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EXPLORATIONS

A JOURNAL OF RESEARCH AT THE UNIVERSITY OF MAINE AT ORONO

Cover

The painting reproduced on the cover is a 22" by 30" acrylic on paper entitled Passage-10, *by James Linehan, Assistant Professor of Art at the University of Maine at Orono, where he teaches painting.*

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EXPLORATIONS

A JOURNAL OF RESEARCH
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POLYUNSATURATED FATS:

are they killing us?

Our story about polyunsaturated fats is the story of a hypothesis: its origins, maturation, and open-ended future. Methodology is determined; equipment is organized; data collected and eventually analyzed before conclusions are drawn, tested and redrawn. We are presenting a real hypothesis: one in the process of testing amid the frustrating awareness of the results' importance to people and the stringent limitations of budgets. Our story unfolds in the 1970s in Minamata Bay in Japan.

by Linda J. Kling

Among the many fears and concerns of the 1970s was the *mercury scare*: clear evidence of the involvement of mercury as the etiological factor in the development of Minamata disease had recently been revealed. Minamata disease was the cause of death of numerous fishermen and their families around Minamata Bay in Japan. The consumption of mercury-contaminated fish was linked with the disease. In addition, accidental consumption of seed grain coated with a mercurial bactericide-fungicide agent was reported responsible for the deaths of at least 460 patients and for the hospitalization of more than 6,000 others in Iraq. The industrial uses of mercury were widespread, and mercury, as an environmental contaminate, was in the limelight.

A report in *Science* magazine on the protective effects of selenium against mercury toxicity caught my attention, and I was intrigued by the relationship between environmental toxicants and diet.

The newly recognized role of selenium in decreasing the toxicity of mercury was interesting. Although selenium had been recognized as an essential trace nutrient in 1957, a biological function for selenium had just been elucidated by scientists at the University of Wisconsin. These scientists had isolated an enzyme, glutathion peroxidase (GSH-Px), which had selenium as a necessary structural component. This enzyme speeds up the biological breakdown of harmful peroxides in the body.

Peroxides are compounds formed in tissues as natural consequences of biological reactions. Although peroxides occur naturally even in healthy states, they are potentially harmful, and the healthy cell has the ability to break these products down into compounds that are inert. When there is excessive production of peroxides, the body's natural defense systems cannot break them down fast enough and they initiate a series of steps that ultimately result in the breakdown of essential biological components such as proteins and fatty acids. Peroxides are similar to compounds found during exposure to radioactivity.

The newly recognized function of selenium as an integral part of an enzyme that could break down harmful peroxides clarified observations that linked selenium and vitamin E. Vitamin E had long been suspected of being a *biological antitoxicant*, a substance in the body that prevents the formation of peroxides. Since the initial discovery of selenium in 1957, it was observed that selenium could prevent or mitigate some of the deficiency symptoms of vitamin E. With the discovery of selenium as a part of glutathion peroxidase (GSH-Px), a complex defense mechanism began to emerge with vitamin E preventing the formation of peroxides and selenium (GSH-Px) breaking down peroxides once formed. This was a major

breakthrough explaining the link between selenium and vitamin E.

An observation that surfaced in the research on the role of selenium was the protection afforded by selenium against the toxicity of certain heavy metals. Even though certain heavy metals may be essential for normal function at very low levels, at higher levels these metals create disturbances in the body that lead to biological dysfunction and are potentially lethal. Exposure to heavy metals such as cadmium, mercury, silver and lead, have, throughout history, created problems. Mercury poisoning has been prominent at times among goldsmiths, mirror makers, and the expression, *mad as a hatter*, derives from the symptoms shown by workers engaged in the treatment of furs with mercuric nitrate.

The neurological hematological (blood) disturbances seen in many young children, particularly those growing up in ghetto areas with deteriorating housing, were attributed to the ingestion of lead-based paint. *Itia-itia* (Japanese for *hurts-hurts*) a disease prevalent in Japanese people living close to smelting plants, was found to be caused by the emission of cadmium during the smelting process.

The observations that selenium could mitigate the toxicity of certain heavy metals presented new questions regarding the possibility of a common denominator which could explain the cause of heavy metal toxicities.

Selenium was found to mitigate the toxicity of cadmium and silver but was ineffective in reducing lead toxicity. Increased dietary levels of vitamin E, however, could reduce the toxic effects of the ecologically relevant form of mercury, methylmercury. The mechanisms of the detoxification processes have remained largely unknown but may involve the antioxidant properties of selenium and vitamin E.

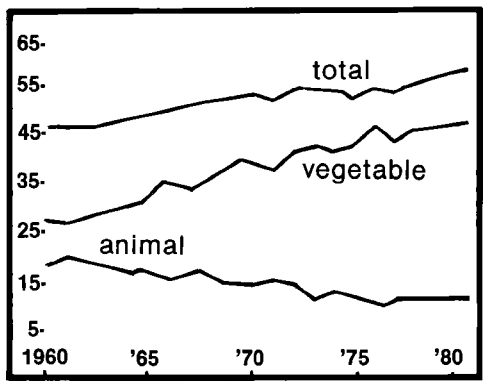
A very complex picture was evolving, involving selenium, vitamin E and heavy metal toxicity that is further complicated by the relationship of vitamin E and polyunsaturated fatty acids (PUFAs). Because PUFAs are highly susceptible to peroxidative damage (commonly called rancidity) the more PUFA in a diet, the greater the nutrient requirement of vitamin E to prevent harmful peroxides from forming and doing further damage.

The consumption of PUFA by the population of the United States has increased steadily over the past century. A shift from foods containing large amounts of saturated fatty acids, such as lard and butter, to foods high in polyunsaturated fatty acids is evident from recent surveys.

Linda J. Kling has been an Assistant Professor of Animal and Veterinary Sciences at UMO since 1980. She earned her Ph.D. at the University of Maryland, and her major research interest is in nutritional toxicology.

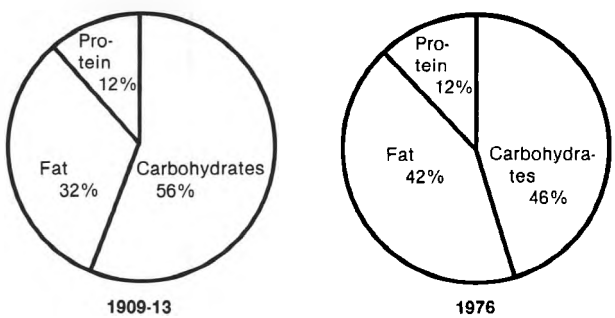
Fats and Oils Consumption

Pounds per capita



Vegetable sources of fat, which are high in PUFA, accounted for only 17 percent of the total fat consumed in 1909-1913. This increased to 25 percent 30 years ago and in 1976, vegetable sources accounted for 40 percent of the total fat consumed by people in the United States. Linoleic acid (found in corn oil, soybean oil, etc.) which is the predominant PUFA in foods, has more than doubled since early in the century increasing steadily from nine to 24 grams *per capita* daily. Moreover, the United States Senate Select Committee on Nutrition and Human Needs has published *Dietary Goals for the United States*, which advocates a shift from foods high in saturated fatty acids to foods having a higher proportion of PUFA, a recommendation that created a great deal of controversy in the scientific community. The controversy emerged between scientists who, in recognizing the relationship of diet and cardiovascular disease, thought that major dietary changes could alleviate major health concerns and more cautious scientists who recognized that any major dietary change could have equally dangerous health consequences, as yet unknown.

CHANGES IN THE MAKEUP OF TOTAL CALORIES



Composition of Dietary Fat

40.6%	saturated	35.7%
40.6%	monosaturated	40.5%
18.8%	polyunsaturated	23.8%

The latter scientists recognize that the effects of the long-range ingestion of high levels of PUFA have not been determined. Dietary fats can greatly influence the type of fats that make up the fatty components of our tissues. Among the risks that might be associated with an increased consumption of PUFA may be the development of certain types of tumors as documented in mice, or the induction of early aging of cells, particularly skin cells, due to harmful peroxides that can form from PUFA undergoing peroxidation.

Concurrent with the trend toward increased consumption of polyunsaturated fatty acids is the increased environmental exposure to the heavy metals cadmium, lead and mercury. While these heavy metals are all natural constituents of the environment, human exposure to them is continuously increasing. The expansion of industrial technology introduces increasing quantities of these toxicants into the ambient environment. Prior to the days of no-lead gasoline, approximately 350,000 tons of lead were used per year as gasoline additives.

The current average intake of these heavy metals from dietary sources appears to be approaching the weekly tolerable intake of heavy metals established by the joint Food and Agriculture Organization/World Health Organization Committee (FAO/WHO).

Recognizing the complex interactions of selenium and vitamin E with heavy metals such as cadmium, lead and mercury, along with the changing dietary fat patterns in the United States, a hypothesis began to emerge.

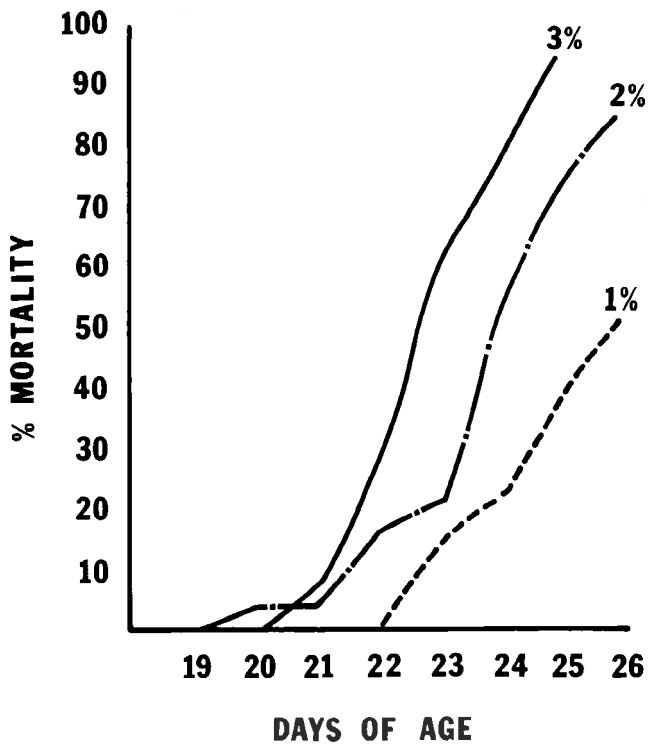
Could the continual exposure to low levels of cadmium, lead and mercury by individuals consuming ever-increasing amounts of polyunsaturated fatty acids in their diets result in an increased production of harmful peroxides?

A preliminary experiment has been conducted to determine if the dietary replacement of PUFA for saturated fats could affect animals' responses to the known toxicant, methylmercury.

Japanese quail were maintained on diets consisting of 1, 2, or 3 percent PUFA for two weeks. At two weeks of age, the quails' diets were intoxicated with an acute dosage of mercury.

Assistant Professor of Animal and Veterinary Sciences Linda J. Kling holds one of the small Japanese quail used in her toxicity experiments.

Laboratory animal technician Ruth Leclair adjusts the toxicity measuring equipment used in Kling's earliest experiments.



The response is depicted at left. The quail receiving the diets with 3 percent PUFA had a greater rate of mortality than those receiving the diets with 1 percent PUFA. This suggests that the consumption of diets with a high proportion of fat as PUFA may increase the risk of exposure to environmental toxicants. However, because of the interplay between vitamin E and PUFA, other interpretations are possible.

Further studies are necessary to determine whether mercury, and other potent environmental toxicants such as cadmium and lead, can initiate the destructive chain reaction events of peroxidation.

The goals seem clear, but the task is complex and difficult. The necessary methodology is being developed to accomplish this goal, and the instrumentation required is being painstakingly assembled from available bits and pieces of equipment.

Are polyunsaturated fats killing us? We are pursuing the answer.

The College of Education at UMO is inextricably linked with the people it serves. Its activities are tightly interwoven with the foundations of education; current problems and puzzles; challenges produced by an ever-changing social fabric, and the need to survey consistently and alertly for better ways to nurture and encourage schoolchildren and older students. One question education professionals are exploring is the Pygmalion effect: self-fulfilling prophecies. Specifically, education researchers have gathered and analyzed oceans of data to determine the aspiration levels of Maine's rural high school students. Those aspiration levels are distressingly low.

WHERE ARE THE

aspirations of Maine's rural high

by Robert A. Cobb, Walter G. McIntire, and Philip A. Pratt

There is a growing body of evidence that suggests that rural youth in general, and Maine youth in particular, have lower levels of academic and vocational aspirations than their counterparts in suburban and urban areas. A report from the Maine State Planning Office, based on information derived from college-bound students, cited findings that Maine youth have lower levels of academic and career aspirations than their agemates in the neighboring states of New Hampshire and Vermont (Barringer, 1984). The Preliminary Report of the Governor's Commission on the Status of Education in Maine also reported that through the Public Hearings conducted across Maine and direct observation of schools, Commission members encountered **aspirations among our students that limit their potential as individuals, and ours as a society** (Commission Report, 1984). Moreover, participants at a recent statewide conference on the aspirations of Maine's youth (initiated by UMO's College of Education), described in elaborate detail problems and examples of low aspirations in youth. The conference attracted nearly 700 people from 70 different communities in Maine. The objectives of the conference were

- to explore further what is meant by the term *aspirations*
- to assist communities and their schools to determine what the aspiration levels of their young people really are and whether or not they are satisfied with them
- to suggest ways in which they might raise aspiration levels among their young people.

The participants and planners committed themselves to further study of this phenomenon.

Earlier studies by the College of Education faculty had compared freshmen entering the University of Maine at Orono to a national sample of freshmen entering similar institutions across the country. Freshmen entering UMO, approximately 83 percent of whom graduated from Maine high schools, rate themselves above their national agemates in terms of their academic ability. However, Maine freshmen rate themselves below their national cohort in terms of leadership ability, social confidence and drive to achieve. In addition, through these earlier studies we have learned that more Maine youth are choosing to attend college without having a specific career goal in mind than their peers from across the country (McIntire and Pratt, 1984).

Robert A. Cobb is Dean of the College of Education. A native of Winthrop, Maine, where he grew up on a dairy farm, he earned a doctorate from Springfield College, Springfield, Massachusetts. His recent research interests have focused on the aspiration levels of rural youth, and more particularly, Maine's youth.

Walter G. McIntire is Professor of Education and Human Development. He has a Ph.D. in Counseling from the University of North Dakota and taught at the University of Connecticut before coming to UMO in 1973. His research interests are in family relations and self-concept development in children. He is also Editor of Research and Rural Education.

DREAMERS?

school students

More recently, the authors have been analyzing data collected from a longitudinal study entitled *High School and Beyond* (HSB) to determine if aspiration levels of rural students nationwide differ from those of students in urban and suburban settings. Our interests have been to determine if Maine's documented problem of low aspirations of its youth might be related to a larger, national phenomenon. This report presents an analysis of some of the aspirations-related variables which are part of this extraordinarily large data base.

The HSB data were collected by the National Center for Education Statistics (NCES) **to study longitudinally the educational, vocational, and personal development of high school students and the personal, familial, social, institutional, and cultural factors that may affect that development** (NCES, 1983).

In the 1980 sample of the HSB data base, students were selected through a two-stage, stratified probability sample with schools as the first stage units and students within schools as the second stage units of the sampling procedure. Strata used in the school sampling included nine U.S. census regions; size of enrollment; racial composition; urban, suburban or rural settings, and public, private or parochial focus. The total number of schools selected for the sample was 1,015 from a sampling of 24,725 schools. Within each school, 36 seniors and 36 sophomores were randomly selected to participate as subjects in the HSB data collection. In those schools with fewer than 36 seniors or 36 sophomores, all eligible students were included in the sample.

In this study, data from 10,416 seniors from rural, suburban and urban areas and matched for socioeconomic variables were analyzed. Rural and urban students in this study are nearly identical in terms of distribution by family annual income; the suburban group is somewhat more affluent. See Table 1.

TABLE 1

FAMILY ANNUAL INCOME

	URBAN	SUBURBAN	RURAL	TOTAL
$6,999 or less	14.4%	7.1%	14.4%	11.1%
$7-11,999	19.5%	12.0%	17.3%	15.5%
$12-15,999	18.9%	15.0%	19.8%	17.4%
$16-19,999	15.6%	15.9%	18.4%	16.5%
$24,999	14.0%	18.6%	13.5%	16.0%
$37,999	10.6%	16.3%	9.2%	12.7%
$38,000 +	6.9%	15.1%	7.4%	10.7%

WHAT ARE ASPIRATIONS?

The term *aspirations* is one which is often used synonymously with goals, ambitions, objectives, purposes, dreams, plans, designs, intentions, desires, longings, wishes, yearnings, cravings or aims. Aspirations are what drive individuals to *do more* and *be more* than they presently are. We may know what we are, but we cannot know for certain what *we can be*. It is this latter condition that necessitates that we be guided and motivated by aspirations.

Philip Pratt is a doctoral student in Counseling and Guidance in the College of Education at UMO and has recently joined the staff of Eastern Maine Vocational Technical Institute.

Some important distinctions need to be made regarding aspirations. First, there is a difference between *educational and career aspirations* on the one hand, and *quality of life aspirations* on the other. Education and career aspirations relate to how much value people assign to formal education and how far they intend to pursue it, *i.e.*, do they seek a high school diploma, a four-year college degree or other post-secondary training, or perhaps a Ph.D. or M.D. degree? Career aspirations pertain to what type of vocation or profession they want to pursue. Quality of life aspirations are those related to such questions as where individuals would prefer to live, what kind of environment they seek for themselves, what kind of community they'd prefer, the kind of family they want, the type of schools they want for their children, *etc*. While these two major categories of aspirations are different, they nonetheless are inextricably interdependent. This study focuses on the variables representative of both educational/career aspirations and quality of life aspirations.

A second distinction should be made between *expressed aspirations* and *manifest aspirations*. Expressed aspirations are self-reported statements of what individuals say they want to do. It is possible for these to be spur of the moment, transient, or the *popular thing to say* reports of personal goals, and as such, do not always present genuine, well-developed aspirations. Manifest aspirations on the other hand are reflected in what individuals actually do with their lives. These readily observable indicators can serve to support or contradict what they say they want to do. For example, the statement, *I really want to do well in high school so that I can go to a good college*, represents an expressed aspiration while that student's actual daily attendance record and overall academic performance in school would serve as manifest aspiration indictors. It should be noted that this study is based upon findings derived from expressed aspirations only.

