traits, Alleles, Heterozygous, Homozygous, DNA

INTRODUCTION

Phenotype comprises all the observable characteristics of an organism that result from the interaction of its genotype (total genetic inheritance) with the environment. Examples of observable characteristics include behaviour, biochemical properties, colour, shape, and size. The phenotype may change constantly throughout the life of an individual because of environmental changes and the physiological and morphological changes associated with aging (Engelman et al., 2015). Different environments can influence the development of inherited traits (as size, for example, is affected by available food supply) and alter expression by similar genotypes (for example, twins maturing in dissimilar families). In nature, the influence of the environment forms the basis of natural selection, which initially works on individuals, favouring the survival of those organisms with phenotypes best suited to their current environments. The survival advantage conferred to individuals exhibiting such phenotypes enables those individuals to reproduce with relatively high rates of success and thereby pass on the successful genotypes to subsequent generations (Bloom et al., 2013).

Populations and species differ from each other, the concept of phenotype, which corresponds to the observable attributes of an individual, was coined in opposition to the genotype, the inherited material transmitted by gametes. There was an early proposal that genotypes and phenotypes form two fundamentally different levels of biological abstraction (Johannsen, 2011).

The challenge has been to understand how they articulate with each other, how genotypes maps onto phenotypes in the last 8 years, more that 1000 examples of DNA sequence changes have been linked to naturally occurring non-deleterious phenotypic differences between individuals or species in Eukaryotes (Martin & Orgogozo, 2013).

Compiling the genetic determinants of disease related phenotypes totals more than 4300 entries and a total of 2493 published Genome-wide Association Studies (GWAS) have been uncovering a wealth of sites in the genome that are statistically associated to complex traits (Welter et al., 2014). As the detection of causal links between genetic and phenotypic variation is accelerating, a re-examination for our conceptual tools may help by finding unifying principles within the swarm of data. Here we reflect on the relationship between genotypes and phenotypes and we address this phenomenon to biologists who are willing to challenge their current understanding of phenotypes. We single out one useful point of view, the differential view. We then show that this simple framework remains insightful in the context of pervasive environmental effects (Dawkins, 2013).

Mutations isolated from laboratory strains have been instrumental to the understanding of the Genotypic and Phenotypic(GP) map. Under the classical scheme, a mutation is compared to a wild – type reference, and its phenotypic effects are used to infer gene function. This framework often leads to a semantic shortcut: from a genetic change causing a variation in phenotype, it is often convenient to assimilate the corresponding gene as a causal determinant of a trait (Keller, 2012).

In fact, a gene alone can neither cause an observable phenotypic trait, nor can it be necessary and sufficient to the emergence of observable characteristics. Genes need a cellular environment, the combined action of multiple other genes, as well as certain physio–chemical conditions to have an observable effect on organisms. For example, brown hair pigmentation in one human being is not just a product of the genes coding for pigment synthesizing enzymes but also of the presence of cells producing pigments of relevant substrate molecules (Such as tyrosine for melanin), and of the amount of received sun light (Liu, Wen & Kayser, 2013).

Thus, the genetic reductionist approach, which only explores a few genetic parameters among the variety of causal factors, is vain to fully address the broad question of what makes hair brown, of what brings forth a particular biological structure or process in its entirety. Nevertheless, genetic reductionism can be perfectly appropriate for identifying genetic loci where

a change causes a phenotypic difference. A difference in hair colour between two individuals could be due in some cases to their genetic difference. We note, however, that not all phenotypic changes can be attributed to genetic changes. A difference in hair colour could also be caused by non- genetic factors such as age intensity, solar radiation or hair dyeing, or by a combination of genetic and non- genetic differences(Carroll, Grenier & Weatherbee, 2015). While modern genetics was in its infancy, Alfred Sturtevant formulated the question of the GP map in simple terms: "one of the central problems of biology is that of differentiation – how an egg develops into a complex many –celled organism? That is, of course, the traditional major problem of embryology; but it also appears in genetics in the form of the question, how do genes produce their effects? (Sturtevant, 2012).

