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MIGRANT AND NONMIGRANT TOKELAUANS
I: DEFINING RESPONSE

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Abstract

A statistical procedure is derived to measure the response of a risk factor to environmental change in a population undergoing environmental transition. The response measure allows discrimination between changes of the risk factor attributable to alterations of established concomitants, and those attributable to novel features of the new environment. Hence, this procedure is especially suitable for studies which aim to partition out the genetic and environmental components of response to environmental change.

In this paper, we use this procedure to determine the magnitude of blood pressure (BP) response in Polynesians migrating from Tokelau to New Zealand. Migrant males exhibit positive BP response, but migrant females and nonmigrants show negative response. The accompanying paper defines the relationship between BP response and certain physiological and socio-cultural factors.

INTRODUCTION

While hypertension in adults is a well-established risk factor for cardiovascular disease, particularly coronary disease and stroke [1], the underlying causes of essential hypertension are still not understood. However, it is apparent that both genetic and environmental factors are important. Cross-cultural studies, in conjunction with epidemiological analyses within populations, indicate that, although a host of environmental factors are associated with the distribution of BP in both children [2] and adults [3], four factors probably predominate. These are: obesity, salt use, physical activity, and psycho-social stress. Evidence suggests that the major differences in BP levels between societies is largely attributable to differences in at least one of these four major risk factors.

Studies of populations in environmental transition provide one opportunity to progress beyond a merely correlational approach, especially when genotype environmental interactions are the issue of interest. A migrating population can provide a "quasi-experimental" situation, allowing detection of the causal role of interactions between the environment and other factors, [4,5,6], if the study is properly designed. In all migrant studies, there must be some assurance that the subset of the population exposed to the changing environment is an appropriately representative sample of the original population. Also, appropriate measures must be made of the relevant environmental variables through time.

Finally, there must be an appropriately defined measure of response to the changing environment. Simple measurements of the variable of interest in the

new environment may be uninformative. In many instances these values will be the result of changes in the distribution of the known concomitants in the new environment. For example, if the change in BP that occurs on exposure to a new environment is determined to be entirely due to changes in obesity, then there is little point in attempting to identify novel factors contributing to the change in BP per se. (The contribution of novel factors to changes in obesity may be of interest, but this is a conceptually different question.) Although this issue is an important one for genetic epidemiology, there have been few attempts to resolve the problem by devising an appropriate measure of "response" amenable to a meaningful analysis.

In this paper, we develop a straightforward statistical procedure that allows definition of "response" in any study design that embodies a longitudinal component. The response model partitions the factor of interest into a component that can be explained by known environmental concomitants, and a response measure that is essentially independent of known effects. The response variable can then be used to determine the effects of other factors, including both environmental and genetic traits.

This paper presents the basic methodology for calculating the response variable and then demonstrates the technique to estimate BP response in Tokelauans. In subsequent papers we identify the physiological and socio-cultural factors associated with response in Tokelauan adults [7] and children [8] for both migrants and nonmigrants.

Method

When populations experience environmental transition, the new environment has the potential to act on the entire set of intercorrelated variables that collectively define risk of disease. Hence the altered distribution of a single risk factor, such as BP, in the new environment, may arise in several ways. These include: a) change in the distribution of previously identified concomitants, including their possible absence in the new environment; b) introduction of a new set of causally important concomitants, which act independently of the previous set; c) interaction between old and new concomitants resulting in a changed relationship between the risk factor and the original concomitants. Components b) and c) may be considered to be the response of the risk factor to the new environment. Component a) is the response of the risk factors to the change in concomitants, which may, themselves, have responded to the new environment. Given such a complex situation, it is apparent that a simple comparison of risk in the two environments will not suffice to identify components of change. This is especially true when migrant and nonmigrant populations are compared, since the nonmigrant population may also have undergone changes. The model

developed below allows identification of these components of change in the new environment. While our argument assumes that migration is the cause of environmental transition, and that BP is the variable of interest, it should be emphasized that this technique is entirely general and can be used in a wide variety of situations.

The main requirement of the model is that the some measures of the distributions of the risk factor (for example, BP) and some relevant concomitants are available in both the original and new environments. When BP is the variable of interest, typical concomitants might be age, sex, weight, obesity, and serum cholesterol.

In both the pre- and postmigrant environments, the risk factor will depend on the known concomitants, x , as well as unknown concomitants and random variation in the population. In the premigrant environment, the relationship between initial blood pressure, BP_0 and the initial values of the concomitants, x_0 can be expressed as

$$BP_0 = \Phi(x_0) + \gamma_0 \quad (1)$$

where Φ is a function expressing the relationship between blood pressure and the concomitants in the premigrant environment and γ_0 is the component of BP which is not explained by the known concomitants. γ_0 consists of a deterministic component relating BP to the unknown concomitants, as well as a random component, unique to the individual.