Aspirations are influenced considerably by the communicated expectations of the significant people who interact with the individual. If those expectations are high and consistent over time, then there is a greater chance that the individual's aspirations will be similarly high. Similarly, low expectations often result in low aspirations. In this study the investigators analyzed selected variables from the **High School and Beyond** data to see what influences students perceive the expectations of their parents, teachers, peers and guidance counselors have had on their aspiration levels.

Rural youth value their jobs more and their academics less than urban and suburban youth.

When asked if a job was more important for them than school, 14.9 percent of the rural youngsters replied *Yes* while only 8.9 percent of the urban and 10.4 percent of the suburban students agreed. (Only 5.9 percent of the total sample reported that they never worked.) Moreover, when asked if their schools should have placed more emphasis on basic academic subjects, 29.2 percent of the urban students strongly agreed while only 22.4 percent of the rural students agreed strongly.

Rural youth place lower value on making lots of money than do urban youth, but value friendship more.

When asked how important in their life having lots of money was, only 30.8 percent of the rural respondents said *very important*, while 38.6 percent of urban youth said it was *very important*. When asked how important good income was in determining the kind of career they would seek, fewer rural students than urban students said it was *very important*. Nearly 78 percent of the rural students consider strong friendships as *very important* compared to 70 percent of the urban youngsters.

Rural and urban students aspire to leadership positions in their communities more often than suburban students.

Fifty-seven percent of rural youngsters report that being a leader in the community is *somewhat* or *very important* compared to the 53 percent of their urban and suburban peers.

A greater percentage of urban students than rural students report that the goal of being able to give their children better opportunities than they had was very important to them.

Nearly 80 percent of the urban youngsters said this was *very important* while only 70 percent of both rural and suburban youth said it was *very important.*

Neither rural nor suburban respondents see themselves correcting social and economic inequities as frequently as urban respondents do.

Only 15 percent of both rural and suburban youngsters view this social responsibility as being *very important* to them while more than 20 percent of the urban youth do see it as being *very important* to them.

Rural youngsters do not aspire to postsecondary educational opportunities as frequently as either urban or suburban respondents do.

When asked how far in school they thought they would get, rural students generally answered with fewer years of study than either urban or suburban students. Table 2 depicts the responses of the three groups. When asked the lowest level of education they would be satisfied with, rural students' responses again depicted satisfaction with lower levels of education than did the urban and suburban students. Table 3 contains the specific responses.

Rural students are not as confident as urban and suburban students in their abilities to complete a college education.

TABLE 2

HOW FAR IN SCHOOL DO YOU THINK YOU'LL GET?

	URBAN	SUBURBAN	RURAL
Less than high school	.7%	.3%	.8%
High school grad only	14.1%	13.7%	22.8%
Less than two years at business or voc. school	5.8%	6.4%	10.2%
Two years or more at business or voc. school	11.9%	10.3%	12.8%
Less than two years college	3.2%	2.8%	2.8%
Two or more years of college with Assoc. Degree	12.3%	12.6%	12.6%
Finish college with Bachelors	26.1%	27.8%	22.6%
Master's or equivalent	13.1%	14.2%	9.0%
Ph.D., or M.D. or equivalent	12.9%	11.8%	6.3%

TABLE 3

WHAT IS THE LOWEST LEVEL OF EDUCATION YOU'D BE SATISFIED WITH?

	URBAN	SUBURBAN	RURAL
Less than high school grad	1.6%	1.1%	1.9%
High school grad	24.1%	25.5%	37.3%
Less than 2 years at vocational school	5.5%	5.7%	6.8%
2 years or more at business or vocational school	10.0%	9.0%	10.7%
Less than 2 years college	6.4%	5.7%	5.2%
2 or more years college	18.4%	18.3%	14.6%
B.S./B.A. degree	22.6%	24.9%	18.1%
Master's degree	7.2%	6.1%	3.2%
Ph.D. degree	4.2%	3.7%	2.1%

When asked if they thought they had the ability to complete college, only 43 percent of the rural respondents said *Yes, definitely* while 50 percent of the urban students and 53 percent of the suburban students answered in the same manner. Greater percentages of rural students responded *Not sure, I doubt it* or *Definitely not*.

Thirty-two percent of rural students see themselves going to work full-time the first year after high school compared to 24 percent urban and 22 percent of the suburban students. More urban and suburban students than rural (43 percent, 44 percent and 32 percent respectively) see themselves going on to a four-year college.

Rural youngsters see themselves completing their fulltime education at a younger age than either the urban or suburban youngsters.

In this study 46.2 percent of rural students report that they already completed or expected to complete their fulltime education at or before the age of 20. Only 30.3 percent of their urban peers report similar expectations.

Are these rural-urban differences related to identifiable family and school factors? The seniors surveyed here clearly report that their families are most influential in their postsecondary planning. See Table 4. There are not significant differences by degree of urbanization. However, in following up this information, we find marked differences between rural and urban families. In response to the question *What do your father and mother think you should do after high school? Clear differences between rural and urban parents emerge. See Table 5.*

TABLE 5

WHAT DO YOUR PARENTS THINK YOU OUGHT TO DO AFTER HIGH SCHOOL?

	FATHER		MOTHER	
	URBAN	RURAL	URBAN	RURAL
College	57.9%	49.2%	72.3%	60.0%
Full-time Job	8.5%	14.1%	9.2%	14.1%
Trade School	6.0%	9.5%	7.7%	11.5%
Military	3.1%	4.1%	2.9%	3.2%
They don't care	2.5%	3.9%	1.6%	3.1%
I don't know	8.8%	9.9%	3.8%	5.7%
Does not apply	13.2%	9.3%	2.4%	2.3%

Rural parents are perceived as much less often supportive of fulltime college (Rural Fathers 49 percent vs Urban Fathers 58 percent, Rural Mothers 60 percent vs Urban Mothers 72 percent) **than their urban counterparts and more supportive of fulltime jobs, trade schools, and the military**. A greater percentage of rural parents also are reported to *Not Care*.

TABLE 4

HOW MUCH HAS EACH OF THE FOLLOWING PERSONS INFLUENCED YOUR PLANS FOR AFTER HIGH SCHOOL?

	MOTHER	FATHER	COUNSELOR	TEACHERS	FRIENDS/ RELATIVES	MILITARY RECRUITER	COLLEGE RECRUITER
Not at all	22.2%	11.1%	44.0%	36.6%	21.4%	83.4%	65.8%
Somewhat	40.7%	40.3%	41.5%	45.3%	50.9%	11.5%	24.4%
A great deal	37.1%	48.6%	14.5%	18.1%	27.7%	5.1%	9.9%

Similarly, students from rural settings report more often than their urban counterparts that their guidance counselors and teachers do not think they ought to go to college. See Table 6. In addition, more rural youngsters than urban report that their teachers and counselors *don't care* what their postsecondary plans are.

TABLE 6

WHAT DO YOUR GUIDANCE COUNSELORS AND TEACHERS THINK YOU OUGHT TO DO AFTER HIGH SCHOOL?

	GUIDANCE COUNSELOR		TEACHER	
	URBAN	RURAL	URBAN	RURAL
College	56.1%	50.1%	56.3%	48.6%
Full-time Job	1.4%	2.0%	2.0%	2.0%
Trade School	4.2%	6.1%	3.9%	4.3%
Military	.8%	.8%	.8%	1.0%
They don't care	5.7%	5.8%	7.5%	8.7%
I don't know	22.7%	26.1%	22.7%	27.7%
Does not apply	9.3%	9.2%	6.8%	7.6%

The students' perceptions of the relative influences of the various *significant others* in their lives appear to bear fruit. The rural students' reported aspirations are consistent with the expectations of their parents and teachers. When asked to respond to the item, *I will be disappointed if I don't graduate from a college*, fewer rural (61 percent) than urban (74 percent) youth responded affirmatively.

TABLE 7

WHICH CATEGORY DESCRIBES THE JOB YOU EXPECT TO HAVE AT AGE 30

	URBAN	RURAL
Clerical	11.3%	11.6%
Craftsman	5.9%	9.3%
Farmer	.5%	3.5%
Housewife	1.2%	3.4%
Laborer	.7%	2.7%
Manager-Admin.	7.6%	5.3%
Military	2.5%	2.6%
Operative	1.9%	3.9%
Professional (Lower)	29.1%	24.2%
Professional (Higher)	15.2%	9.0%
Proprietor-Owner	2.9%	3.3%
Protective services	1.7%	1.7%
Sales	1.8%	1.8%
School teacher	3.5%	4.2%
Service	3.3%	4.1%
Technical	9.7%	8.1%
Not working	1.3%	1.5%

More evidence of differences in the aspiration levels of rural and urban students appears in their responses to a question of what kind of job they expect to hold at age 30. See Table 7. **Rather consistently the rural students depict themselves less often in higher level positions, and more often in lower level, less skilled areas**. Our rural sample reports itself more often as expecting to be in clerical, craftsman, farmer, housewife, laborer, operator, proprietor-owner, and services roles and *not working* than the urban students. The urban students more often suggest they will be managers and administrators or in the various professions (accounting, nursing, engineering, medicine, law, college teaching, *etc.*).

This career expectation data at age 30 is congruent with the educational expectations of these students. In response to the question, *How far in school do you think you'll get?* the rural students expect to obtain less education than their urban peers. See Table 2. Not surprisingly, and consistent with the picture emerging here, our rural subjects report that their mothers also hold lower expectations for the students' ultimate level of education than the mothers of urban students. See Table 8.

TABLE 8

HOW FAR IN SCHOOL DOES YOUR MOTHER WANT YOU TO GO?

	URBAN	RURAL
Less than high school	.6%	.5%
High school graduate	5.3%	10.6%
Less than 2 years vocational or business	3.2%	5.6%
More than 2 years vocational or business	6.9%	10.2%
Less than 2 years of college	1.5%	2.0%
2 or more years of college	7.5%	8.8%
B.A.	28.7%	26.0%
M.S. or equivalent	10.5%	7.8%
Ph.D., M.D., other	18.5%	9.7%
Don't know	17.4%	18.8%

One last area of interest relative to rural-urban differences is the expectation or willingness of our high school students to *leave home. When asked if they were willing to move for a job they wanted, more rural than urban youth not only were willing to move, they prefered to move*. See Table 9. This finding is, of course, consistent with demographic history. Rural Americans have sought out city life in increasing proportions for more than a century.

The data here suggest that the desire to move is somewhat related to family relations. When asked to respond to the question *How important to you is living close to*

parents and relatives? 14 percent of rural youth responded *very important* compared to 17 percent of urban youth, and 33 percent responded *not important* compared to 30 percent of their urban peers.

TABLE 9

ARE YOU WILLING TO MOVE FOR THE JOB YOU WANT?

	URBAN	RURAL
Yes, prefer	28.3%	40.0%
Yes, no difference	30.3%	31.6%
Yes, but prefer to stay	33.2%	23.8%
No, won't move	8.2%	4.7%

In summary, an analysis of these national data suggests that the phenomena we have observed in Maine are not unique to Maine. We have a population of young people in Maine whose aspiration patterns reflect those of rural youth throughout the United States. In terms of postsecondary educational aspirations, America's rural high school seniors aspire less often than their urban and suburban peers to continuing their education. And when they do aspire to postsecondary education, their expectations for the level of educational attainment are lower, their expressed levels of self-confidence in completing degree requirements are not as high, and they don't expect or pursue further education for as long as urban youngsters.

With respect to career aspirations, rural students, more often than their urban counterparts, expect to enter the work force immediately following high school. Moreover, their aspirations for specific careers or professions are generally at lower levels than their urban peers.

In terms of the impact that the expectations of parents, teachers and counselors have on aspiration levels of young people, this study substantiates that a relationship exists between the communicated expectations of these *significant others* and the resulting expressed aspirations of the youngsters. The expectations rural adults hold for their youth are reflected clearly in the aspiration statements of rural high school seniors. Rural parents, teachers and counselors evidently do not hold career and educational aspirations for youth that are as high as those held by urban and suburban adults, and their students' goals are not as high as those of urban and suburban students.

With respect to *quality of life* aspirations, rural youth value *making lots of money* less than urban youth, and they value friendships more. They aspire to leadership positions in their communities more often than suburban youth, but they do not see themselves correcting social and economic inequities as often. And finally, they seek opportunities to move away from their home communities after high school more often than urban youngsters.

Our purpose in undertaking this study was to determine if the problem of low aspirations among youth was unique to Maine. The results of our analysis have convinced us that low aspirations among rural youth is a problem which exists nationally. This provides little comfort, however. We are still obligated to respond to the problem as we see it in Maine. The first steps have been taken: more than 70 communities and their schools have recognized that the problem doesn't ameliorate itself, that it demands conscious planning and concerted efforts by parents, school personnel, concerned citizens and municipal officials, and that *we can do something* about raising the aspiration levels of Maine youngsters. The goals, dreams, and ambitions of our young people should not be a function of whether they live in a rural, urban or suburban environment. But the evidence is clear: those who live in rural America are evidencing generally lower aspirations. Rural states have a special responsibility to their young which must be recognized and fulfilled. Certainly, Maine's young people are deserving of any measures which can be undertaken to promote higher aspirations among its students.

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ELSEWHERE IN EDUCATION

a research sampler

PHYSICAL EDUCATION AND HANDICAPPED CHILDREN

Public Law 94-142 mandates the education of handicapped children in the least restrictive environment. With the advent of this landmark legislation in 1975, handicapped children could routinely participate in their schools' regular physical education program. Unfortunately, research describing the psychomotor characteristics of handicapped children has not kept pace with the needs of practitioners in the field. The tradition of physical education research has been to describe children's abilities in terms of measurable end products: how fast (running and agility); how far (throwing); how high (jumping); and how many (push-ups, pull-ups, sit-ups, and squat thrusts). Although such information is useful in classifying children according to their physical and motor fitness abilities, additional types of information are needed if physical education programs which appropriately include handicapped children are to be developed. There has been a need for research that allows practitioners to evaluate children in terms of their skill development rather than solely on the results of their performance.

Dr. Steven Butterfield, Assistant Professor of Physical Education, has been conducting research designed to summarize and describe the gross motor development of deaf children as well as to examine factors thought to affect their development. He has studied 132 hearing-impaired children between the ages of 3 and 14. The children were enrolled in schools for the deaf in Rhode Island, Vermont, New York, and Ohio and represented various socioeconomic and cultural backgrounds. Each child was individually assessed on 11 fundamental motor skills (walking, running, stair and ladder climbing, jumping, hopping, skipping, catching, kicking, throwing and striking) and two balance skills (static and dynamic). Information was also obtained regarding cause of deafness and degree of hearing loss for each child.

Performance, as expected, showed steady improvement with age for both gross motor and balance skills. Of greater interest though was the similarity in motor performance of males and females. Previous investigations of hearing children have found significant differences favoring males in throwing and kicking and females in hopping and skipping. His explanation is that it is possible that deaf children placed in special schools are removed from cultural expectations which favor one sex over the other in certain skills. The skills presenting the most difficulty to deaf children were kicking, jumping, catching and hopping. Three of these skills (jumping, kicking, and hopping) place a premium on efficient balance. It is likely that balance deficiencies, which are more prevalent among the deaf, contributed to the children's poorer performances in these skills. Those subjects who did perform well on the balance tasks tended to be more advanced in overall gross motor development.

The results of Dr. Butterfield's research have contributed to increased awareness of the specific needs of handicapped children in physical education programs.

Stephen A. Butterfield is currently Assistant Professor of Education in the College of Education at UMO. He is involved in helping area schools set up programs for handicapped children in physical education. He has more than 10 years experience teaching handicapped children in Vermont and Ohio. He was educated at Springfield College and Ohio State.

SCHOOL CLIMATE AND TEACHER EFFICACY

A recent thrust in educational research has been in the area of teachers' sense of efficacy: the beliefs teachers hold about their ability, both as individuals and a profession, to influence student achievement. While the studies to date have provided encouraging results about teacher efficacy and its correlates, existing measures of teacher efficacy have both theoretical and statistical shortcomings.

Dr. Theodore Coladarci, Assistant Professor of Educational Psychology in the College of Education, is currently conducting a study of teacher efficacy designed to overcome the problems of earlier research in this area and explore the relationship between teacher efficacy and an established correlate of school effectiveness: *school climate*. Professor Coladarci asked a representative sample of 364 Maine elementary school teachers to respond to a series of statements concerning their efficacy beliefs and their perceptions of select aspects of school climate: the principal's decision-making style, the sense of collegiality and rapport in the school, and the instructional leadership provided by the principal.

There are three general objectives to the study. Factor analyses of the efficacy data, coupled with the factor analytic work of earlier researches, will contribute to the present understanding of the teacher efficacy construct. Secondly, the results will provide knowledge regarding

the relationship between teacher efficacy and teachers' perceptions of important dimensions of school climate, a relationship that remains unexplored. Consequently, the results associated with these two objectives should enhance existing models of teacher and school variables and their effects on student outcomes. Finally and more practically, descriptive information regarding teachers' efficacy beliefs and perceptions of school climate will provide Maine educators with an important empirical basis for analyzing various facets of school life, their interrelationships, and the corresponding implications for school improvement.

Theodore Coladarci is Assistant Professor in the College of Education where he is responsible for courses in educational psychology and research methodology. Dr. Coladarci obtained his Ph.D. in educational psychology from Stanford University. His research interests are in the area of research on teaching and teacher education.

THE PRINCIPAL PRINCIPLE

The role of the school principal as an educational leader has drawn attention in recent years as researchers and practitioners seek anew to understand what makes a school effective. The role has changed considerably as schools' functions have diversified, and expectations for academic performance have risen.

Dr. Gordon Donaldson, Assistant Professor of Educational Administration, is currently conducting research on the Maine school principalship. One effort takes a unique approach to the analysis of the principal's effects and style. Dr. Donaldson is conducting a case study which compares one principal's perceptions of his own role, style, and rationale for decisions to his staff's perceptions. Based on the assumption that the principal's effects are interactive, Dr. Donaldson's study explores the degree of congruence between principal and faculty perceptions.

The results to date suggest that teachers employ a considerably different system for assessing leadership than does the principal. While the principal views his success in academic/instrumental terms, the teachers primarily regard him from an interpersonal/collegial framework, tending to rate him according to his *handling* of them rather than on the basis of his goals and accomplishments.

Another of Dr. Donaldson's projects explores the existence of a career ladder for Maine principals leading from small, rural schools to larger, urban schools. The study, based on Department of Educational and Cultural Services' statistics from the past twelve years, will document the extent to which this happens, as well as provide a picture of principals who remain in smaller schools.