For long some geneticists may have thought that they were dissecting the morphogenetic mechanisms underlying the formation of phenotypic traits, while their experimental approach were in fact uncovering genes whose absence or alteration (mutations deletions, duplications,and rearrangements) leads to phenotypic differences. In fact, the sentence "Your hair is brown" can be interpreted either as an absolute observation (a description of a particular assemblage of molecules containing defined levels of the dark pigment eumelanin and of the pale pigment pheomelanin) or with implicit reference to other possibilities (it is brown and not of another color) (French, Daborn, & Le Goff, 2014).

Misconceptions arise because phenotypes are usually defined relative to possibilities that are not formulated explicitly. Our minds and our language often tend to confuse the objects whose variation is under consideration with the variation itself (Palmer, 2014).

In genetics the objects of interest (for example a given genotype an allele or a phenotype) deserve to be defined relatively to another reference state. In summary, the classical genetic reductionist approach is inherently unable to elucidate all the factors responsible for observable characteristics in the living world, but it is a powerful and relevant method for dissecting the genetic levers of heritable phenotypic variation (Salazar-Ciudad., & Marín-Riera, 2013).

Focusing on phenotypic variation between individuals rather than on absolute characters present in single organisms is key to better comprehend the genetic causes of phenotypic diversity (Stotz, 2012).

In current genome annotation database, a gene is usually defined as a stretch of nucleic acid that is transcribed and codes for RNA or a polypeptide with a known or presumed function. The genetic locus underlying a phenotypic difference is not necessarily a gene in the strict sense; it span a particular base-pair or a coding region, or extend to a gene cluster.(Gerstein et al., 2014).

The concept of gene in developmental biology and in current genome annotation databases is distinct from the concept of gene in evolutionary biology. Here the emphasis is not on the gene itself as defined in genome databases, but rather on a case–by–case functional partitioning of the genome into difference – making loci (Gompel & Prud'homme (2019), Graur, Zheng & Azevedo,(2015).

The difference in color pigmentation between dark and light – colored beach mice mentioned previously is not only due to mutations in agouti but also to a coding mutation is the gene that decreases pigmentation (Steiner, Weber & Hoekstra (2013),Tautz & Schmid (2018).

For an unknown number of segments of the human genome current variation among human beings or among primates reflects the impact of natural selection in these cases while population processes and event have played a role, differences in fitness between individuals with different alleles have had greater influence(Gjuvsland, et al; 2013).

Other studies particularly those that focus on reproductive behaviour such as mating and offspring care, examine an individua's decisions when situated in a particular social milieu (Stewart, Schilling & Wilson, 2017), (Coen, 2012).

DNA transmitted to the next generation has been shielded from most of the interactions that occur during an organism's lifetime (both within the organism and with the environment), there is a barrier to ''peculiar'' traits (Johannsen's label for traits acquired during the specific development of the parents) being passed on to their offspring. (A key part of this shielding is the one-way transcription of DNA to RNA {which then codes for the amino acids that make up proteins}, not in general, transcription in the other direction). While the genotype-phenotype distinction can be seen to signify the existence of this barrier, there is a long history of researchers claiming to show ways around it (Wilkins, 2014).

MATERIALS AND METHOD

The data is on the heights of the parents and offspring's from two hundred family folders at the records office of Dr Gwamna Awan General Hospital Kakuri-Kaduna South in Kaduna State. In a trivariate distribution in which each of the variable Y, X_1, and X_2 has n observations the multiple correlation coefficient of Y on X_1 and X_2 is denoted by $R_{y;x_1,x_2}$ which gives the simple correlation coefficient between Y and the joint effect of X_1 and X_2 on Y. The strength of the association is measured by the sample multiple correlation coefficient R. The multiple correlation coefficient between a dependent variable Y and two independent variables X_1 and X_2 is :

$$R_{y,x_1,x_2} = \sqrt{\frac{r_{y.x_1}^2 + r_{y.x_2}^2 - 2r_{y.x_1}\left(r_{y.x_2}\right)\left(r_{x_1.x_2}\right)}{1-r_{x_1.x_2}^2}} \quad \ldots\ldots\ldots (1)$$