Although other functional forms are possible, we will assume that Φ is linear. That is, we will assume that

$$BP_0 = x_0 \beta_0 + \gamma_0$$

where β_0 is the vector of coefficients in the premigrant environment. In this formulation, multiple linear regression on the premigrant data can be used to estimate β_0 . The residuals from the regression estimate γ_0 . The deterministic component of γ_0 is then orthogonal to the measured concomitants.

In the postmigrant environment, there may be a change in the association of BP with the concomitants, due to changes in unmeasured concomitants, including the introduction of new unmeasured risks, which are associated with

both BP and the concomitants. Letting BP_1 and x_1 denote, respectively, BP and the measured concomitants in the postmigrant environment, we can write the relationship between BP and the concomitants as

$$\begin{aligned} BP_1 &= \Omega(x_1) + \gamma_1 \\ &= \Phi(x_1) + \theta(x_1) + \gamma_1 \end{aligned} \tag{2}$$

where Ω represents the functional relationship between BP and the concomitants in the postmigrant environment, and $\theta = \Omega - \Phi$ represents the change in the functional relationship. (If Ω and Φ are both linear, then θ represents a change in the regression coefficients.) γ_1 represents the component of BP not explained by the concomitants in the new environment.

This model expresses the three distinct sources of changes in the risk factor in the postmigrant environment. One source is the possibility of change in the values of the known concomitants. The second component is the change in the functional relationship between the known concomitants and the risk factor. The third source is the change in values or introduction of unknown concomitants.

The second and third components are what we have defined as the response of the risk to the new environment. It is the component of the risk factor that cannot be predicted from Φ and x_1 . From (1) and (2) we define the response, ρ , as

$$\begin{aligned} \rho &= BP_1 - \Phi(x_1) \\ &= \theta(x_1) + \gamma_1 \end{aligned}$$

When the risk factor and concomitants have been measured on the same individuals in the pre- and postmigrant environments, a longitudinal analysis is possible. In this case we can combine (1) and (2) to obtain

$$\begin{aligned} \Delta BP &= BP_1 - BP_0 \\ &= [\Phi(x_1) + \theta(x_1) + \gamma_1] - [\Phi(x_0) + \gamma_0] \end{aligned}$$

where Δ denotes the change from the pre- to the postmigrant environment.

When Φ is assumed to be linear,

$$\Delta BP = \Phi(\Delta x) + \theta(x_1) + \Delta \gamma$$

In this case, Φ can be estimated by regressing ΔBP on Δx . Call this estimate Φ_L .

The residuals from this regression are the longitudinal response ρ_L :

$$\begin{aligned}\rho_L &= \Delta BP - \Phi_L(\Delta x) \\ &= \theta_L(x_1) + \Delta \gamma_L\end{aligned}\tag{3}$$

Another estimate of response can be obtained from a longitudinal study using the data from each environment separately. The premigrant data can be used to estimate Φ and γ_0 . Call these estimates Φ_c and γ_{0c} . The cross-sectional response, ρ_c is then defined by

$$\begin{aligned}\rho_c &= BP_1 - \theta_c(x_1) - \gamma_{0c} \\ &= \theta_c(x_1) + \Delta \gamma_c\end{aligned}$$

Finally, if two cross-sectional samples are available, a somewhat less precise estimate of response can be derived:

$$\begin{aligned}\rho_c &= BP_1 - \theta_c(x_1) \\ &= \theta_c(x_1) + \gamma_{1c}\end{aligned}\tag{4}$$

where γ_{1c} is the unexplained component in the post-migrant environment.

Study Design

The Tokelau Island Migrant Study (TIMS) was initiated in 1967/68 to study the effects of migration to New Zealand on the prevalence of hypertension and other risk factors for cardiovascular disease in the Tokelau population. Data was collected from the atoll of Fakaofu in 1968 and from Nukunonu and Atafu in 1971 [9]. The data from these initial surveys essentially define the "pre-migrant" population, even though some migration has already occurred. One important result of the pre-migrant survey was the demonstration that individuals who would subsequently migrate and those who would not were similar with respect to the values of cardiovascular risk variables [10] and other disease conditions [11]. Follow-up data was collected in Tokelau in 1976 (the non-migrant population) and in New Zealand in 1975 and 1977 (the migrant population) [9,10]. A second follow-up was done in 1980/81 in New Zealand, and 1981/82 in Tokelau. Data from the second follow-up is not used in this paper.

Tokelau society now comprises approximately 4000 individuals split into two components: the migrant population, which comprises about 60% of the total, is distributed mainly in three centres of the North Island of New Zealand. The

remaining nonmigrant component is distributed in the three homeland atolls, with Fakaofu having the largest population (42% of the nonmigrants), Nukunonu the smallest (24%), and Atafu the remainder (34%) [4].