Gordon A. Donaldson, Jr., is currently Assistant Professor of Education in the College of Education at UMO. He is involved in assisting schools assess and improve school climate and in efforts to train principals in the support and development of good teaching. He trained at Harvard and has taught and served as principal in Ellsworth and in North Haven.

ASSESSING LEADERSHIP

There is a growing body of research suggesting that the school principal is a major agent in creating conditions for school effectiveness. One approach to enhancing school performance is to optimize conditions for principal effectiveness. Central to this approach is a clear understanding of how principals feel about their role. **Dr. Ron Sparkes**, the first graduate of UMO's doctoral program in educational administration and Superintendent of Schools for the Labrador East Integrated School Board, has recently completed a study which indicates that the size of both the community and the school in which the principal serves are major determinants of job satisfaction. His study surveyed Newfoundland's and Labrador's 618 principals. Respondents completed the *Minnesota Satisfaction Questionnaire*, a measure of job satisfaction that provides a measure of general satisfaction as well as scores for 20 reinforcers contributing to satisfaction.

He found that principals of small schools in small communities reported significantly lower levels of satisfaction than their large-school peers on general satisfaction and 12 of the reinforcement areas.

When compared to their large-school counterparts, principals of smaller schools in small communities are clearly disadvantaged. Sparkes found that these principals generally carry heavy teaching loads that place constraints on the amount and quality of time available for leadership activities. They often must function without the benefit of services and resources available to schools in larger areas. Sparkes goes on to caution against using *big school solutions* for *small school problems*, a course of action which he suggests has been too often descriptive of educational practice. His study is an important contribution to our understanding of the very nature and special needs of the small school in the small community. Confronting the realities of working in such schools is a necessary first step in improving the quality of life for principals who serve as educational leaders in small schools everywhere.

Ronald L. Sparkes is a native of and was educated in New Brunswick, Canada. He has taught in the schools, has been a principal, and is currently Superintendent of Schools in Goose Bay, Labrador. He completed his doctoral studies in educational administration at the University of Maine at Orono in August 1985.

The United States is a land of plenty, but food is still a problem for many whether it be too little, too much, or not the right kind. Malnutrition exists in this country, but its presence is more subtle than that found in poorer nations. Nutrition surveillance data have identified short stature and overweight as potential public health problems among Maine children of low socioeconomic status. Concern at the State level has brought UMO nutrition scientists into the field to assess the seriousness of the situation and to make recommendations for statewide intervention.

MALNUTRITION IN MAINE

by Richard A. Cook

To most individuals in the United States, the topic of malnutrition will most likely ellicit images of famine in Ethiopia or perhaps emaciated people begging for food among the 200 million undernourished in India. Most surely, anyone travelling in developing nations, outside of tourist resorts, sees clinical evidence of nutrient deficiency diseases and starvation among those in the street, particularly young children. It is unsettling to know that one-half billion people worldwide are experiencing extreme poverty, and of these, 450 million have severe malnutrition from lack of food while 15-20 million die annually from hunger-related causes.

What most Americans don't realize is that there are those in this country who are included among the two-thirds of the world's population estimated not to have enough to eat or the right kinds of foods to eat. Nutrient deficiences among our population are usually non-evident since they are in a subclinical stage and are not visually observable. Even routine checks by a family physician are not adequate to identify problems. However, functional aspects of our lives such as growth velocity, fecundity, pregnancy outcome, lactation performance, cognitive performance, work capacity, activity, social behavioral performance, morbidity and mortality can all be affected by marginal malnutrition. What most of us *are* aware of as a malnutrition problem in this country are the results of overnutrition, manifested as overweight or obesity among greater than 40 percent of the population as a whole.

Human nutrition researchers in the School of Human Development at UMO have been concerned with malnutrition problems among Maine citizens for many years. In the 1930s, 40s and early 50s work was concentrated in the areas of vitamin A, vitamin C and iron nutrition. Targeted groups included children and teenagers. Emphasis shifted in the late 50s and 60s to basic nutrition studies involving fats, including cholesterol, and proteins.

In the 1970's teams went back into the field for nutritional status assessments of several population groups spanning the life cycle from the very young preschooler to elderly nursing home patients. Physical measurements, nutrient intakes, and blood and urine biochemistries were all utilized for identification of nutritionally *at risk* groups. A need was established for an ongoing nutritional status monitoring system with supporting in-depth nutrition research studies to facilitate preventive health measures.

At the same time these studies were being carried out, a national nutrition surveillance system *was* being developed through the Center for Disease Control (CDC) in Atlanta. More than 20 states contributed information from health department clinics; Medicaid; Early Periodic Screening, Diagnosis and Treatment programs; Women, Infants, and Children (WIC) programs, and other health care facilities. Surveillance was accomplished for growth deficit, iron deficiency anemia, and obesity among individuals from birth through 18 years of age.

WIC programs (Special Supplemental Food Program for Women, Infants and Children) formed the core of the national nutrition surveillance system. WIC is a fairly new federal food assistance program, becoming operative in 1973. Under administration from the Department of Agriculture, local agencies are funded to distribute coupons redeemable for specific nutrient-dense foods, or

Richard A. Cook is Associate Professor of Nutrition and Coordinator for the Division of Human Nutrition and Foods in the School of Human Development. He earned a Ph.D. in Nutrition at the University of Maine; has worked extensively in community nutrition research projects across the state, and has participated in five Northeastern Regional Research projects. His interest extends to international nutrition through study at M.I.T., Penn State University, and the Philippines. His present research work is focused on the utility of microcomputer-adapted food data bases.

the foods themselves, to populations at risk for nutrition related health problems, *i.e.*, pregnant and lactating women and children from birth to 5 years of age. Maine WIC programs are coordinated through the Maine State Department of Human Services, Bureau of Health, and a State WIC Nutritionist. Maine WIC centers joined the CDC surveillance system in the fall of 1979.

Analysis of the WIC surveillance data during the 1980's showed overweight and short stature as serious health problems. Underweight did not appear to be a level of special concern.

Prevalence of overweight was twice the level of concern expected nationally. When compared to 1980 data from all states involved in the CDC nutrition surveillance system and a 1983 nutrition survey of low income preschool children in Massachusetts, the Maine WIC population had a higher incidence of overweight.

Prevalence of short stature at a level suggesting a need for intervention was 200 percent higher among the Maine children than was expected nationally. It was also higher than 1980 CDC data and the 1983 Massachusetts nutrition survey data. Incidence of this growth retardation problem ranged from a high of 23 percent to a low of 6 percent in two counties with high Franco-American populations: Androscoggin County and Aroostook County, respectively, as shown in Table 1.

Although surveillance techniques identified problems, they were not designed to determine causes. Of particular concern was the high prevalence of short stature, as it might be an indicator of long-term illness or undernutrition among the infants and children screened. By not identifying and following up individuals for causes and treatment, possible depressed psychomotor development or limited mental capacity could result, although normal WIC practices of food assistance would be expected to have a positive impact. Overall, some type of intervention strategy was warranted.

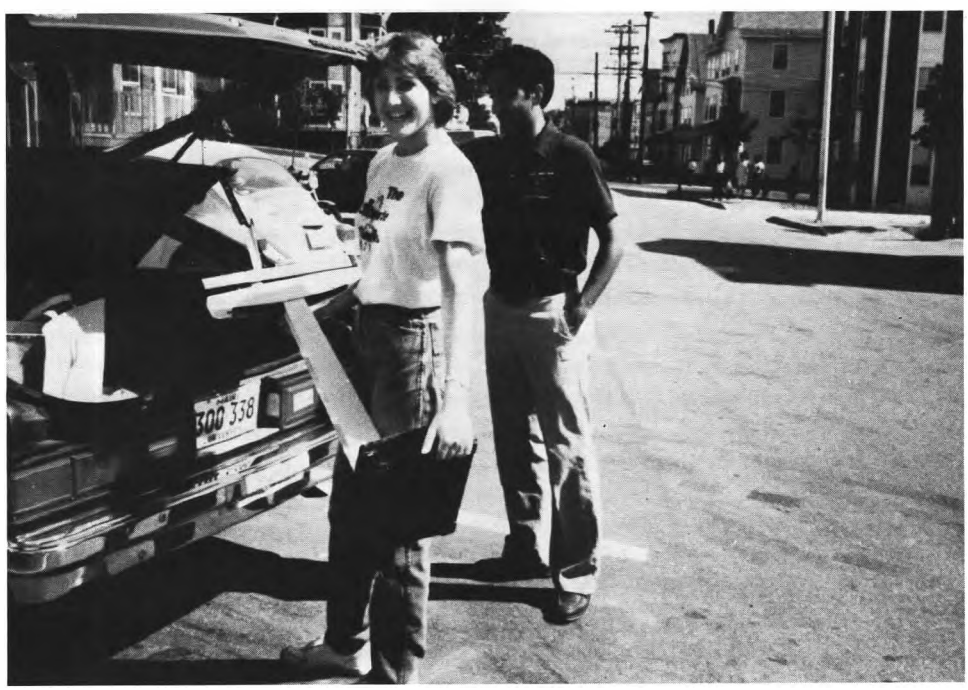
MAINE WIC PROGRAM - NUTRITION SURVEILLANCE DATA
1982 ANNUAL SUMMARY
FOLLOW-UP VISITS ONLY

Table 1. Rank order of counties by prevalence of short stature.

Rank	County	Ht/Age < 5 percentile (%)
1	Androscoggin	22.9
2	Waldo	16.1
3	Franklin	15.8
4	Sagadahoc	15.4
5	Oxford	13.3
6	Lincoln	13.1
7	Hancock	12.9
8	Cumberland	12.7
9	York	12.5
10	Kennebec	11.8
11	Somerset	10.6
12	Knox	8.6
13	Penobscot	8.1
14	Washington	7.0
15	Piscataquis	6.7
16	Aroostook	6.3
	CDC (1980)*	8.5
	Mass. Survey (1983)	9.8
	U.S. Reference Population	5.0

*White population only

Source: M. Ibrahim Parvanta, Nutritionist, Department of Human Services, Division of Maternal and Child Health, State House Station 11, Augusta, ME 04333



Paula Quatromoni and Ibrahim (Abe) Parvanta, nutritionist with the Maine State Department of Human Services, Division of Maternal and Child Health, carried the necessary equipment from car trunk to city apartments so that interviews and measurements could be done most conveniently for participants in the study. Parvanta earned his baccalaureate degrees at UMO in zoology and in foods and nutrition. Human nutrition was the focus of his masters' degree at UMO.

To determine what role malnutrition might play in the public health problem identified, a pilot study of growth deficit and overweight among the WIC population (age 1-5) in Androscoggin County (principally Lewiston area) was approved and supported by the State Department of Human Services, Division of Maternal and Child Health, for the year 1985. The sample population was drawn from WIC program children in the county who expressed a risk factor of less than the fifth percentile nationally for height-for-age, weight-for-height, or weight-for-age. Those also included for study were the very overweight and the anemic (very low hemoglobin level). Out of more than 100 possible subjects for study, 41 gave permission to be contacted. A control population was also generated from the remainder of the Androscoggin WIC group.

More than 100 names were randomly selected and of that number, 20 families agreed to participate. In an attempt to find causes of the risk problems identified, it was necessary to reassess anthropometric status, and generate new data on growth history, general health status, dietary intake and family history.

One field worker carried out all interviews and gained firsthand knowledge of life among the more unfortunate young mothers and their children in our society. (Unexpected circumstances were tolerated such as being asked to enter a third story apartment through a window instead of a door, while trying to carry a physician's weighing scale.) Field studies were completed during the months of July and August.

Data are now being analyzed for rapid turnaround to State officials. With attention to reasons for the existence of such high rates of short stature and overweight, special emphasis will be placed on nutritional concerns. Methodological problems will be elucidated and opinions made as to whether the pilot study should be expanded to the collection of data on a statewide basis.

As this study is being carried out, the UMO nutrition scientists involved are simultaneously carrying out basic research for more appropriate methods for rapid screening of diet intake problems of *at risk* populations, particularly WIC children. This work is invaluable to ongoing studies of the type described here, and will allow quantitative nutrition monitoring and surveillance, even with the use of paraprofessionals, by appropriate utilization of microcomputers.

Paula Quatromoni, research assistant at UMO, worked as field supervisor during the summer of 1985 gathering new data and reassessing the anthropometric status of children in the nutritionally *at risk* group. An M.S. degree candidate, Quatromoni is in an accelerated graduate program at UMO where she participated in the Honors Program as an undergraduate. She presented her honors thesis, one of only eight accepted juried papers, at the 76th Annual Meeting and Exposition of the American Home Economics Association held last June in Philadelphia.

The linking of a University-based research team with state agency personnel concerned with public health nutrition is an effective intervention strategy to reach populations most in need, who themselves feel helpless in a high-paced, affluent society. Making a positive impact on the nutrition of the young among the nation's poor must surely benefit society as a whole in the future.

The aging process is an appropriate and cyclic mechanism rife with visible and measurable characteristics, but illnesses can produce signs and symptoms which may be confused with the more common traits of aging. To differentiate between common traits of the aging process and subtle disease processes which cause changes over time is a vital, yet infrequent, subject of inquiry. Researchers at UMO have been addressing the relationships between hypertension and the aging for several years. This is their story. Thanks are due Mike Robbins for his patience with the editorial process.

HYPERTENSION
aging and intellect

by Merrill F. Elias and Michael Robbins

The study of disease is an important aspect of research on aging because a portion of the age-associated decline in intellectual functioning is related to *disease processes*, as distinct from biological changes characteristic of *normal aging*. Often the relationship between severely debilitating diseases and intellectual performance is so obvious that there is little doubt that accelerated intellectual decline has been caused by illness. When diagnosed and treated properly, essential hypertension is not a debilitating disease and does not result in obvious alterations in intellectual functioning or other aspects of behavior. Therefore, it is a good model for insidious disease processes which effect changes in cerebrovascular physiology over a long period of time, and thus have the potential for influencing intellectual performance in a negative manner. In this context, methodological lessons learned as one studies hypertension and behavior may be applied to the study of other subtle disease processes resulting in progressive change over time. But aside from methodology, the study of the effects of hypertension on cognitive functioning has a practical value. The prevalence of hypertension in the United States was estimated at approximately 15 percent in 1960 but was found to rise with advancing age, from 1 percent to 2 percent in those below the age of 25 to 40 percent in those over the age of 65. (Gavras & Gavras 1983)

Our studies have addressed four related questions:

- Are there differences in performance between hypertensives and normotensives and are they replicable;
- If so, are the differences of practical importance;
- Can one actually predict hypertensive status from tests sensitive to cognitive dysfunction;
- Are the negative effects of hypertension on performance, if any, greater for older than for younger persons?

Merrill F. Elias is Professor of Psychology at UMO. He earned his Ph.D. in Experimental Psychology at Purdue University and trained as a postdoctoral fellow in gerontology and neuropsychology at Duke University. His research in behavioral medicine has centered around the effects of cardiovascular diseases and aging on mental performance. He has served as a consultant to the National Institute on Aging and is Editor-in-Chief of Experimental Aging Research *and Executive Editor of* Gerodontology.

Michael A. Robbins is Research Associate in Psychology at UMO. He earned his Ph.D. at UMO and has worked with Elias in the development and execution of several projects funded by the National Institute of Aging. His research interests involve the role of personality and social characteristics in health-related behavior.

DEFINITIONS

Basically, uncomplicated essential hypertension is a symptom. See Table 1. One infers the presence of disease, even though the defective physiological mechanisms are not clinically detectable within limits defined by currently available medical technology. Thus it is a symptom of any one of a number of diseases of unknown etiology. By definition, no causal process is immediately apparent, yet it places the individual at risk for complications such as stroke, heart disease and other debilitating and life-threatening medical complications. In fact, hypertensives who develop complications are referred to as complicated, essential hypertensives. Complicated and uncomplicated essential hypertension can be distinguished from secondary hypertension where elevated blood pressure values are a result of a clearly identifiable disease such as an endocrine disorder, a tumor, kidney failure, *etc.* While it is

important to distinguish between different types of hypertensives, these subdivisions are crude, and individuals within them cannot be considered a homogeneous group with respect to a specific etiology or prognosis. It comes as no surprise that variability in the performance and the personality characteristics of essential hypertensives may be as large as the variability observed for older groups of normotensive patients, depressed patients or any other group formed for research purposes. This point is illustrated by examining a plot of performance values versus blood pressure values for any number of published studies. In this case, a study by a doctoral student provides a good illustration.

In a well-controlled study an adequate number of subjects (n = 203), Light (1978) reported a Pearson correlation coefficient of .34 between mean arterial blood pressure (diastolic blood pressure + 1/3 pulse pressure) and serial-discrimination reaction time. Inspection of the plot for this correlation is revealing in terms of individual differences. See Figure 1. Part of the variation in performance is related to the fact that the exact values of systolic and diastolic blood pressure used for separating the hypertensive persons from normotensive persons are arbitrary with respect to a given individual. They are based on group statistics and reflect a point beyond which an increased incidence of morbidity and mortality occurs with further increases in blood pressure. In our studies, consistent, untreated blood pressure values above 140/90 mmHg are considered to be hypertensive values. Studies using very elderly persons have used cutting scores of 150/95 or even 165/95 mmHg.

HYPER- AND NORMOTENSIVES

Reviews of the literature leave little doubt that hypertensives and normotensives differ with respect to mean levels of performance (Elias & Streeten, 1980). However, in some studies, too few subjects were used. In others, differences reflect a failure to control for mean levels of education and for occupational status, but most studies have been done adequately. In our published studies (Pentz *et al.*, 1979; Schultz *et al.*, 1979, in press; Wood *et al.*, 1978, 1979 a & b) and for our studies currently underway, we have had the opportunity, thanks to our very profitable collaboration with Dr. D.H.P. Streeten, Professor of Medicine, Upstate Medical Center Syracuse, New York, and Dr. Norman Schultz, Associate Professor of Psychology, Clemson University, to test hypertensive and normotensive subjects who have been very carefully examined in the context of a comprehensive medical diagnostic study, including endocrine studies. Details on the nature of the examination have been published (Streeten, *et al.*, 1975) along with a list of symptoms and medical history variables resulting in exclusion from our study (Schultz, *et al.*, 1979). Briefly, essential hypertensives were free from hypertension-associated complications and neurological and psychiatric disorders. Normotensives were healthy and free from psychiatric disorder. Our sample was, on the average, quite highly educated (Mean = 14.95 years, SD = 2.21), and consisted mainly of professionals, executives, college professors, semi-skilled laborers, and some blue-collar workers. For comparisons between hypertensive and normotensive groups, education, occupation, and age variables were matched. For regression type analyses, correlations between blood pressure values and performance scores were adjusted for these subject variables.