And the pairwise product moment correlation coefficients is:

$$r_{x,y} = \frac{n\sum_{i=1}^{n} X_i Y_i - \sum_{i=1}^{n} X_{ii} \sum_{i=1}^{n} Y_i}{\sqrt{\left(n\sum_{i=1}^{n} X^2{}_i - \left(\sum_{i=1}^{n} X_i\right)^2\right)\left(n\sum_{i=1}^{n} Y^2{}_i - \left(\sum_{i=1}^{n} Y_i\right)^2\right)}} \quad \ldots\ldots\ldots (2)$$

Suppose we seek a model for k predictor variables from $y = a + b_1 x_1 + b_2 x_2 + b_3 x_3 + \ldots + b_k x_k + e_i \quad --(3)$

Most often, our interest will be to estimate a and $b_1, b_2, \ldots, b_k$; To test $b_1, b_2, \ldots, b_k$ for the significance of the associated predictor; use the regression equation to estimate Y from $X_1, X_2, \ldots, X_k$ and measure the error involved in the estimation. The basic assumptions for the multiple regression models are outlined as follows:

1 Randomness of e_i. The variables e_i is a real random variable which is normally distributed with zero mean for each value of X. That is $e_i : \ N\left(0, \sigma_{ei}^2\right)$

2 Homoscedasticity. The variance of each e_i is the same for all the X_i values. That is $\in\left(e_i^2\right) = \sigma_{ei}^2$ is constant.

3 No multi-collinearity: Multiple regression assumes that the independent variables are not highly correlated with each other. This assumption is tested using variance inflation factor (VIF) values.

4 No errors of measurement in the X's. That is, the explanatory variables are measured without errors (deterministic).

5 Identifiability of the function, the relationship being studies is identified.

Multicollinearity can be assessed using the variance inflation factors (VIF), which is defined for each predictor variable as;

VIF= $\left(1-R_j^2\right)^{-1}$ where; R_j^2 is the multiple coefficient of determination for the regression of X_j on the other explanatory variables (a regression that does not involve the response variable Y). This identity separates the influences of several distinct factors on the variance of the coefficient estimate. When R_j^2 is near 1, it indicates significant correlation with the remaining predictor variables, which results in a large value of VIF. A VIF larger than 10 is usually taken as an indicator of high multicollinearity. Moreover, the VIF reflects all other factors that influence the uncertainty in the coefficient estimates. The VIF equals to 1 when the vector X_j is orthogonal to each column of the design matrix for the regression of X_j on the other covariates. By contrast, the VIF is greater than 1 when the vector X_j is not orthogonal to all columns of the design matrix for the regression of X_j on the other predictors. Finally, we note that the VIF is invariant to the scaling of the variables (that is, we could scale each variable X_j by a constant C_j without changing the (VIF).

Table 1: The basic computations in applying ANOVA in regression

Source of Variation	Sum of square	D.F	Mean square	F
Regression	$\hat{B}'(X'Y) - n\bar{Y}^2$	$p-1$	$MSR = \frac{\hat{B}'(X'Y) - n\bar{Y}^2}{p-1}$	$F = \frac{MSR}{MSE}$
Residual	$Y'Y - \hat{B}'(X'Y)$	$n-p$	$MSE = \frac{Y'Y - \hat{B}'(X'Y)}{n-p}$	
Total	$Y'Y - n\bar{Y}^2$			

Where; p is the number of parameters in the model

RESULTS AND DISCUSSION

The model summary below **shows** the relationship between parents and grown up offspring's using offspring's height as the dependent variable

Table 2: model summary

Model	R	R Square	Adjusted R Square	Std. Error of the Estimate
1	.870[a]	.757	.754	1.74682

a. Predictors: (constant), Mothers height (x_2), Fathers height (x_1)

The column labelled R show that linear relationship (correlation=0.870) between the offspring's and the parents height. The column that is labelled as R square (R^2) is the square of the correlation coefficient is telling us that 75.7% (0.757) of the growth in offspring's is explained by the parents height.

The SPSS output below shows the degree of the relationship between parents and their offspring's, taking height as the phenotypic trait.

Table 3: The degree of the relationship between parents and their offspring's

		FATHERS HEIGHT (X_1)	MOTHERS HEIGHT (X_2)	GROWN UP OFFSPRINGS HEIGHT (Y)
FATHERS HEIGHT (X_1)	Pearson Correlation	1	.524**	.857**
	Sig. (2-tailed)		.003	.003
	N	200	200	200
MOTHERS HEIGHT (X_2)	Pearson Correlation	.524**	1	.575**
	Sig. (2-tailed)	.003		.003
	N	200	200	200
OFFSPRINGS HEIGHT (Y)	Pearson Correlation	.857**	.575**	1
	Sig. (2-tailed)	.003	.003	
	N	200	200	200

**. Correlation is significant at the 0.01 level (2-tailed).

Table 3 above shows that the correlation between the father's heights, mother's heights is (0.524), the p-value of the correlation coefficient (0.003) is less than α-value (0.01) and we therefore conclude that the correlation between the fathers and mothers heights is significant at 1% level. While the correlation between the father's and offspring's is (0.857), the p-value of the correlation coefficient (0.003) is less than α-value (0.01), we therefore conclude that the correlation between the father's and offspring's is significant at 1% level. And also the correlation between the mother's and offspring's is (0.575) the p-value of the correlation coefficient is (0.003), is less than the α-value (0.01), we therefore conclude that the correlation between mother's and offspring's is significant at 1% level.

The SPSS output below is on the multiple regression model for parents and their offspring's. It test how significant is the individual parameter of the model and test for multicollinearity.

Table 4: The Standardized and unstandardized coefficient between parents and their offspring's

Model		Unstandardized Coefficients		Standardized Coefficients	t	Sig.
		B	Std. Error	Beta		
1	(Constant)	58.589	5.130	-	11.422	.001
	FATHERS HEIGHT (X_1)	.535	.029	.766	18.551	.001
	MOTHERS HEIGHT (X_2))	.140	.033	.175	4.231	.001

a. Dependent Variable: OFFSPRINGS HEIGHT (Y)

From table 4 above, we obtain the multiple regression model, $Y_t = 58.589+0.535X_1+0.140X_2$.

Where Y_t is the offspring's height, X_1 is the father's height and X_2 is the mother's height and also the height of the offspring's increases by 0.535 and 0.140 of the father and the mother respectively

Table 5: Test for Multicollinearity

	Collinearity Statistics	
Variables	Tolerance	VIF
Father's Height	0.726	1.378
Mother's Height	0.726	1.378

A rule of thumb for interpreting the variance inflation factor (VIF). A VIF of 1 indicates that the variables are not correlated; VIF between 1and 5 shows moderately correlated variables, a VIF greater than 5 indicates a highly correlated. Since our VIF value is 1.378 which is between 1and 5 it shows the variables are moderately correlated. Since it does not exceed 5, there is no need for further investigation

Table 6: Test of significance of the model using ANOVA

Model		Sum of Squares	Df	Mean Square	F	Sig.
1	Regression	1867.833	2	933.916	306.064	.002[b]
	Residual	601.122	197	3.051		
	Total	2468.955	199			

a. Dependent Variable: OFFSPRINGS HEIGHT (Y)

Reject H_0 if p-value <α-value (0.05), otherwise do not reject. Table 6 above shows that the p-value of the ANOVA of the model (0.002) is less than α-value (0.05), this implies that the model is fit for use.