Figure 2 summarizes the results of one of our very early studies (Schultz, *et al.*, 1979) with the most widely used standardized measure of general intelligence, the Wechsler Adult Intelligence Scale (WAIS): mean performance values were lower for the hypertensives than for the normotensives on the verbal and performance scales of the WAIS (as well as on eight of the subtests making up the scales). In another study, hypertensives (n = 25) and normotensives (n = 37) differed with respect to the number of errors on the Halstead-Reitan battery test most sensitive to brain damage (Pentz *et al.*, 1979), the categories test, a test of ability to shift discrimination learning sets. See Figure 3. In a recent, yet unpublished, study from our laboratory, hypertensives (n = 45) and normotensives (n = 44) differed on three (Categories, TPT Memory, TPT Localization) out of seven neuropsychological tests administered, with higher mean performance values on each test for the normotensives. Table 2 describes these seven tests. These findings are *not* specific to our highly educated and carefully medically examined sample. In a recent review of the literature we (Elias & Streeten, 1980) cited more than 18 studies reporting mean performance differences between essential hypertensives and normotensives, as well as citing our own series of animal studies. (Elias, Elias & Schlager, 1975; Elias *et al.*, Elias & Pentz, 1977; Elias & Schlager, 1974; Elias *et al.*, 1975.) In these studied, mice genetically selected for high blood pressure differed in discrimination reversal learning, activity level, endocrine response to stress, and aggression from those genetically selected for low blood pressure. (Mice can be studied over a lifetime and need not be placed on antihypertensive medication.) While mean levels of performance between hypertensives and normotensives differed in each of these studies, variability among subjects was large, with overlap between the distributions of performance scores for hypertensives and normotensives.

This individual variability is illustrated in figure 4, which plots diastolic blood pressure and category scores, individual by individual.

VARIABLES

Since antihypertensive medications affect our test performance, as well as medical diagnostic tests, subjects in our studies are removed from medications several weeks before testing. Since being removed from one's medication might affect performance, patients with and without a history of medication were compared (Schultz *et al.*, 1979). They did not differ with respect to any psychometric measurements. Further, even though high plasma renin activity levels (PRA) are believed by some investigators (*e.g.*, Brunner, *et al.*, 1972) to represent a more severe form of hypertension with a poorer prognosis, we found no performance differences when we compared subjects in high, medium and low renin groups. We may have had too few subjects in the low and high renin groups for maximum statistical power, and thus we have continued to collect data during the last eight years with the objective of performing new analyses of PRA groups with substantially larger numbers of subjects. Moreover, we hope eventually to achieve large enough samples so we will be able to analyze the effects of the type and dosage level of antihypertensive medication on the test performance for a subgroup of subjects who remain medicated during testing.

As in most studies, age was associated with poorer performance scores and higher blood pressure values. See Table 3. Further, as one may infer from the differences between blood pressure groups, various measures of blood pressure and performance were correlated. See Table 4. To assess the contribution of blood pressure to the age effect, we statistically adjusted age for blood pressure values via partial correlational techniques. The result was essentially the same no matter what blood pressure measure we used. As may be seen in Table 3, the correlation between age and performance is reduced when the blood pressure-age and blood pressure-performance correlations are adjusted statistically but age still correlates significantly with most performance measures. Findings such as these have led researchers on aging to argue that some of the decline associated with age is related to disease processes, not natural aging processes *per se*. A better test of this hypothesis is provided by longitudinal studies which we describe later.

PRACTICAL IMPLICATIONS

Given the types of tests we used, we cannot speak directly to performance differences between hypertensives and normotensives with respect to various occupations. But examination of absolute levels of performance for our laboratory and psychometric measures of performance indicates that differences between mean values, though statistically significant, are not significant in a practical sense. For example, mean performance scores for our highly educated hypertensives on WAIS were considerably above the average score for the U.S. normative sample (1.5 standard deviations) used to standardize the WAIS. See Figure 2. Poorly educated hypertensive subjects perform at average or slightly below average levels, but this is true of poorly educated normotensives. Given the large individual differences in performance within hypertensive and normotensive groups, it is clear that the prediction of individual performance scores from blood pressure values, and vice versa, is relatively poor.

This point may be illustrated by comparing age, education, anxiety, depression, and mean arterial blood pressure values as predictors of one of the most sensitive tests of cognitive function on the Halstead Reitan battery: the categories test. The square of the correlation coefficient, converted to a percentage, is typically used by psychometricians as an indication of how well one variable predicts another. These values are shown in Table 5. It is clear that

- **age and education are as good or better predictors of performance than blood pressure**
- **blood pressure does not predict much of the variance in performance (or vice versa)**
- **if we statistically adjust the blood pressure values for both age and education, the correlation coefficients are reduced**
- **if we add to this adjustment for measures of anxiety and depression, they are reduced further**
- **using all of these variables to predict performance results in a better *multiple prediction* than the use of any one alone.**

This demonstrates the obvious fact that performance is affected by many factors. Given that many factors predict an individual's performance and that blood pressure values are not among the best predictors, one might question job discrimination on the basis of blood pressure for essential hypertension. Such discrimination may be justified with respect to critical, safety-related skills such as those needed by an aircraft pilot. Even then the prediction values between age and complex discrimination reaction time are higher than those between mean arterial blood pressure and complex discrimination reaction time.

CAUSAL MODELS

Poor prediction notwithstanding, modest correlations between blood pressure and performance and statistically significant mean performance differences do exist between hypertensives and normotensives. Investigators have not been reluctant to offer explanations. Our reviews of the literature (*e.g.*, Elias & Streeten, 1980; Elias, Robbins & Schultz, in press) indicate that these *speculative explanations* fall in three general, but related, categories:

- *subclinical* or yet undetected cerebral vascular lesions or pathology
- hypoxia resulting from impaired cerebral blood flow
- impairments in autoregulation.

The impaired autoregulation hypothesis has become a necessary adjunct to explanations one and two because under normal autoregulatory circumstances, blood flow to the brain remains within an acceptable homeostatic range, regardless of perfusion pressure (Streeten, 1980; Gavras & Gavras, 1983), and even when perfusion pressure falls below the autoregulatory capability of the brain, there is compensation in terms of oxygen extraction from the blood (Gavras & Gavras, 1983). Thus the notion that *autoregulatory processes* are impaired is necessary to support the cerebral hypoxia hypothesis.

An example of this *organic model* in an explanatory context was provided by a paper which ultimately served as a strong stimulus to our longitudinal work. Wilkie and Eisdorfer (1971) found that a *younger-elderly* (60-69 years) group of patients with borderline hypertension actually showed a significant improvement on the WAIS over a ten-year period. Normotensives exhibited no change. In contrast an *older-elderly* group of borderline hypertensives, (70-79 years), exhibited a significant decline. In other words, blood pressure interacted with age; a performance-facilitating effect was observed for the younger group of elders and a negative effect was observed for the older-elders group. Wilkie and Eisdorfer explained the improvement in the younger hypertensives as a facilitation of cerebral blood flow, hence cerebrovascular oxygenation, induced by elevated blood pressure. The decline in performance for the older groups was explained in terms of the development of cerebrovascular lesions that had progressed to the point where compensation in terms of increased peripheral blood pressure, presumably influencing cerebral pressure, could no longer act in a compensatory manner. Thus, both improvement in performance and decline were explained via the same mechanisms.

The hypothesis was not frivolous. The likelihood of arteriosclerosis and atherosclerosis for the elderly sample was high. Obrist (1964) had presented data indicating that autoregulation might well be impaired in older persons suffering from cerebral vascular lesions; indeed hypertension itself may involve impaired autoregulation (Gavras & Gavras, 1983), particularly for elderly persons taking antihypertensive medication. However, as the investigators themselves pointed out, there was no direct evidence, *e.g.*, cerebral blood flow data, gathered in parallel with the behavioral study, to support the conclusion that physiological mechanisms were *causal* with respect to impaired performance. Other investigators have found these *organic* explanations compelling (*e.g.*, Light, 1978; 1980), but, as yet, a study providing direct evidence in support of these hypotheses has not come to our attention. Moreover, alternative explanations have not been fully explored.

ALTERNATIVE MODELS

Much of the evidence that has encouraged hypotheses explaining how hypertension influences cognitive function is based on the fact that mean performance scores are better for normotensives. We have become increasingly disenchanted with the notion that normotensives are really good control groups against which to compare hypertensives. Under the very best conditions it is almost impossible to detect, or control, the subject-self selection factors, or motivation variables, that lead to volunteering. In many studies, hypertensives are *pseudocaptive audiences*. They are not forced to participate, but they are already in clinic and need only to spend a few more hours, or are pleased to fill in time while waiting at the diagnostic clinic or in the hospital.

There is also evidence that once hypertensives are diagnosed as hypertensive, some become anxious about their jobs, their futures and their performances in general (Haynes *et al.*, 1978). This may be especially true of highly educated hypertensives who, upon learning they are hypertensive, tend to read about the disease, but often fail to discriminate between descriptions of the disease in advanced, untreated stages as compared to essential-hypertensive-disease that has been aggressively treated. It is well-known that anxiety over one's performance serves as a detriment to optimal performance. Further, our data (Wood *et al.*, 1979), and the data of others (Baer *et al.*, 1979) indicated that hypertensives exhibit higher levels of anxiety and depression than do normotensive subjects. We don't know if the anxiety plays a role in the development of the hypertension or whether hypertensives are more anxious and/or depressed because they have been diagnosed as hypertensives and warned of the consequences if not treated properly. Regardless of why hypertensives tend to exhibit higher mean anxiety and depression scores, these variables have potential effects on their performances that have not been explored adequately.

In this respect, reinspection of our data provided some hypotheses about anxiety and stress which have stimulated new work involving the Spielberger state anxiety scale and measurement of neuroendocrine response to stress.

NEW STUDIES

Examination of the anxiety and depression measures, collected in parallel with the general intelligence and neuropsychological performance measures, indicated that

- a larger proportion of subjects scoring in the upper quartile in state and trait anxiety were found in the hypertensive groups (Elias, *et al.*, in press)
- mean state anxiety (presumably an anxiety response to the immediate situation) is higher for the hypertensives (Wood *et al.*, 1979)
- subjects exhibiting higher levels of state anxiety exhibited mean performance levels significantly lower than those who showed lower levels of anxiety (Elias *et al.*, in press).

Further, the correlations between performance and blood pressure values were reduced when they were adjusted statistically for anxiety scores. While these data do not indicate that state anxiety (situational anxiety) causes lower mean values of performance for hypertensives, they did stimulate a *model* which is currently being tested. Essentially, we hypothesize that not all, but some, hypertensives respond to the testing situation with heightened state anxiety. This elevated anxiety state, a stress response, in turn affects performance negatively. Since some, but not all, hypertensives display this phenomenon, mean performance levels for hypertensive groups are decreased, but individual differences in performance are observed.

One difficulty in examining a *stress response* to a cognitive test is that paper and pencil tests of emotional responses such as anxiety are considerably less reliable than endocrine measures. A second problem is that paper and pencil tests cannot be administered concurrently with measures of intellectual performance and thus might be insensitive to short-lived stress response.

As a solution to this problem, we have begun a study with Drs. Streeten and Shultz at the SUNY Medical Center in which plasma epinephrine and norepinephrine responses to a stressful testing situation are being recorded for hypertensive and normotensive subjects. The question is whether some hypertensives (neurogenic hypertensives) will exhibit abnormally high plasma levels of these *stress-sensitive hormones* in response to testing and whether these plasma levels will be related to performance scores.

BRAIN DAMAGE HYPOTHESIS

We employ neuropsychological tests in our studies because they measure specific domains of intellectual and perceptual motor functioning that are not normally measured by more general tests of intellect. While they have proven reliable with regard to discriminations between brain damaged and normal persons, it is widely recognized that lower levels of performance on such tests may reflect factors other than the anatomical or biochemical integrity of the cerebral cortex (Parsons & Prigatano, 1978). Yet, using neuropsychological tests results, a number of investigators have reached the conclusion that hypertensives suffer from *cognitive dysfunction*. Perhaps these studies have been influenced by the emphasis in the literature on physiological mechanisms as *causal* with respect to lower average performance levels observed for hypertensive groups. While it makes good sense to explore the possibility of brain damage for hypertensives suffering from severe cerebrovascular complications, it would not seem likely that the incidence of brain damage would be substantially higher for hypertensives than for any groups of normotensives randomly selected for study. Yet several investigators (Apter *et al.*, 1951; Boller *et al.*, 1977; Goldman *et al.*, 1974; Shapiro, 1982) have concluded that medically screened, uncomplicated hypertensives suffer from brain impairment and at least one investigator has suggested specific areas for brain lesions (Fransceschi *et al.*, 1982). The earlier studies in this area suffered from small numbers of subjects, inadequate health screening, and lack of controls for age and education. Several recent studies have been methodologically adequate as far as demonstrating mean differences between hypertensive and normotensive groups (Shapiro, 1982; Fransceschi, *et al.*, 1982). Our objection to their conclusions is based on the fact that comparisons among mean performance levels do not address the issue of brain damage.

An acceptable neuropsychology research protocol requires the investigator to predict, blind to the diagnosis, membership in the brain damaged or nondamaged group. Often this is done on the basis of *cutting scores*. Cutting scores used in neuropsychological test batteries, *e.g.*, impairment indices, are established empirically by demonstrating that the particular score reflecting performance on a battery of tests reliably discriminates between brain damaged and normal individuals. If hypertensives are, indeed, brain damaged, it should be possible, using well-established neuropsychological cutting scores, to assign subjects to hypertensive or normotensive groups with a high degree of accuracy.

Figure 5 summarizes the results of just such an attempt with data from our study using seven measures from the

neuropsychology test battery (Reitan, 1959) and calculating the average index of impairment (Russell *et al.*, 1970). In this case a score above 1.55 is in the range of no brain impairment; a score below is considered in the brain damaged range. The average impairment index is merely the mean score from seven separate test scores, each converted to a value ranging from 0 to 4 and reflecting a range from no impairment to severe impairment. It should be stressed that the index has been well-grounded in the empirical literature and demonstrated repeatedly its validity for accurate discrimination between brain damaged individuals and normal individuals (Russell *et al.*, 1970), using the same tests used in our battery. It may be seen in Figure 4 that prediction is poor; 92 percent of the normotensive subjects are accurately diagnosed as unimpaired (n = 87). But 81 percent (n = 38) of the hypertensive subjects are also diagnosed as unimpaired and this does not fit well with the notion that hypertensives are brain damaged. Perhaps these investigators meant to imply that *some* hypertensives are brain damaged. Indeed, 19.1 percent (n = 9) of the hypertensives were diagnosed as impaired. However, 7.4 percent (n = 7) of the normotensives were similarly diagnosed: *a false positive diagnosis*. Thus our data indicate the hypertensives are no more likely to be diagnosed as brain impaired than normotensives, even though as a group, the hypertensives exhibit lower mean levels of performance.

What about the hypertensives and normotensives who scored in the brain damaged range on the tests? Are all these people brain damaged? Obviously not. Scores on a neuropsychological test are sensitive to a host of factors influencing performance, including brain damage. Careful follow-up testing is necessary to determine what factors are operating in any given individual.

LONGITUDINAL STUDY

Thus far our investigators have answered three of the easier questions: hypertensives and normotensives do differ with respect to mean performance values. However, differences are not large from a practical standpoint, and many hypertensives perform better than normotensives. Thus the prediction of hypertensive status from test scores, or vice versa, is neither good from a correlational nor a clinical-predictive point of view, and the notion that hypertensives are brain damaged can be dismissed on both logical and empirical grounds. The important question of an age by blood pressure interaction has not yet been answered.

Many investigators feel that changes in intellectual functioning that occur in the elderly are largely related to disease processes. Studies such as Wilkie's and Eisdorfer's (1971) longitudinal study of performance on the Wechsler Adult Intelligence Scale (WAIS) are usually cited as evidence for this conclusion. In this study, a group of 60 to 69 year old subjects was given the Wechsler Adult Intelligence Scale, the same test used in our studies. Ten years later the test was given again. As may be seen in Figure 5, normotensives showed no significant change on the performance scales over this time period, even though previous cross-sectional studies had characterized decline as an inevitable consequence of aging. For this age group, only the hypertensives exhibited decline normally associated with aging.

It is important to note, however, that the hypertensives in this study were not uncomplicated essential hypertensives. Most displayed hypertension-associated complications, *e.g.*, retinopathy, cerebrovascular and cardiovascular disease. Further they were all taking antihypertensive medications, and many were taking other medications with potential for influencing performance in a negative manner. Thus the complications and the medications may have resulted in the decline in performance rather than elevated blood pressure *per se*.

On the basis of this finding, and because we began to recognize the problems involved in comparing different age groups, we initiated a longitudinal study in 1976 that has continued to the present time. Figure 6 shows the design of the study. The dotted line box within the overall design shows the measurement points for which we have data thus far. The time intervals between measurements are approximately five and one-half years. Our ultimate objective is to obtain enough measurement points through the adult life span that we can not only observe changes in performance for much younger age groups than those employed by Wilkie and Eisdorfer, but so we can ultimately obtain data for subjects of the same age range as employed by these investigators. The question is whether we will, with our carefully medically treated essential hypertensives, replicate the age by blood pressure interaction observed for Wilkie's & Eisdorfer's complicated hypertensives.

The initial (T-1 versus T-2 for Group 1) study has been completed (Schultz *et al.*, in press). At the beginning of the study, normotensives and hypertensives were divided in two age groups: those 25-39 years and those 45-69 years. However, both age groups exhibited nearly identical performance trends and thus were combined. Figure 7 shows our data for the performance scores on the WAIS (the measure reported by Wilkie & Eisdorfer). Normotensives exhibited no change in performance, and hypertensives exhibited a statistically significant but very minor decline. Differences between hypertensives and normotensives were not significant at T-1 but they were at T-2. The question is whether this extremely modest divergence in performance for the hypertensives and normotensives will become more exaggerated as time goes on. It is important to determine whether this same result will be obtained when T-1 versus

T-2 data for Group 2 have been gathered at the end of the next year. See Figure 6.