CONCLUSION

Based on the analysis and results obtained, we conclude that most phenotypic characteristic in grown up offsprings are dependent on phenotype of the parents. Observing phenotypic trait in offspring is of great necessity since there are several claims in family history that sometimes results to doubt among family members. Researchers have conducted several analyses using various kinds of variables in determining the phenotypic trait in an offspring. This research provides a medium through which family member can be identified and grouped using the height as the phenotypic trait. **T**he results obtained leads to the following recommendations.

i. Government should create awareness to the parents in order to be able to counter negative phenotypic traits.

ii. Parents should put more efforts on preventing negative phenotypic traits from transferring from the environment to their grown-up offspring.

iii. Couples should try to check their Genotype and Blood group since they highly contribute to the Offspring characteristics.

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APPENDIX 1

The table below shows the parents height and their offspring's

S/N	FATHER HEIGHT (CM) X_1	MOTHER HEIGHT (CM) X_2	OFFSPRINGS HEIGHT (CM) Y
1	171	168	178,169,173
2	172	167	179,175,168,170
3	168	165	174,169
4	180	175	185,182,179,175,170
5	176	161	179,168,177,174
6	165	172	174,179,170,
7	170	179	180,184,178,175
8	175	169	179,176,173,171
9	160	159	171,168,165
10	169	165	170,175,178,173
11	172	170	175,179,173
12	179	175	182,179,185,170
13	165	160	165,168,170
14	170	168	175,170,178,169
15	162	165	170,169,172,168
16	172	160	178,169,175
17	170	160	178,169,175,173,170
18	180	169	184,179,182,176
19	169	170	174,172,176
20	175	169	179,176,
21	169	170	174,176,172,178
22	165	159	170,173,169
23	170	169	176,174,179
24	175	165	178,176,170,173
25	179	170	179,184,186
26	169	165	170,178,175,173
27	170	160	178,173,176
28	179	169	182,180,178
29	166	169	168,170,176,172
30	172	170	179,175,177,173
31	165	160	170,168,165,174,176
32	169	160	172,175,168
33	170	168	176,170,167,173
34	175	165	178,174,169
35	168	165	170,176,173,167
36	170	169	173,179,176
37	174	170	179,175,170,172,177
38	165	160	172,170,168
39	180	169	179,176,185,182
40	160	165	170,168,173
41	169	172	173,176,169
42	176	168	179,176,172
43	170	175	178,182,173
44	175	169	174,170,177
45	165	160	171,168,176
46	169	162	172,174,169,165
47	178	169	172,176,178
48	185	170	186,183,177,180
49	169	165	173,170,165,168
50	175	165	178,176,174,
51	169	160	170,175,167,172
52	172	164	179,176,170,166
53	170	168	172,175,169
54	165	160	165,170,173
55	169	160	170,176,167
56	170	165	175,173,170,168
57	169	166	170,167,172
58	172	165	176,178,170,174
59	178	168	176,180,170
60	170	169	175,178,168,173
61	175	170	177,174,180
62	185	169	186,175,182,178
63	165	160	167,170,174

64	170	160	175,173,168
65	172	169	173,167,177,170
66	165	160	168,171,175
67	180	175	186,182,176,180
68	170	165	177,170,174
69	170	160	167,170,175
70	165	160	170,165,168,175
71	186	169	189,185,179
72	169	165	175,168,171
73	170	168	173,170,176,165
74	175	160	179,176,169,174
75	169	165	172,176,168
76	174	170	178,170,174,167
77	175	160	179,176,174
78	169	165	170,173,175,165
79	170	166	167,172,176
80	175	160	178,166,175,170
81	165	169	170,167,174
82	168	170	175,168,170
83	169	160	165,169,173
84	170	165	173,176,171,167
85	169	162	170,168,174
86	176	168	179,175,183,170
87	176	165	180,176,172,169
88	170	169	175,172,178,168
89	175	160	178,176,169
90	173	168	176,173
91	182	174	186,184,179,176
92	169	160	170,176,167
93	170	165	174,170,177,168
94	180	169	181,178,184
95	175	169	178,174,167,170
96	179	165	176,184,173,179
97	169	160	170,174,167
98	170	168	172,176,170
99	184	170	186,175,179,182
100	169	165	179,174,165
101	176	160	179,175,170
102	170	165	174,169,170,176
103	168	165	170,174,167
104	169	160	172,176,168
105	175	160	167,179,175,170
106	169	166	170,174,165
107	170	169	167,175,170
108	175	170	178,174,182,170
109	176	169	179,176,171
110	170	165	168,174,177
111	169	160	170,165,174,162
112	172	169	176,174,171
113	170	165	172,167,170,175
114	169	160	170,173,166
115	176	168	178,174,169,181
116	170	165	172,175,167,170
117	182	169	186,176,179
118	172	166	175,178,168,173
119	177	170	181,174,178
120	169	165	170,165,162,174
121	170	169	173,175,168
122	169	165	170,172,165,176
123	170	169	171,175,167
124	182	165	186,169,175,180
125	169	160	168,173,175
126	169	168	170,174,176,165
127	175	169	178,176,172
128	180	173	184,180,177,173
129	165	169	168,171,175

130	170	169	167,173,176
131	167	168	163,170,165,174
132	169	160	168,173,176
133	172	165	178,167,170,174
134	170	169	173,168,178,180
135	182	170	183,176,186
136	169	165	170,175,178,168
137	170	169	170,174,167
138	168	169	171,175,179,168
139	170	165	173,178
140	175	169	168,174,180
141	168	165	169,174
142	172	160	173,178
143	180	169	181,178,175,171
144	172	165	178,170
145	182	169	186,182,179
146	170	160	173,176,163,170
147	169	165	170,174
148	174	160	175,179,169
149	169	165	171,176,167
150	170	160	167,170,174,179
151	167	165	171,166
152	168	160	175,170,168
153	169	160	172,168,176
154	183	174	183,178
155	178	173	179,174,183
156	170	168	178,173,168
157	176	170	176,180,
158	174	169	178,175,171,168
159	184	178	185,181,178
160	178	173	179,176,184
161	182	170	179,175,170,186
162	175	169	178,174
163	173	165	178,175,171
164	177	170	176,170,168
165	169	164	168,173,176,170
166	174	169	177,173
167	170	165	178,174,169
168	175	169	175,179,170
169	180	175	182,179
170	179	169	180,178,175,184
171	169	160	170,175,178
172	176	174	179,176,169
173	174	169	177,173
174	175	173	180,177,173
175	178	170	180,174
176	169	165	173,166,170,176
177	170	173	172,174,167
178	168	160	175,171,168,165
179	176	169	176,170,180
180	170	168	178,173,169
181	177	167	176,180
182	172	170	179,175,170
183	180	176	180,177,184
184	170	165	175,172,169,165
185	169	160	178,173,167
186	175	169	176,170,168,179
187	170	165	170,175,179
188	169	160	171,175,168
189	173	169	176,174,169
190	183	170	184,180,177
191	165	160	169,165,173,176
192	169	163	169,174
193	170	169	175,168,170
194	174	172	178,174,171,167
195	180	170	183,178
196	171	168	176,172,168

197	175	172	178,171
198	169	165	170,175,168,165
199	182	170	184,178
200	179	168	180,184,178

APPENDIX 2

The table below shows father's height, mother's height and average height of offspring's