Using a smaller number of hypertensives (n = 14) and normotensives (n = 14), we have also completed a study employing the seven tests from our neuropsychological battery discussed previously. In this case, both groups were very similar with respect to performance at T-1. Normotensives and hypertensives showed an *improvement* on the performance of two timed tests, the Tactile Performance Test-time and the Finger-Tapping-Test. Inspection of individual scores indicated that only one hypertensive and one normotensive exhibited a change in the negative direction over the 5.5 year period.

Regardless of aggressive medical treatment, some essential hypertensives will develop complications as time goes on. There are eight such subjects now, too few for meaningful comparisons with uncomplicated hypertensives, but ultimately we should have enough for comparison of change scores and absolute differences in performance. This will require many more subjects than we presently have in the longitudinal study. But it will be recalled that the project calls for several longitudinal studies running in parallel, but starting at different times. If we find that differences between these groups are minimal, we can then combine all T-1, all T-2 *etc.*, scores to achieve a larger sample (as indicated by the bidirectional arrows in Figure 6).

We continue to collect data on every subject possible even from subjects not in the longitudinal study. This will enable us to do subgroup analyses involving

- comparisons of different antihypertensive medications
- comparisons of those who develop complications with those who do not
- comparisons of subjects with high, normal and low plasma renin activity levels.

We hope to obtain enough subjects to allow a comparison of hypertensives who do, and who do not, respond to the apparent stress of testing with elevated levels of plasma epinephrine and norepinephrine.

Longitudinal data on healthy aging subjects, our normotensives, are of considerable interest because there is mounting evidence that progressive decline in intellectual functioning from middle adulthood to old age may be related to illness and may be a result of the cross-sectional design in which different age groups are compared. Our data speak to these issues, but one must remember that they do so for a highly educated sample of individuals who recognize the value of careful detection and treatment. Work needs to be done at all socioeconomic levels, and we plan to become part of a large national study with this objective.

Acknowledgements

The most important element in our longitudinal study has been our subjects, more correctly termed participants. Without their willingness to return for retesting every five and a half years, there would be no study.

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TABLE 1 Glossary

Arteriosclerosis is a group of vascular diseases which are characterized by hardening, thickening, and loss of elasticity of the vessel walls.

Atherosclerosis is an occlusive disease that influences the nutrient vessels of the brain, heart, and kidneys by a progressive choking of the flow of blood. It is almost inevitably associated with advancing age, but may not be clinically significant if it does not interfere with blood flow in a significant manner.

Blood pressure (arterial blood pressure) is determined by the amount of blood that is pumped by the heart per unit of time and the diameter of the blood vessels.

Cerebrovascular accident is the technical name of a stroke. An ischemic stroke is defined as interruption in cerebral blood flow which results in one of several neurological symptoms lasting for more than one day.

Diastolic blood pressure is the minimum pressure occurring during the relaxation phase of the cardiac cycle.

Essential hypertension is hypertension with no immediately identifiable pathology. It is very likely a common symptom of a number of disease processes.

Mean arterial pressure is 1/3 pulse pressure + diastolic pressure.

Normal blood pressure (normotensive) has never been precisely specified as it depends in part on statistical averages and clinical judgement as to what is not normal. A commonly accepted set of "average" values is 120 systolic and 80 diastolic, although 140/90 mm Hg, 145/90 mm Hg, and 150/95 mm Hg have been considered the upper limits of normal depending on an individual's age. Within the normal range of blood pressure, a sudden upward rise might be considered abnormal for a given individual.

Pulse pressure is the difference between systolic and diastolic blood pressure values.

Secondary hypertension is hypertension caused by a known disease process such as heart disease or renal insufficiency.

Systolic blood pressure is the maximum pressure occurring during the systolic (contraction) phase of the cardiac cycle.

Transient ischemic attack is a term reserved for ischemic stroke with symptoms that clear up after a day or seconds and may be associated with cell death.

Table 2
Brief Summaries of the Content of the Neuropsychological Tests Used in Our Studies of Essential Hypertension*

Category Test. The Category Test is the most sensitive test of brain impairment in the Halstead-Reitan battery. It involves abstract concept formation and the ability to form learning sets. Subjects are presented with stimulus figures. After viewing a set of four figures, the subject is asked to indicate which of the four figures is "correct." Correctness is based on stimulus dimensions which change through the series of presentations. The total number of errors on the test is recorded.

Tactual Performance Test (TPT)-Time Component. This test measures ability to adapt to a novel problem-solving situation and to perform a tactual-sensory motor task without the aid of visual cues. The subject is blindfolded and instructed to form wooden blocks of different common geometric shapes into a form-board. The task is performed first with the preferred hand, next with the nonpreferred hand, and finally with both hands. The sum of these three scores is used as a time score. (In clinical settings, times required to complete the task with the right, left, and both hands are compared.)

TPT-Memory Component. This test measures ability to recall spatial relationships. After the subject completes the timed portion of the TPT (see above), the formboard is removed from sight and the blindfold is removed. The subject is then required to draw the forms of the blocks that were placed in the formboard (but not seen). The score is the number of shapes correctly reproduced. The score is determined by whether or not the shape is correctly recalled, rather than the accuracy of the drawing *per se*.

TPT-Localization Component. This portion of the TPT measures the ability to remember spatial relationships. When the subject is instructed to draw the blocks for the TPT-Memory Tests, he is also asked to place the blocks in their correct relationships with respect to their relationships on the formboard. The score is the number of shapes that are drawn in their correct position.

Finger Oscillation (Finger Tapping) Test. This test measures speed of tapping with the index finger. A special key arrangement records the number of taps. The mean of five consecutive 10-seconds finger taps (within five of each other) is computed for the preferred hand and the nonpreferred hand. (In clinical practice, performances for the dominant and nondominant hands are compared.)

Trail-Making Tests (Parts A and B). These tests measure the subject's ability to scan visual materials and to perform sequential behavior under time constraints. Circled letters or numbers are arranged in a scattered manner on a sheet of paper. There are two parts to the test. For part A, the stimuli are circled numbers, and the subject is instructed to draw lines to connect the numbers in order. For Part B, the stimuli are circles containing numbers or letters. The subject is instructed to draw lines from numbers to letters in an alternating numerical-alphabetical sequence. The time to complete each test is recorded as well as the number of errors made in connecting the circles. (In clinical practice, performance for letters versus performance for letters and numbers are compared.)

Impairment Index. This index provides a summary of scores on the battery that fall in the brain-damaged range based on cutting scores established by Halstead and Reitan. The Impairment Index indicates the proportion of tests failed. It ranges from 0 to 1.0. Scores of 0.5 and above are generally considered in the brain-damaged range. The index is normally calculated on the basis of 10 scores but a prorated score can be obtained for five tests.

Note: These are only some of the many tests employed by Halstead and Reitan (see Reitan and Davidson, 1974).
*From D. Klisz in the Clinical Psychology of Aging. Reprinted by permission.

	AGE	
	Pearson r	Partial r[a]
Mean Arterial Blood Pressure	.18*	
Performance Measure		
Categories	.39***	.29***
TPT Total	.48***	.39***
TPT Memory	−.25**	−.12
TPT Localization	−.40***	−.35***
Finger Tapping	.01	−.08
Trails A	.35***	.31***
Trails B	.38***	.35***
WAIS Verbal	−.15*	−.08
WAIS Performance	−.36***	−.31***

Table 3. Correlations of age with performance measures and blood pressure values (n-137)

Note. [a] Correlations adjusted for the effects of mean arterial blood pressure. *p < .05, **p < .01, ***p < .001

Performance Measure	Mean Arterial Blood Pressure Pearson r
Categories	.34***
TPT Total	.16*
TPT Memory	−.37***
TPT Localization	−.31***
Finger Tapping	.01
Trails A	.08
Trails B	.12
WAIS Verbal	−.24***
WAIS Performance	−.22**

Table 4. Correlations between Mean Arterial Blood Pressure and performance measures. *p < .05, **p < .01, ***p < .001

Predicators	Categories Test Errors Pearson r	r^2(%)
Age	.39***	15.2
Education	-.36***	13.0
Mean Arterial Pressure	.34***	11.6
State Anxiety	.07	0.5
Trait Anxiety	.02	0.0
Depression	.15*	2.3
Multiple Composite	.53***	28.0
Mean Arterial Pressure[a]	.18*	3.2
Mean Arterial Pressure[b]	.17*	2.9

Table 5. Prediction of Categories Test errors.

Note. [a] Partial correlation adjusted for the effects of age and education.
[b] Partial correlation adjusted for the effects of age, education, state and trait anxiety, and depression.

*p < .05, **p < .01, ***p < .001

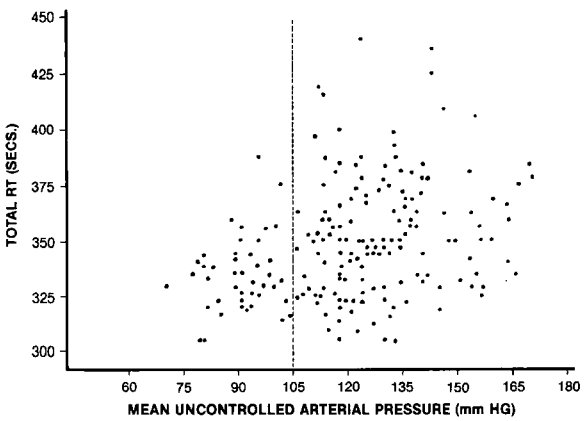

Figure 1. Relationship between total reaction time and uncontrolled blood pressure for all 203 subjects (r = 34, w = 0.12). Vertical dashed line separates normotensive and hypertensive subjects. Taken from K. C. Light [1980] by permission of the author and publisher.

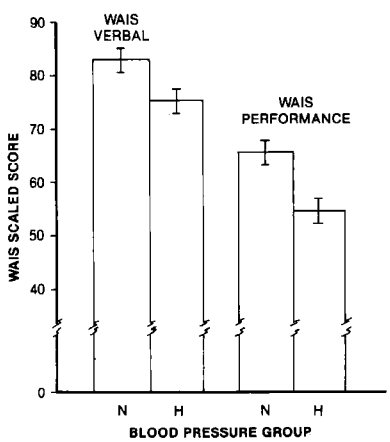

Figure 2. Mean WAIS Verbal and WAIS Performance scaled scores and standard errors of the mean for blood pressure groups (N = Normotensive, H = Hypertensive)

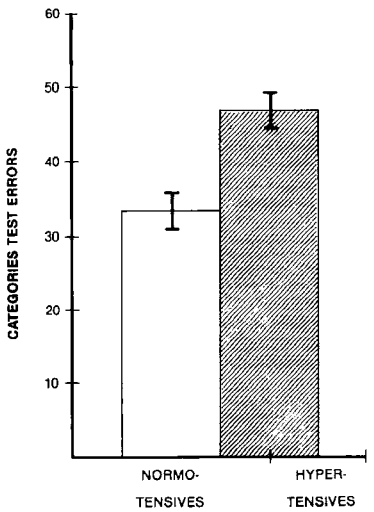

Figure 3. Mean Categories test scores (total errors) and standard errors of the mean for blood pressure groups.

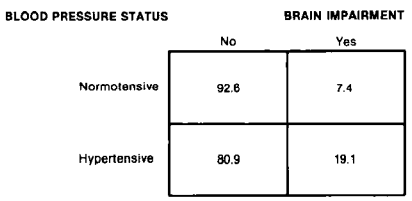

Figure 4. Distribution by percentage of normotensives and essential hypertensives into impairment categories.

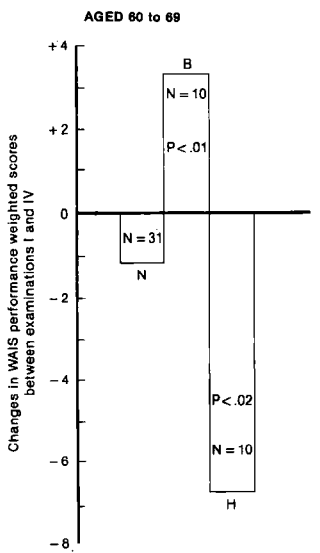

Figure 5 Intellectual change (Delta Scores) over a 10 year period as measured by the WAIS among individuals intially examined at ages 60 to 69 with either normal (N), borderline (B), or heightened (H) diastolic blood pressure on the initial examination. Taken from Wilkie and Eisdorfer [12] by permission of the authors and the publisher. Copyright © by the American Association for the Advancement of Science.

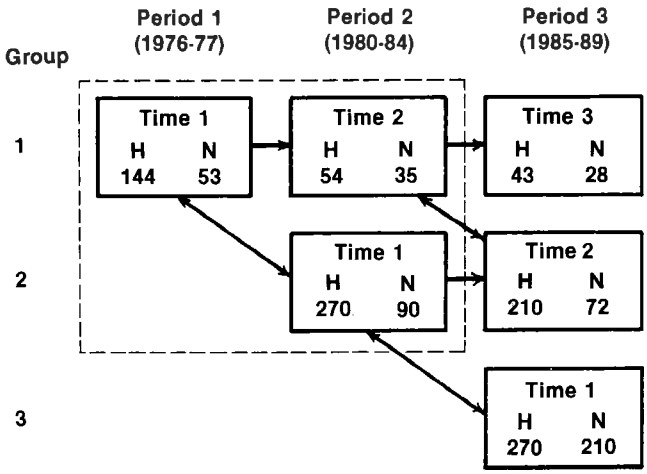

Figure 6. Research design for the overall study, with numbers of hypertensives (H) and normotensives (N) tested or proposed to be tested.

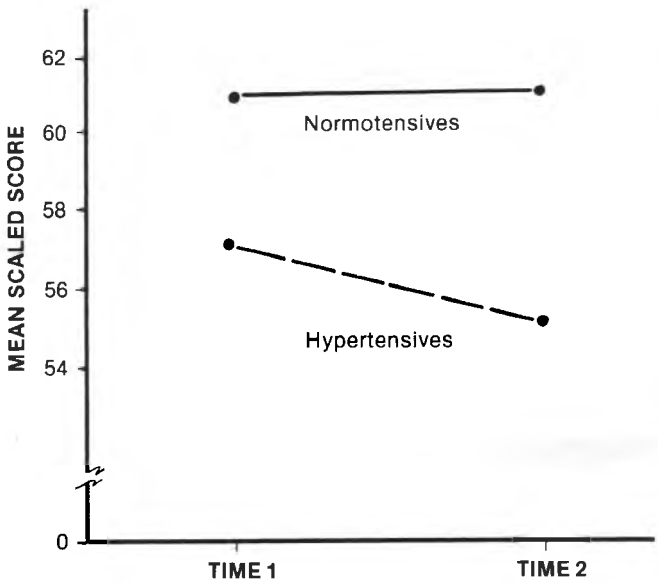

Figure 7. WAIS Performance Scaled Scores (non-age corrected) at Time 1 (1976-1977) and Time 2 (1980-1984) for normotensives and uncomplicated essential hypertensives.

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FROM CAMPUS TO PUBLIC SCHOOLS

New Terrain, oil on canvas, 1985, is composed of three panels each measuring 7' by 3 1/2'. The completed work, with overall dimensions of 7' by 10 1/2', is located in the Library of the Brunswick Junior High School, Brunswick, Maine.

The particular painting was executed as a commissioned painting under the Maine State Commission on the Arts and Humanities Percent for Art Project.

Linehan, the featured cover artist for this issue of EXPLORATIONS, also has works included in collections at the Fayetteville Museum of Art, Fayetteville, N.C.; Madison Art Center, Madison, Wisc.; Appalachian State University, Boone, N.C.; University of Wisconsin-Madison, Wisc.; St. Andrews Presbyterian College, Laurinburg, N.C.; Yarmouk University, Irbid, Jordan, and the Bissel Corporation, Charlotte, N.C.

The artist in his studio with completed work May, 1985, Bangor, Maine.

One of the most basic needs of human beings is shelter. And as humans have moved out of the cave and begun constructing homes in which to live, new questions and problems have arisen. Cost is a basic problem, and high costs can make some homes impossible for many people. Government regulation is another force impinging on shelter: some of it protects the people who wish to take advantage of relatively low-cost housing, and some of it protects neighborhoods and even towns from what many view as questionable or even absurd threats. The mobile home is affected by all of these forces.

A CEILING ON SHELTER

by Peggy K. Schomaker

Buying a home is for most families a big if not the biggest expenditure of their lifetime. On a day-to-day basis, housing takes a great share of the money that families spend for current living. The Bureau of Labor Statistics Family Budgets show that 30 percent of the total consumption in an intermediate budget in the United States goes for housing (U.S. Department of Labor, 1982).

To own one's own home is an American dream. But high prices and high mortgage rates have made the purchase of a single family house very difficult for the average American family. As the prices of new site-built houses have risen, more Americans have been priced out of that market, and they have turned to mobile homes.

Mobile homes have become important in today's housing. More than 10 million Americans live in approximately 5 million mobile homes in our country. In 1983, 32 percent of all single-family homes sold and 82 percent of those under $50,000 were mobile homes (Manufactured Housing Institute, 1984:6).

Mobile homes are an important source of affordable housing for low and moderate income families, for first-time home buyers, and for the elderly. In 1983, the median sales price in the United States was $75,300 for a new one-family house and $70,300 for an existing one. On the other hand, a new mobile home placed for residential use had an average price of $21,000 (U.S. Bureau of the Census, 1985).

Sixty-three percent of the mobile homes in the United States are located in rural areas. Many rural areas have limited housing: there are few apartments available and many of the affordable older homes need repairs. Although a mobile home does not provide the living space of a single family site-built home, its space is quite close to that found in an apartment.[1] Mobile homes with their modern heating and plumbing systems, new equipment and furnishings fulfill the needs of many people in rural areas much better than other types of housing they can find.

Peggy K. Schomaker is Associate Professor of Consumer Economics and Management in UMO's School of Human Development. She earned her Ph.D. at Michigan State University where she did her reseach on financial decisions of families. Her current interest is major financial decisions of consumers with a focus on housing.

MOBILE HOMES IN THE STATE

Of the six New England states (Connecticut, Maine, Massachusetts, New Hampshire, Rhode Island, and Vermont), Maine has the largest number of mobile homes (35,009). On the other hand, Maine has the lowest median family income ($16,167) and the lowest per capita money income ($5,768) of the six New England states. Maine also has the highest percentage of persons living below the poverty level (13 percent). Mobile homes may be the only affordable housing for many people in the State.