S/N	FATHER HEIGHT(CM) X_1	MOTHER HEIGHT (CM) X_2	OFFSPRINGS HEIGHT (CM) Y
1	171	168	173
2	172	167	173
3	168	165	172
4	180	175	178
5	176	161	175
6	165	172	174
7	170	179	179
8	175	169	175
9	160	159	168
10	169	165	174
11	172	170	176
12	179	175	179
13	165	160	168
14	170	168	173
15	162	165	170
16	172	160	174
17	170	160	173
18	180	169	180
19	169	170	174
20	175	169	176
21	169	170	175
22	165	159	171
23	170	169	176
24	175	165	174
25	179	170	183
26	169	165	174
27	170	160	176
28	179	169	180
29	166	169	172
30	172	170	176
31	165	160	171
32	169	160	172
33	170	168	172
34	175	165	174
35	168	165	172
36	170	169	176
37	174	170	175
38	165	160	170
39	180	169	181
40	160	165	170
41	169	172	173
42	176	168	176
43	170	175	178
44	175	169	174
45	165	160	172
46	169	162	170
47	178	169	175
48	185	170	182
49	169	165	169
50	175	165	176
51	169	160	171
52	172	164	173
53	170	168	172
54	165	160	169
55	169	160	171
56	170	165	172

57	169	166	170
58	172	165	175
59	178	168	175
60	170	169	174
61	175	170	177
62	185	169	180
63	165	160	170
64	170	160	172
65	172	169	172
66	165	160	171
67	180	175	181
68	170	165	174
69	170	160	171
70	165	160	170
71	186	169	184
72	169	165	171
73	170	168	171
74	175	160	175
75	169	165	172
76	174	170	172
77	175	160	176
78	169	165	171
79	170	166	171
80	175	160	172
81	165	169	170
82	168	170	171
83	169	160	169
84	170	165	172
85	169	162	171
86	176	168	177
87	176	165	174
88	170	169	173
89	175	160	174
90	173	168	175
91	182	174	181
92	169	160	171
93	170	165	172
94	180	169	181
95	175	169	172
96	179	165	178
97	169	160	170
98	170	168	173
99	184	170	181
100	169	165	173
101	176	160	175
102	170	165	172
103	168	165	170
104	169	160	172
105	175	160	173
106	169	166	170
107	170	169	171
108	175	170	176
109	176	169	175
110	170	165	173
111	169	160	168
112	172	169	174
113	170	165	171
114	169	160	170
115	176	168	176
116	170	165	171
117	182	169	180
118	172	166	174
119	177	170	178
120	169	165	168
121	170	169	172
122	169	165	171
123	170	169	171

124	182	165	178
125	169	160	172
126	169	168	171
127	175	169	175
128	180	173	179
129	165	169	171
130	170	169	172
131	167	168	168
132	169	160	172
133	172	165	172
134	170	169	175
135	182	170	182
136	169	165	173
137	170	169	170
138	168	169	173
139	170	165	176
140	175	169	174
141	168	165	172
142	172	160	176
143	180	169	176
144	172	165	174
145	182	169	182
146	170	160	171
147	169	165	172
148	174	160	174
149	169	165	171
150	170	160	173
151	167	165	169
152	168	160	171
153	169	160	172
154	183	174	181
155	178	173	179
156	170	168	173
157	176	170	178
158	174	169	173
159	184	178	181
160	178	173	180
161	182	170	178
162	175	169	176
163	173	165	175
164	177	170	171
165	169	164	172
166	174	169	175
167	170	165	174
168	175	169	175
169	180	175	181
170	179	169	179
171	169	160	174
172	176	174	175
173	174	169	175
174	175	173	177
175	178	170	177
176	169	165	171
177	170	173	171
178	168	160	170
179	176	169	175
180	170	168	173
181	177	167	178
182	172	170	175
183	180	176	180
184	170	165	170
185	169	160	173
186	175	169	173
187	170	165	175
188	169	160	171
189	173	169	173
190	183	170	180

191	165	160	171
192	169	163	172
193	170	169	171
194	174	172	173
195	180	170	181
196	171	168	172
197	175	172	175
198	169	165	170
199	182	170	181
200	179	168	181