	Mobile Homes Year-Round Units	Median Family Income	Per Capita Money Income	% of Persons Below Poverty Level
ME	35,009	$16,167	$5,768	13.0
NH	20,795	19,723	6,966	8.5
MA	14,399	21,166	7,458	9.6
VT	13,631	17,205	6,178	12.1
CT	9,097	23,149	8,511	8.0
RI	2,535	19,488	6,897	10.3

Source: Data derived from 1980 U.S. Census. Income figures are those reported for 1979.

Another reason that Maine may have the largest number of mobile homes in the New England area is that it has the land for placement of mobile homes. Maine has the lowest population density per square mile of land area (37). On the other hand, Rhode Island has the highest density population per square mile of land area (908), and the lowest number of mobile homes.

Of the year-round housing units in the state, mobile homes comprise 8 percent: duplexes, 9 percent; multi-units, 17 percent; and single family homes, 66 percent.

WHAT ARE THE MOBILE HOMES LIKE?

The size of a mobile home as measured by number of rooms is smaller than the average housing unit in Maine. In 1980, the median number of rooms in mobile homes was 4.3 compared to 5.2 rooms for all housing units. Mobile homes that were owned had more rooms than those that were rented. Also, mobile homes had fewer bedrooms than the average housing unit.

Eighty-six percent of mobile homes in Maine had central heating systems compared to 78 percent for all housing units in the State. The fuel used most for heating was fuel oil or kerosene. (Gas or electricity are the fuels used most nationwide.) A higher percentage of mobile homes had air conditioning and complete plumbing facilities for their exclusive use[2] than all housing units in the State.

In 1980, 62 percent of the mobile homes in Maine were no more than 10 years old, 28 percent were 11-20 years old, and 10 percent were more than 20 years old. The mobile homes are older than the national average, but Maine's housing stock in general is also older.

Nearly 80 percent of the mobile homes in Maine are located in rural areas. It is therefore not surprising that 73 percent of the homes have a septic tank or cesspool for their sewage disposal and 57 percent get their water from a well. In comparison, only about half of the total housing units in the State are located in rural areas; 46 percent have a septic tank or cesspool, and 35 percent, a well.

WHO LIVES IN MOBILE HOMES?

Mobile homes are used by a cross section of low and moderate income households in Maine. Forty percent are households with the householder under 35 years of age; 42 percent are those in the middle stage of the life cycle (35 to 64 years), and 18 percent are those with the householder 65 years or older. A higher percentage of householders under 35 live in mobile homes than in all other housing units in the State.

Nearly 60 percent of the households living in mobile homes are married couples. Sixty percent of the households have no children under 18 years of age, and 17 percent have only one child under 18. The median number of persons living in a mobile home in Maine is 2.33. This is less than the number in all housing units.

Incomes were lower for households in mobile homes than for all households in the state. The 1980 Census shows that the median income for all households in the state was $13,816. The median income for households living in owner-occupied mobile homes was $11,777 and the median income for households in renter-occupied mobile homes was $8,705. Twenty-one percent of households renting a mobile home spend 50 percent or more of their income on rent.

Eighteen percent of households living in mobile homes in Maine had incomes below the poverty level. This is compared to 9 percent below the poverty level for all households in the state. Fifteen percent of the households in owner-occupied mobile homes and 31 percent of those in renter-occupied mobile homes had incomes below the poverty level.

Eighty-five percent of the households were living in owner-occupied mobile homes and 15 percent in renter-occupied mobile homes in 1980. The householders who owned their mobile homes were older than those who rented.

GROWTH IN MOBILE HOMES - 1979 TO 1980

From 1970 to 1980, Maine had 126 percent increase in its mobile homes but only a 28 percent increase in its single family site-built homes. Maine had the highest percentage increase in mobile homes of any of the New England states and the lowest percentage increase in single family homes.

In the wealthier New England states, the growth of mobile homes has been restricted by zoning ordinances. In some municipalities mobile homes have been banned entirely and in other municipalities, they have been restricted to mobile home parks.

All of the 16 counties in the state except Franklin County had more than a 100 percent increase in their mobile home stock from 1970 to 1980. The two counties, Washington and Waldo, that had the greatest increases (166 and 156 percent) in mobile homes had the lowest per capita incomes in the state. On the other hand, the three counties, York, Kennebec, Cumberland, that had the greatest growth (43, 41, 40 percent respectively) in single family site-built homes had the highest per capita incomes in the state.

Cumberland and York Counties added more total housing units to their stock from 1970 to 1980 than any other counties: Cumberland added 18,721 units, York added 14,548 units. Both had the highest per capita incomes in the state. These two counties also experienced high rates of growth in their mobile homes, 136 and 149 percent increases.

Sixty-seven percent of the municipalities in the state had 100 percent or greater increase in their mobile home housing from 1970 to 1980.

If we look at the number of new mobile homes and the number of single family site-built housing starts in Maine for the year 1980, we will see that more than one-third of the new homes for single families were mobile homes. New mobile homes (1,665) amounted to 50 percent of the single family site-built housing starts (3,298).

In 26 percent of the municipalities in Maine, new mobile homes were equal to or exceeded the number of

single family site-built housing starts. See map. In 19 towns, mobile homes were the only additions to the housing supply.

The map also shows that in 14 percent of the municipalities, new mobile homes equaled 50 to 99 percent of the single family site-built housing starts, and in 16 percent, new mobile homes equaled 1 to 49 percent of the housing starts. Twenty-two percent of the municipalities had no new mobile homes in 1980 and another 22 percent had no new mobile homes or housing starts.

OUTLOOK FOR MOBILE HOMES IN MAINE

By 1990, we will most probably see a higher quality and a greater number of mobile homes in Maine.

Our newer mobile homes have been upgraded in quality by the National Manufactured Home Construction and Safety Standards Act of 1974 which was established and is enforced by the Department of Housing And Urban Development. All mobile homes built after June 15, 1976, must meet HUD standards. These cover construction of body and frame, plumbing, heating and electrical systems of the home. Fire retardant materials are required near the furnaces, water heater, and range. Insulation must meet the needs of different climates. Safety requirements include two exterior doors spaced far apart in the mobile home, at least one egress window in each sleeping room, smoke detectors, and tie-down systems. Every new home must bear a seal indicating that it was built to these standards.

In 1983, the Maine Legislature through its passage of a new law, *Regulation of Manufactured Housing* (Title 30 MRSA 4965), made it easier for consumers to place their new mobile homes on individual house lots in the different municipalities. This new law required that by January 1, 1985, cities and towns with overly restrictive zoning ordinances change them to allow mobile homes manufactured after June 1, 1976, to be located on individual house lots in a number of locations where single-family dwellings are permitted. Thirty-nine municipalities in the state were affected by this new law. The Maine State Planning Office (1985) reported mid-year that 33 of the municipalities had amended their ordinances to comply with the requirements of the new law.

In *Building Tomorrow: The Mobile/Manufactured Housing Industry*, Bernhardt states that *the world needs more, better, and lower cost shelter* (1980:IX). He takes the case of the mobile home industry in the United States and shows that *this industry can and does produce low cost housing without impairing quality*. Bernhardt believes that the *mobile home industry is the most efficient building industry in the world* and that this ability can be extended beyond the industry's current product. The mobile home industry, he says, with the traditional building industry, can produce low cost shelter, of high quality for more people of the world.

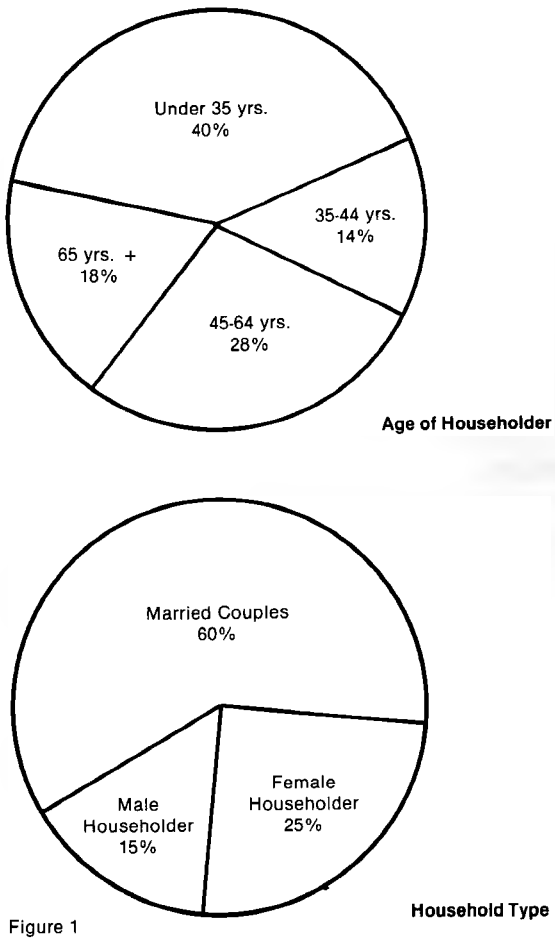


Figure 1

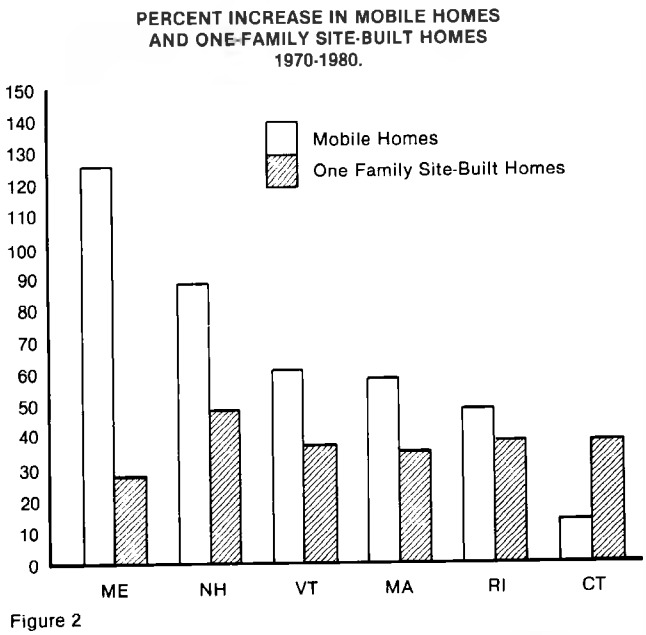
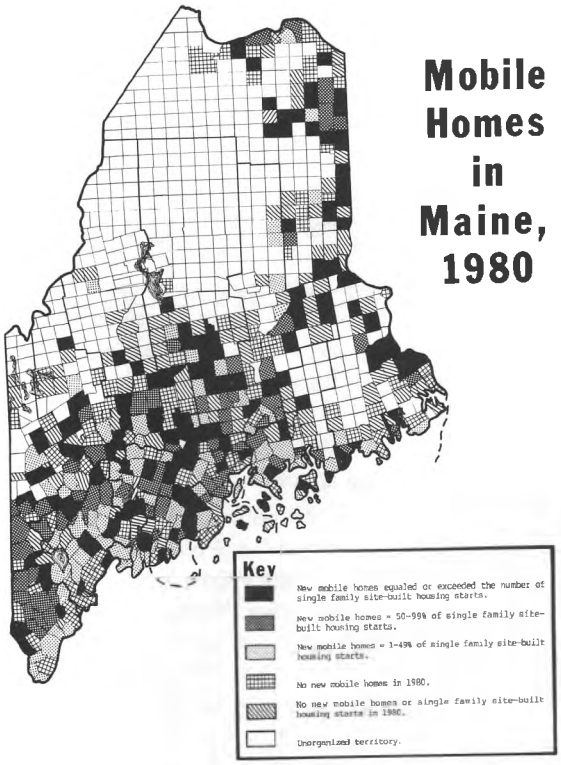


Figure 2



FOOTNOTES

1. The average square footage per household in 1981 was 880 square feet for a mobile home, 826 square feet for an apartment in a building with 5 or more units, and 2,093 square feet for a single family house. Source: U.S. Energy Information Administration, *Residential Energy Consumption Survey*: Housing Characteristics, 1981, August 1983.
2. *Complete plumbing for exclusive use* consists of units which have hot and cold piped water, a flush toilet, and a bathtub or shower inside the housing unit for the exclusive use of the occupants of the unit.

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Although the abundance of land in Maine makes it possible for some mobile homes to be sited attractively, clockwise from upper left, many towns and cities require that they be located in designated parks. Older mobile homes in Maine are often built around or built into other structures. Fire in mobile homes can follow the improper installation of wood-burning stoves, and injuries commonly follow in older mobile homes with small crank-operated windows which do not allow human egress.

Readers of EXPLORATIONS met Professor R.D. Blake a year ago when we published THE DNA MOLECULE: Mapping its Mysteries. Inclusion of his most recent work with DNA appropriately christens our new update section: From the Dispatch Case.

FROM THE DISPATCH CASE

Control of Cell Growth at the Level of the Genetic Code

by R.D. Blake

More than 400 gene sequences from *E. coli* and common bakers yeast have been characterized according to the nature and level of average codon use. There is a high correlation between the levels of codon bias and the abundance of protein products, indicating that the biased pattern is exploited by these organisms for the production of widely different levels of gene product. In *E. coli* this relationship is especially striking in genes necessary for rapid growth and cell division, for which this bacterium is best known. Overall, the genes for these processes generate about five-fold more protein than the average gene, and use about five fewer codons. The codon bias in yeast sequences is even more pronounced. One large family of sequences uses an average of 38 out of 64 possible codons and another family uses just 29 codons. Bias levels in this entire latter group, some of which use only 27 codons, far exceed those of any other sequence examined thus far. Sequences in this highly biased group are dominated by genes for enzymes of glycolysis and fermentation, for which this microbe is best noted and used.

Mouse and human sequences indicate similar broad distributions of average codon use with few examples of sequences with extraordinarily high levels of codon bias found in yeast and *E. coli*. The bias in the mouse genome has remained conserved to a remarkable degree in the human genome, indicating a sensitivity or resistance to change are important factors in the preservation of the biased codon pattern in more complex cells.

Within the past few decades, the development of powerful new methods for studying cells and their molecular contents has led to significant advances in our understanding of their behavior.[1,2] Longstanding questions are beginning to yield answers: *How do cells grow and carry out their routine housekeeping chores; how do they develop into specialized tissues; how do they differ among different species; how do they evolve; how do they multiply; and how do they go awry* (with dramatic self-destructiveness) *as in cancer or upon infection by certain viruses*? Considerable mystery about the workings of cells still exists, yet what we have learned these past few decades is quite remarkable when viewed against the knowledge acquired over centuries.

Little of this vast new knowledge has made its way into the public consciousness. One reason is, of course, that much of it is still too fragmentary; I expect, however, the principal reason is that the inquiring mind is besieged by too many new developments. Moreover, developments of a technological nature command our priorities since they promise widespread and immediate benefit. This was brought home recently when I received an article of mail soliciting subscriptions to a new journal on *high technology*, which stated: *most of what's happening in pure science today you can safely ignore*. As this new biology moves irrevocably through the successive stages of discovery, analysis and application, in just a matter of a few short years we may expect to experience a tremendous impact of technological developments in cellular and molecular biology. Already new and better drugs are in production by processes made possible through the manipulation of bacterial genes using laboratory techniques not known or imagined as recently as 1965.[3]

One feature of cells that has attracted our attention lately is the extraordinary capacity shown by certain primitive microbes to react in very characteristic ways to environments that may swing abruptly from extremes of plenty to scarcity. Consider the remarkable endurance of *E. coli*, the ancient bacterium that makes up most of our intestinal flora. *E. coli* has survived as well as it has because of its fitness in such an environment; its ability to multiply in sudden bursts when conditions are favorable is legendary. One cell becomes a billion cells in just 10 hours. The ability to grow so rapidly has made *E. coli* the premier laboratory specimen of researchers and more recently it has become the favored microbial *factory* of the pharmaceutical industry for the production of rare and expensive drugs and diagnostics. The production of insulin, growth hormone and numerous other complex and exotic drugs can now be produced at a portion of their

previous cost. Human growth hormone is normally produced in a tiny gland at the base of the brain, but tumors or genetic disorders can prevent its synthesis, thereby leading to abnormally short stature or dwarfism. The remedy may require daily injections of growth hormone at a cost of approximately $40. Such a remedy is obviously not available to everyone who needs it. Previously, the only available source of this hormone was the glands of cadavers, the gland of one cadaver supplying enough hormone for just three injections. By contrast a single, genetically-engineered *E. coli* cell with the capacity to produce just one molecule of human growth hormone each minute will turn out one pound of the stuff in just 24 hours.

The remarkable fitness of the common yeast used by brewers and bakers for millenia has similarly attracted our interest. The unique ability of yeast cells to carry out anaerobic fermentation of sugars with extraordinary speed and efficiency preserves for this ancient microbe the title of being our single most important living tool. The different growth patterns of *E. coli* and yeast are due to their different genetic makeups. What molecular differences must exist in the genes of these two microbes to provide them with the special chemical reactions we recognize as being characteristic of the species?

One of the most active (and controversial) areas of biological research has been on genes at the molecular level, which I refer to as molecular biology. The genes of all creatures are made of DNA. Genetic instructions encoded at the molecular level are written using the same chemical alphabet in organisms as different as *E. coli* and *homo sapiens*. That's why genes for exotic hormones are interchangeable between human brain cells and lowly microbes. The initial observations on the molecular structure of genes, which only began in the 1930's and 40's, were made by some rather colorful individuals. Even those not involved in the science have found themselves captivated by the human side of this subject, if the tremendous success of books by J. D. Watson, H. F. Judson and E. Chargaff is any indication.[4,8]

It is generally considered that W. T. Astbury, an English X-ray crystallographer, was first to begin serious study of gene structure. Others following soon after included the microbiologist O. Avery, and, of course, J. D. Watson and F. H. C. Crick. There is no question that Watson and Crick's classic one-page paper on the structure of DNA in 1953,[9] was the key impetus to the explosive growth of molecular biology. In one quick stroke their discovery laid out the entire basic scheme for gene expression in all living organisms. The two intertwined strands of the DNA helix, like spiral staircases, are joined by relatively weak bonds. See Figure 1. The innermost chemical groups that are directly involved in these weak bonds are the only distinguishable feature of each rung of the helical ladder. The remaining chemical groups making up the repeating backbone on the outside of the helix are the same throughout. The sequence of the innermost segment of the helix consists of only four different chemical groups, abbreviated A, T, G, C (paired respectively to T, A, C, G in the opposite strand) yet they can spell the instructions for everything that lives. The sequence of As, Ts, Gs, and Cs represents the special hieroglyphics of genetic instructions, and since DNA is the master copy of all instructions needed by the organism, it must be quite long. In fact it is more than six feet long in the human cell. (The operation of the new compact disks for high fidelity music is remarkably analogous to the operation of the genetic information storage system. Compact disks also store information digitally, in a continuous linear array that is everywhere equally accessible.)

After arriving at a structure for DNA, Watson and Crick saw immediately the possibilities for the molecule to perform its two principal functions.[9] On the one hand, the two strands can be peeled apart, separated and copied (*replication*) so that exact copies of the genetic information in the DNA of the parent cell can be distributed to the two daughter cells during cell division. On the other hand, the two strands can undergo partial separation at specific places, allowing copies to be made of single genes (*transcription*) for distribution into the cell for one mission or another. The structure of DNA is the perfect essence of its functions. These functions are common to all cells; consequently the scheme, summarized in Figure 2, is referred to as the *Central Dogma* of biology.[4] In this scheme, DNA prescribes the synthesis of both of its own strands along their entire lengths during *replication*. It also prescribes the synthesis of faithful copies of just short stretches (from just one strand, the *sense* strand, of the helix, by the way) in a process called *transcription*. These single-stranded copies made during the latter process have a slightly different chemical structure than DNA and are called RNAs. Since they are transcripts of single genes off the DNA, they are only a tiny fraction as long. RNAs are produced from different genes in the DNA as the cell requires, since they, in turn, prescribe the synthesis of proteins. In the cellular feudal system, protein molecules perform all the complex tasks that we refer to as the *life-force* of cells. They are the subservient *betas* of Aldous Huxley's parlance in *Brave New World*. Enzymes, skin, insulin, muscle, growth hormone, hemoglobin, eye lens, hair, skin, antibodies, blood clot, tortoise shell, horn, are all protein. Protein has a totally different chemical makeup than DNA or RNA and so the flow of genetic information at this step is called *translation*. Clearly, the occurrence of mistakes of one sort or another in the DNA sequence may be seen as changes in protein sequence further down the line. Such mistakes can be neutral; they can be deleterious; they can be fatal,

or they can be beneficial. Deleterious mistakes have attracted great attention over the years, and more than 3,000 different *genetic diseases* of faulty protein in humans have been described.[10]

Questions about mistakes in the genetic instructions encoded in DNA sequence leads to a Catch-22 situation: *What actually constitutes a normal DNA sequence or a best protein*? Can we assume the characteristics of individual proteins have been honed to perfection by millions of years of evolution and represent the acme of their type? The answer is a weak, barely audible no. We know that genes are in a constant state of change; not that it's obvious, of course, but as much and as rapidly as the equilbrium between constancy and change allows. Slow evolutionary changes of all species reflect miniscule changes in DNA sequences. It stands to reason that changes will vary in extent and rate in different proteins and, therefore, the corresponding genes. Local equilibria between constancy and change in neighboring genes will also be perturbed, since the fitness of the species is determined by the collective behavior of all its genes. Perhaps, then, we should talk about variances of gene sequences in a population. It has seemed to us that this question of variance and what constitutes a backward, sideways or forward step in molecular evolution is dependent on the type of cell and its niche in the environment. According to our reasoning, free-living, rapidly dividing, single-celled microbes such as *E. coli* and yeast, that have been through countless generations, will exhibit smaller variations in DNA sequences, while the cells of more complex mutlicellular organisms will display greater diversity. Our particular focus on this question of variance is directed at DNA sequences, but our interpretations depend on the relationship of encoded information as it occurs in DNA and protein sequences. This necessitates a further description of the *Central Dogma*.

DNA, RNA and protein are all *informational macromolecules*. The sequence of monomeric units in the DNA, called bases, prescribes the sequence of bases in RNA by the process of *transcription*. The next step in the information flow involves the prescription of the sequence of monomeric units in protein by the process of *translation*. The monomeric units of proteins are amino acids, which are both physically and chemically different from bases. So a translation of the genetic code is necessary at this step. Translation of the genetic code into each amino acid of a protein requires a sequence of three successive bases in RNA, *e.g.*, a triplet code. The reason three bases are needed is that the simple four-letter alphabet of DNA and RNA is insufficient to code in a direct 1:1 way for the twenty-letter alphabet of proteins. Though more than 300 amino acids have been found in nature, only 20 are found in proteins. A triplet code is more than sufficient since there are (4 x 4 x 4) 64 possible ways to arrange the four bases of DNA and RNA into triplets. Three of the extra triplets serve as *stop* signals, while the remaining 61 serve variously as degenerate or synonymous *codons* for 20 amino acids.

The code is universal throughout the biosphere so the list in Table 1 of the 20 amino acids with their corresponding triple-base codons is applicable to *E. coli* as well as *homo sapiens* and everything in between. A striking feature of the genetic code is the different numbers of synonymous codons for each of the 20 amino acids. Some amino acids are coded for by as many as six codons *e.g.*, arginine, leucine and serine, while others have just one codon.

The dimensions of codons and amino acids are very different; therefore the process of translation must allow for a colinear realignment between the two each time the growing protein is extended by one amino acid. A special class of intermediary molecules called transfer RNAs (tRNA) assumes the role of keeping the disparate sizes of codons and amino acids in some sort of linear register. The tRNA molecules are small as RNAs go, but large enough to bind simultaneously to a triplet codon and an amino acid. The whole process takes place on a large particle in the cell called a ribosome that serves more or less as an intelligent workbench for holding the transcribed RNA (mRNA) and tRNAs in place as the process of protein synthesis goes. A sketch outline of the process is shown in Figure 3.

The critical step during protein synthesis is the concomitant linking of one end of tRNA, the *anticodon* end, to the appropriate codon of an mRNA (by the very same weak bonds that join the two strands of DNA); while the other end of the tRNA is held into position by the ribosome to promote the extension of growing protein with the appropriate amino acid. This process goes on step-by-step, amino acid by amino acid until a *stop* codon turns up. There is a specific tRNA for each codon, and if it were not for some *wobbling* of the weak bonds between the anticodons of tRNAs and codons of RNA, there would have to be 61 different tRNAs in the cell to match the 61 codons. With wobbled bonding, the anticodons of some tRNAs can match up with several different codons; even so there are more than 40 different tRNAs in cells.

An interesting question about the codon table is the frequency that synonymous codons for a particular amino acid are used by the organism. Reports by Grantham and his coworkers in Lyon, France,[11,13] as well as some from our own and other laboratories,[14,20] have shown that synonymous codon usage is often far from that expected from a random choice. Suggestions have been made to explain the bias, and some have even gained a certain popularity. Unfortunately, as we have improved and broadened the statistical basis for the bias in different species, we find that most of these earlier explanations

cannot be sustained. Our own thoughts on this phenomenon are that a codon bias may be just what is needed to help explain the differences in growth patterns and metabolic capacities that we described above for *E. coli* and yeast cells. Consequently, we have pursued the question of codon bias in these and other species in some depth. Such a study obviously requires a DNA sequence database of considerable magnitude. With the development in 1975-77 of rapid and efficient methods for determining the base sequence of DNAs,[21,23] a large database now exists. As seen in Figure 4, the database of DNA sequences has been growing exponentially and at this moment contains more than 6 million bases in cumulative length. The database of all sequences published since 1977 is available on magnetic tape, which is the grist for our analysis of synonymous codon usage.[20]

Table 1 indicates frequencies of codon occurrence in sequences from *E. coli*, yeast, mouse and human. (We have included the latter two for comparison and because of their intrinsic interest.) Biases in Table 1 are greatest in yeast and least in mouse and human sequences. We find a good correlation between the maximum level of bias and the number of synonymous codons. Those amino acids with six codons have an average maximum bias of 250 percent in yeast sequences, that is, some codons are being used 2 1/2 times more often than would be expected from a random choice among the synonyms. In yeast the codon AGA is used 72 percent of the time for the amino acid arginine, but is expected to be used only 17 percent of the time. In *E. coli* a different codon, CGT is used 53 percent of the time for this amino acid, three times more often than expected. And, so it goes up and down the table; however, the biases are considerably less in mouse and human. In general, the bias is both quantitatively and qualitatively different among *E. coli*, yeast and mouse; however, the bias in mouse DNA has remained conserved to a remarkable degree in human DNA. Such conservation indicates that vulnerability to survival or resistance to change have been important factors in the preservation of biased codon patterns.

We have characterized a large number of gene sequences from *E. coli* and yeast according to the nature and level of average codon preference.[17] We obtain an average codon probability for a sequence by assigning a probability to each triplet equal to the relative overall frequency of use as a codon and then dividing by the number of triplets. Characterization of gene sequences this way masks local variations within genes, which may be important for control of translation. However, the average allows distinctions to be made between sequences. A distribution was then determined for the variation in average codon probability in both reading and nonreading frame sections of each sequence, and the results illustrated in Figure 5. The average codon probability of triplets in reading frame segments is represented by the clear bars in this figure, and appears to be biomodal in distribution with about 40 percent of sequences having an average probability of 0.028 and the remaining 60 percent with values near 0.024. Sequences with the higher value use an average of just 36 codons while those in the lower group use 42.

The average codon probability of triplets in nonreading frame segments, represented by the crosshatched bars in this figure, form a normal distribution with a mode at 0.0160, which is almost the numerical equivalent of 1/64 and therefore corresponds with the random occurrence of all triplets.

Figure 6 shows the distribution of average codon probabilities for both reading and nonreading frame triplets in yeast sequences.[20] Nonreading frame segments, represented by the crosshatched bars in this figure, have average codon probabilities that cluster with a mean of 0.0155, corresponding almost precisely to the random occurrence of triplets (1/64). A distinct distribution for reading frame segments is not yet apparent, perhaps indicating the population of yeast sequences may still be too small. There seems to be one large block of sequences with an average codon probability near 0.026 (1/38) and another block at 0.035 (1/29). The codon bias levels in this latter group far exceed any found in any single sequence from *E. coli* or any other species that we have examined so far. Indeed, all sequences from yeast indicate a significantly higher bias, with the average sequence using just 34 codons. Those at the extreme upper end of the distribution scale, at 0.037, are using an average of only 27 codons, which is remarkably close to the absolute minimum number.

Analysis of mouse and human sequences indicates similar broad distributions of average codon probabilities,[20] with few examples of sequences with the extraordinarily high levels of codon bias found in yeast and *E. coli*. A more meaningful interpretation of the bias in mouse and human should be possible as more sequences are added to the database.

In summary, we find sequences from the rapidly dividing *E. coli* cell to exhibit a very characteristic pattern of preferences among the synonymous codons. This pattern corresponds to an average level of preference for only 39 codons, and it extends throughout the genome. In yeast, a primitive eukaryotic cell with approximately 1,000 times greater internal volume than *E. coli*, but with only three-fold longer generation time, the average level of preference is for only 34 codons. Some sequences even use as few as 27 codons. Again, the biased codon pattern is distributed throughout all sequences from yeast that have been examined thus far.

A strong case can be made for the concept that biased patterns are exploited by the cell for the production of

widely different levels of gene product.[17] We find high correlation between the level of codon bias, the tRNA population and the abundance of protein product. To achieve a maximum rate of gene expression, we imagine the levels of specific codons will converge to meet those of their cognate tRNAs. We also note the existence of a correlation between the level of codon bias in a gene, and the level of gene expression. Table 2 lists 15 sequences from *E. coli* with the highest average codon probability from among almost 200 sequences in our list. The last column in this table gives the approximate numbers of proteins produced by each sequence. These seem to vary, yet their average output exceeds 50,000 copies. This contrasts with the output of sequences at the lower end of the scale, which is often just a few molecules per sequence. Thus a general consequence of increased codon bias is an increased abundance of gene product. Genes with a high codon bias almost invariably produce, or have the capacity to produce, extraordinarily high levels of protein, while the converse is found among sequences with a low bias.

This table of sequences of highest codon probability is particularly rich in genes involved in the production of components for transcription and translation, denoted in Table 2 by an asterisk. The genes for these constituents comprise only 5 percent of the *E. coli* genome, yet their products make up almost 40 percent of the dry cell mass. These components are essential for rapid growth and cell division; which, as noted above, are the primary characteristics of *E. coli* cells. The occurrence of large codon biases throughout these structural genes for the enzymatic machinery of transcription and translation then provides the basis for a simple, autogenous mechanism for their coordinate synthesis, which operates as follows: the level of RNA polymerase in the cell affects the level of specific tRNAs according to the gene frequencies of the latter. Since the levels of specific tRNAs affect the levels of translation, the expression of those genes with large codon biases will be disproportionately amplified.

Table 3 lists the top 11 sequences of highest average codon probability in yeast, which is 10 percent of the present yeast database. Interestingly, eight of these sequences code for enzymes involved directly in glycolysis and fermentation, which is what this organism has been selected for over the past several millennia.

These observations indicate that biased patterns exist to help fill the need for different levels of gene product. A codon bias offers a primitive, mass-action form of control over the levels of gene expression, therefore, we imagine it would have originated shortly after the code itself. Indeed, we note the bias reflects the widest differences among the most divergent species. Selective pressure would favor the bias so long as existing rates of codon-anticodon interaction during translation were unable to meet the demand for more and different gene product. Of course, the bias would be subject to the counterbalancing pressure for more random codon usage among the synonymous codons; nevertheless, we expect the level of bias for a particular amino acid to exhibit some correlation with the frequency that that amino acid occurs in protein. Figure 7 shows that such a correlation exists.[20] In this figure the maximum frequency a synonymous codon is used is plotted against the frequency that the same amino acid is found in proteins. While there is some scatter, the data clearly supports the existence of a correlation. Since this plot represents an average over all sequences from *E. coli*, a scatter would be expected. A scatter will arise in any case if there are unequal numbers of tRNA genes for each amino acid. The dashed line at the bottom of the figure with the low slope represents the position that would be expected if there were no bias, while the upper dashed line corresponds to that for maximum bias, in which only 20 codons are used. The same plot for the yeast codon pattern shows a steeper slope, and perhaps a slightly better correlation.

I'll close this brief description of our research on the evolution of DNA sequences with two short comments on incidental consequences of the codon bias. First, it is clear that what we have previously labeled as neutral or silent mutations[25] may in fact produce significant physiological or developmental effects. Also, we can expect that the expression of recombinant sequences in either *E. coli* or yeast will generally be less than optimal, particularly if the sequences are derived from higher eukaryotic cells.

Acknowledgements

The work I have described represents the combined efforts of some very enthusiastic and able students and research associates: Phil Hinds, Gary Day, Scott Earley, Jon Blake and Al Hillyard. Our efforts have never lacked for willing and skillful assistance from Merton Nickerson, John Sherblom, Prudence Kennedy and Shawn O'Brien of the University of Maine Computer Center.

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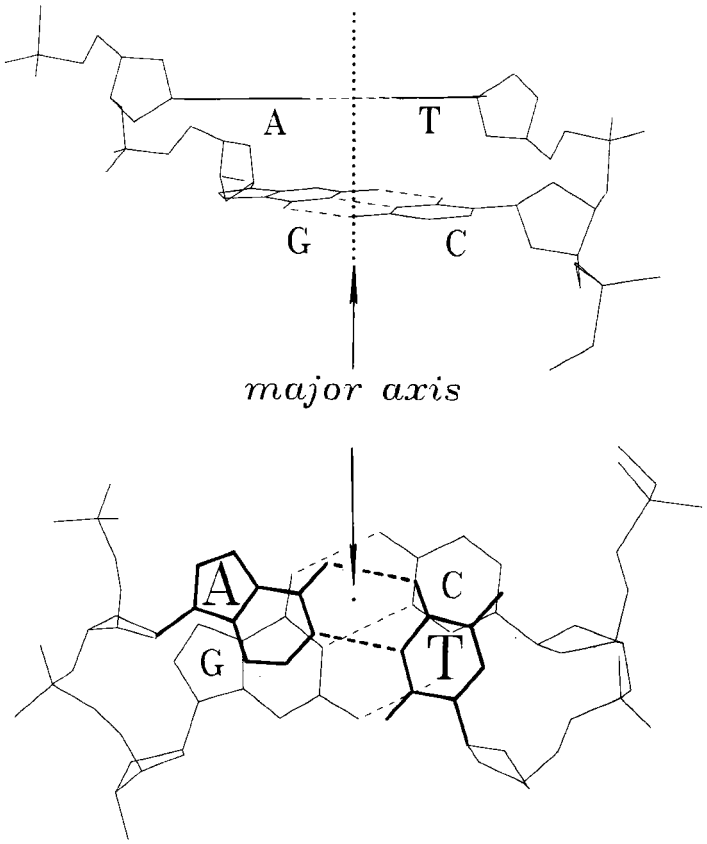
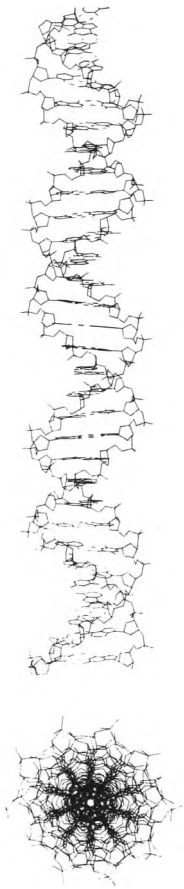




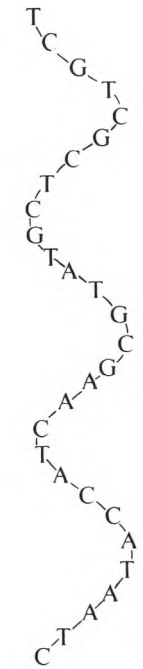
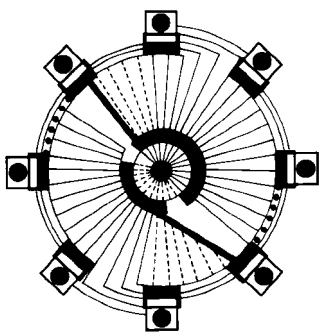
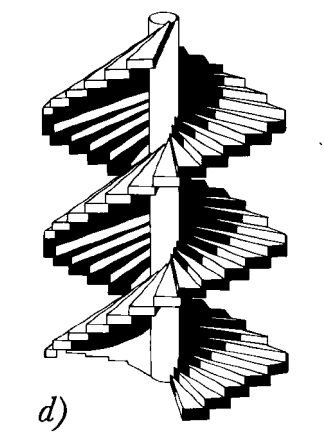


Figure 1. a) b) c)





d)

Figure 1. The double helix. Under *a*) a greatly magnified view of just two segments of the enormously long DNA molecule can be seen in this pair of computer graphics illustrations, showing the structure in very accurate but skeletal detail. Both illustrations under *a*) are exploded views of sections from the pair of illustrations under *b*). The upper view under *a*) shows the major axis of the helix as a dotted line running vertically while the same axis is shown in the lower view coming out of the page (represented by the point). Strong (covalent) bonds are represented by solid connecting lines while the much weaker (noncovalent) bonds joining the two strands of the double helix are represented by dashed lines. The pentagonal elements are the sugars of the phosphate-sugar backbone, and the hexagonal-pentagonal elements are the chemical groups that serve the all-important role as alphabet for the genetic code. The sequence of these groups in one strand of the double helix under *b*) is given under *c*). The sequence under *c*) can be "read" only from top down (the structure of that strand actually differs when looked at from the two ends). The DNA in human cells is approximately 200 million times longer than the sequence illustrated under *c*).

Under *d*) is shown reproductions from original 16th century engravings in the British Museum, of the Grand Escalier, the magnificent double helical staircase at Chambord, the most magnificent of all Loire chateaux. The staircase was designed and built by Denis Sourdeau for Francis I exactly 100 years before the landing of the Mayflower at Plymouth. According to tradition the design originated with Leonardo de Vinci (1453-1519), who was an honored guest of Francis I at Clos-Luce for the last five years of his life. The two staircases, like strands of DNA, wind around one another with a right-handed screw symmetry, never intersecting; so that tenants from among the 440 rooms of the chateau could ascend and descend simultaneously without meeting. The staircase is located in the central keep of the chateau and is surmounted by a 70 foot high lantern tower. Francis I also built a sandstone octagonal tower with helical staircase at Blois which is an architectural masterpiece, rivaled only by the magnificent double helical wooden staircase built in 1839 by Micajah Burnett of Kentucky. One staircase was for the male and one for the female residents of Pleasant Hill Shakertown, with remarkable associations to molecular genetic events.

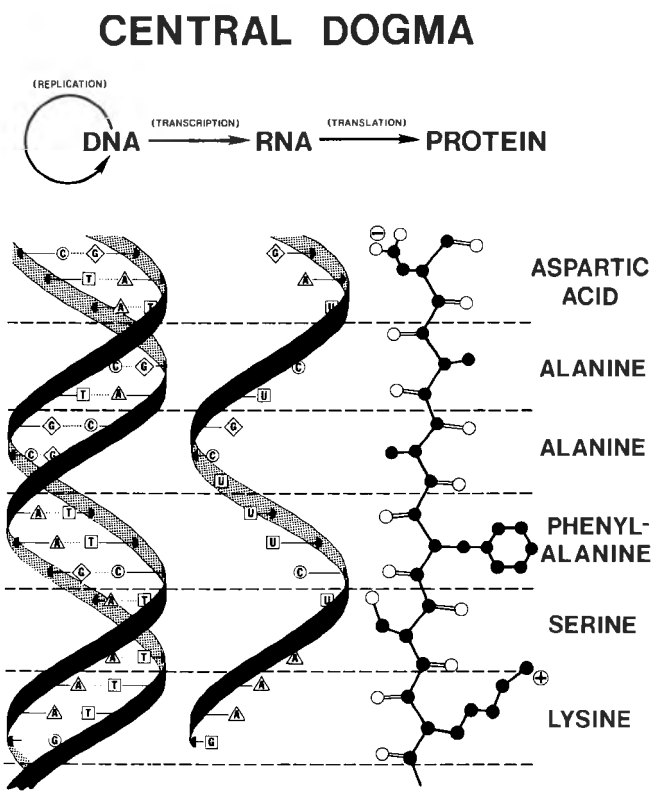


Figure 2.

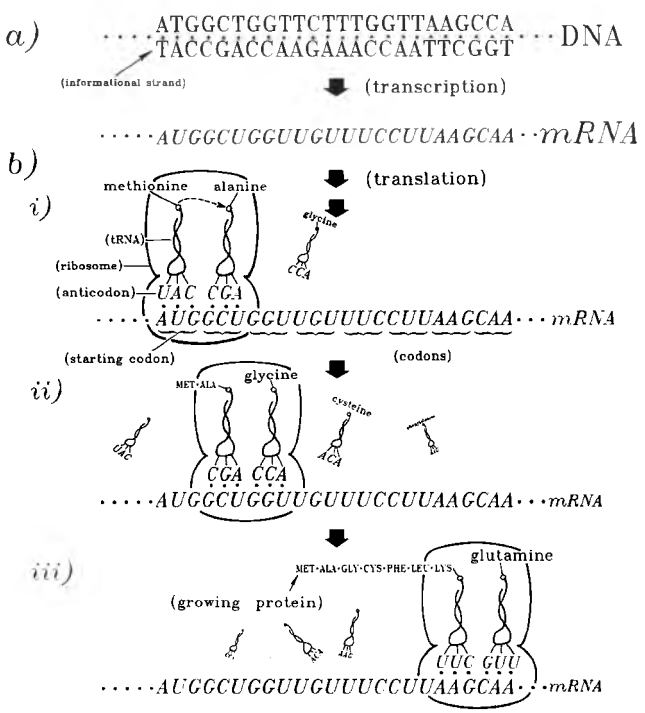


Figure 3.

Figure 2. The "central dogma" of molecular biology: DNA reproduces itself and makes RNA, which makes protein. This is a concise way of saying that the ultimate source of information inherent in the sequence of amino acids in protein is to be found in the sequence of nucleotide units in the DNA of chromosomes.

Figure 3. This figure summarizes the salient features of the translation of the genetic code and protein biosynthesis. *a*) This first step illustrates the transcription process and synthesis of a messenger RNA (mRNA) using one strand of DNA, the informational strand, as template. *b*) Succeeding steps illustrate the translation process. In step *i* the sequence of three monomeric residues in mRNA that signals the starting point, *AUG*, has become associated with the ribosomal particle that serves as a "workbench" for protein synthesis. The tRNA for the starting amino acid is shown to be bound by its specific anticodon to the starting codon of the mRNA. The starting amino acid, methionine, is linked to the opposite end of the tRNA. A tRNA specific for alanine has become bound to the next codon, and sits adjacent to the right of the methionine tRNA on the surface of the ribosome, while the bond joining the methionine amino acid is transferred to the free "tail-end" of alanine, which is close by. This is the first bond to be formed in the synthesis of the protein, producing a simple *di*amino acid that remains attached to the alanine tRNA. The "spent" methionine tRNA is then expelled from its site on the ribosome, allowing the ribosome to ratchet to the right by one codon, so that alanine tRNA with its freshly made MET.ALA *di*amino acid now occupies the vacant left-hand side of the ribosome, as shown in step *ii*. The next codon, *GGU*, codes for glycine, so that a glycine tRNA moves in and occupies the vacant right-hand side of the ribosome. The bond joining MET.ALA to alanine tRNA is then transferred to the free end of glycine close to it, forming a MET.ALA.GLY *tri*amino acid. This process is repeated over and over with the ribosome ratcheting to the right, codon by codon, with the formation of each new bond, so that the resulting *poly*amino acid grows longer and longer, as shown in step *iii*. Further elongation of the nascent protein finally ceases when the ribosome encounters a "stop" codon; at which time the freshly minted protein is detached, to diffuse to the site where it is required.

Figure 4. Variation in the number of nucleotide bases that have been sequenced with time. (Note that the vertical axis increases on a logarithmic scale.) The open circles represent sequenced RNA molecules only, since the ability to sequence DNA molecules was not developed until 1975-1977.

Figure 5. Distributions of average codon probabilities in assigned coding (clear bars) and noncoding regions (crosshatched bars) in *E. coli* sequences. The correspondence between coding probabilities and number of codons being used can be determined from the upper horizontal scale.

Figure 6. Distribution of average codon probabilities in assigned coding (clear bars) and noncoding regions (crosshatched bars) in yeast sequences.

Figure 7. The maximum codon frequency among synonymous codons for each amino acid in *E. coli* coding regions is plotted in this figure against the frequency that that same amino acid is found to occur in *E. coli* proteins. The line labeled 'random choice' represents the variation we would expect if each codon were used randomly from among each synonymous family. The line labeled 'maximum bias' represents what we would expect if one codon were used exclusively from among each synonymous family.

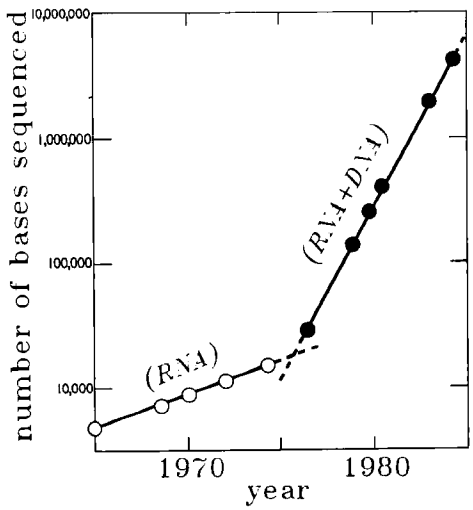


Figure 4.

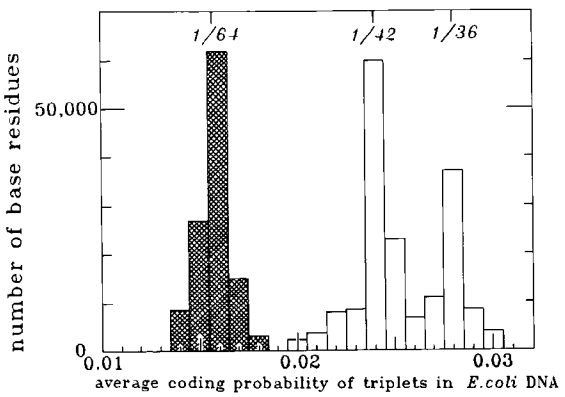


Figure 5.

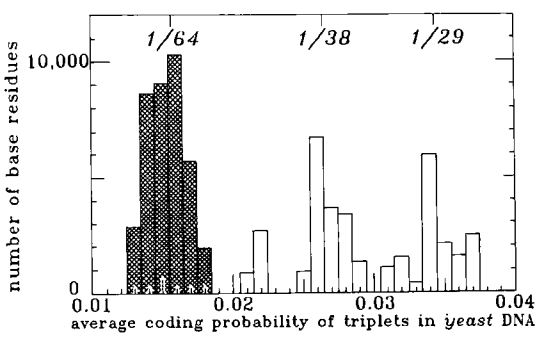


Figure 6.

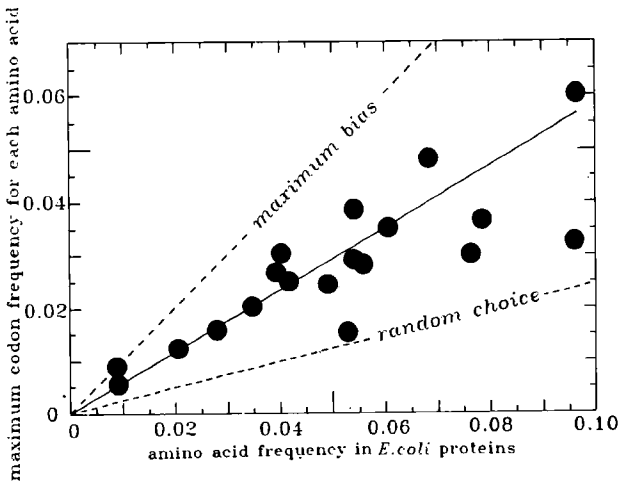


Figure 7.

Table 1.

Genetic Code and Synonymous Codon Usage

GENETIC CODE			SYNONYMOUS CODON USAGE (Percent)				
	amino acid	codon	E.coli	yeast	mouse	human	(expected)
1	alanine	GCA	22.7	11.2	19.2	14.1	25.0
		GCG	31.8	3.3	15.4	10.6	
		GCC	21.0	25.7	30.4	43.3	
		GCT	24.5	59.8	35.0	31.9	
2	arginine	AGA	3.8	72.0	28.8	28.0	16.7
		AGG	1.5	8.0	22.7	26.6	
		CGA	4.3	1.9	8.7	9.0	
		CGG	4.2	1.1	12.3	13.8	
		CGC	33.4	2.6	19.3	17.7	
		CGT	52.9	14.4	8.1	5.1	
3	asparagine	AAC	67.5	67.8	56.9	62.5	50.0
		AAT	32.5	32.2	43.1	37.5	
4	aspartic acid	GAC	50.5	47.3	53.7	61.2	50.0
		GAT	49.5	52.7	46.3	38.8	
5	cysteine	TGC	53.8	17.9	52.2	52.6	50.0
		TGT	46.2	82.1	47.8	47.4	
6	glutamine	CAA	27.6	86.1	22.8	26.1	50.0
		CAG	72.4	13.9	77.2	73.9	
7	glutamic acid	GAA	71.4	84.5	32.4	39.1	50.0
		GAG	28.6	15.5	67.7	60.9	
8	glycine	GGA	5.9	5.3	30.4	21.3	25.0
		GGG	8.8	4.3	20.9	21.3	
		GGC	39.3	7.8	32.5	40.8	
		GGT	46.0	82.7	16.3	16.7	
9	histidine	CAC	58.6	58.4	53.9	51.8	50.0
		CAT	41.4	41.6	46.1	48.2	
10	isoleucine	ATA	5.3	8.7	12.9	11.3	33.3
		ATC	57.7	40.5	47.2	61.8	
		ATT	37.0	50.8	39.9	26.9	
11	leucine	TTA	9.3	19.0	3.8	4.3	16.7
		TTG	9.6	57.7	9.9	11.6	
		CTA	2.1	8.2	5.8	5.5	
		CTG	62.3	5.3	45.5	48.1	
		CTC	8.1	2.4	22.9	22.7	
		CTT	8.7	7.3	12.2	7.8	
12	lysine	AAA	73.7	33.1	39.8	34.2	50.0
		AAG	26.3	66.9	60.2	65.9	
13	methionine	ATG	100.0	100.0	100.0	100.0	100.0
14	phenyl-alanine	TTC	57.6	62.6	60.8	60.9	50.0
		TTT	42.4	37.4	39.2	39.1	
15	proline	CCA	18.4	67.5	27.4	18.3	25.0
		CCG	60.9	3.0	13.3	11.3	
		CCG	8.1	7.3	21.7	37.4	
		CCT	12.6	22.2	37.6	33.0	
16	serine	AGC	23.3	4.6	21.3	30.5	16.7
		AGT	6.8	8.4	9.6	10.6	
		TCA	9.6	11.4	11.3	10.0	
		TCG	12.0	3.9	4.7	4.8	
		TCC	23.4	28.2	27.3	24.4	
		TCT	24.9	43.4	25.8	19.8	
17	threonine	ACA	8.8	16.3	21.1	21.9	25.0
		ACG	16.3	5.8	15.4	8.7	
		ACC	50.2	35.9	44.0	41.8	
		ACT	24.8	42.0	19.6	27.6	
18	tryptophan	TGG	100.0	100.0	100.0	100.0	100.0
19	tyrosine	TAC	55.8	67.1	62.5	59.1	50.0
		TAT	44.3	32.9	37.5	40.9	
20	valine	GTA	21.2	6.9	6.7	6.3	25.0
		GTG	27.2	8.8	54.8	51.8	
		GTC	14.4	36.2	22.2	27.4	
		GTT	37.3	48.2	16.4	14.5	
--	STOP	TAA	77.1	72.4	25.0	38.5	33.3
		TAG	2.1	17.2	3.9	20.5	
		TGA	20.8	10.3	71.2	41.0	

Table 2.

E.coli Sequences of Highest Average Codon Probability

	gene product	average codon probability	length,bp	approximate number in E.coli cell
*	ribosomal protein S1	0.030	1671	150,000
*	tufA,elongation factor Tu	0.030	1185	60,000
*	β' subunit, RNA polymerase	0.029	4224	7000
*	tufB, elongation factor Tu	0.029	1185	20,000
	recA, recombination-repair	0.029	1062	-
	α subunit, ATP synthetase	0.028	1542	-
	β subunit, ATP synthetase	0.028	1383	-
*	β subunit, RNA polymerase	0.028	3423	7000
*	ribosomal protein, L11/L1	0.028	1134	20,000
*	ribosomal protein, L7/L12	0.028	366	40,000
	outer membrane lipoprotein	0.027	237	720,000
*	ribosomal protein, L10	0.027	498	20,000
	outer membrane protein II	0.027	1041	10,000
	SSb,helix destabilizing protein	0.027	537	500
*	tryptophanyl-tRNA synthetase	0.026	1005	2000

* denotes involved in transcription/translation.

Table 3.

Yeast Sequences of Highest Average Codon Probability

	gene product	average codon probability	
*	enolase-I	0.037	(1/27)
*	enolase-II	0.037	
*	3-phosphoglycerate kinase	0.037	
	histone H2A-I	0.036	(1/28)
*	glyceraldehyde-3-P dehydrogenase-I	0.035	(1/29)
	ribosomal protein L3	0.035	
*	alcohol dehydrogenase-I	0.035	
*	pyruvate kinase	0.034	
	histone H2A-II	0.034	
*	glyceraldehyde-3-P dehydrogenase-III	0.034	
*	glyceraldehyde-3-P dehydrogenase-II	0.034	

* denotes involved in glycolysis/fermentation.

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In the Spring issue of EXPLORATIONS: The sure but silent force in American foreign policy in post World War II Japan—Harry F. Kern

At the close of World War II, the United States occupied Japan under the leadership of General Douglas MacArthur. Demilitarization and democritization were the initial occupation goals. While MacArthur clung to those goals throughout his tenure in Japan, a new force in foreign policy arose in the form of Harry F. Kern.

Foreign affairs editor of *Newsweek* at the war's conclusion, Kern was the pivotal member of a small pressure group which sought to turn U.S. policy in Japan away from the idealistic focus favored by MacArthur.

Who was this very powerful man with the very low profile?

Did he use the American press to change public attitudes toward postwar Japan?

What were his connections to the CIA? To the Secretary of State?

What were his motives for trying to create a favorable climate for foreign investment in Japan?

How did he see the *Communist threat* to Japan?

Read History Professor Howard Schonberger's acount of Harry F. Kern, the man virtually unknown to the public until he was named in 1979 by Grumman Aircraft as the individual responsible for a major political scandal in Japan.

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