It has already been shown [5] that BP varies significantly among the three survey groups. As well, Ward et al [4] have shown that, in the premigrant population, there are distinct juvenile and adult phases in the distribution of BP, height and weight with age. For the cross-sectional study we have used only people who are in the adult phase for all three variables, as defined by the premigrant analysis; that is, those 18 years and older. Due to the smaller sample size in the longitudinal study, persons between 14 and 18 years of age at the premigrant time period, who had not grown taller by the postmigrant time period, were also included. Pregnant women and persons with discrepant measures between two surveys were excluded from the sample. Table 1 summarizes the number of people used in the analysis for each time period.

In this paper, we define BP response in terms of the relationship of the concomitants, atoll of origin, sex, height, weight, fat index, body mass index (weight/height²) serum cholesterol, and age (expressed as a third degree polynomial), in the premigrant population. The distribution of BP response identifies changes in the relationship between BP and the concomitants in the nonmigrant and migrant populations. In the accompanying paper, we explore the role of various physiological and socio-cultural variables in mediating these changes.

Except for the all subsets regressions, which were done with BMDP [14], all analyses in this paper were done using the MIDAS [15] statistical package.

Description of Variables

The response model divides BP in the changed environment into two components, the component associated with the change in distribution of the concomitants, and the response, which is independent of the change in the concomitants. If we hope to determine causes for the changed distribution of BP in the new environment, we must investigate both the response variable, and the distribution of the concomitants in this environment.

Figure 1 summarizes the distribution of BP by age and sex in the three populations. Except for the youngest group of women, migrants have the highest BP in all sex-age groups. Nonmigrants generally have the lowest BP. Both SBP and DBP generally increase with age.

Figure 2 summarizes the distribution of the concomitants with age for each of the survey populations. For all variables but cholesterol, there were no

significant differences between premigrants and non-migrants in any sex-age groups, and the plots have been combined into a single curve.

The obesity indices all have an "inverted U" shape with lower values for the young and old. Older adults tend to be shorter than young ones and to have higher serum cholesterol. It is interesting to note that the premigrant population exhibits the highest levels of serum cholesterol.

Results

Longitudinal analysis

The response model provides an assessment of the contribution of the new factors to the distribution of BP in the new environment. This component, which may include both genetic and environmental influences, can be identified for all individuals measured in the new environment, even if baseline data from the original environment is missing for some. If longitudinal data is available for all individuals in a study, the change in the risk factor of interest, appropriately standardized for changes in the identified concomitants, is a natural measure of response to environmental change. In either case, an estimate of response to the new environment is obtained, which is independent of the effects of change in distribution of the original concomitants.

The Tokelau data offers an opportunity to test the validity of the response model. Although the TIMS data sets are cross-sectional samples, they contain baseline data for a sizable portion of both the nonmigrant and migrant populations. We can use this subset to test the adequacy of the response model by comparing the results of longitudinal and cross-sectional analyses, for individuals measured at both time periods. If the response model is valid, the two estimates should be close in value.

In the conventional longitudinal model, change in BP is related to change in concomitants and baseline values. In the longitudinal response model, change of BP is related to change in the concomitants and postmigrant values. Clearly the two models are equivalent in their predictive power. The strength of the response model is that changes in BP can be separated into those caused by changes in the concomitants, and those caused by new influences in the new environment.

The conventional longitudinal analysis is summarized in Table 2. The change in BP was regressed on baseline BP, baseline concomitants, and change in the concomitants. Stepwise regression was used to select a subset of the variables which provided an adequate fit. The selected variables were then added sequentially to the regression equation. The sequential contributions to R^2 are listed in Table 2.

The final regression equations had R^2 ranging from 27% to 41%. For both migrants and nonmigrants, initial BP, fatness and age were the most important factors.

The first step in computing the longitudinal response variable is to estimate the baseline regression equation, Φ , by regressing change in BP on the change in concomitants. The results of this regression are summarized in Table 3. The longitudinal response is the residual from this regression.

For nonmigrants, change in age and fatness were the most important predictors. However, the regression equations had little predictive power, despite their statistical significance. Among migrants, change in age, fatness and serum cholesterol were significant. Interestingly enough, although there was a strong relationship between change in SBP and change in concomitants, for male migrants no significant relationship existed for change in DBP.

A second estimate of change in response was determined for this group of individuals from the cross-sectional analysis.

Cross-Sectional Analysis

For the cross-sectional analysis, the full set of data was used, including individuals for whom either baseline or follow-up data was missing. The premigrant regression equation $\Phi(x_0)$ was estimated by regressing the premigrant BP on the full set of concomitants. Stepwise regression was then used to reduce the concomitants to a smaller set, containing all significant terms. The contributions to R^2 in the order entered is summarized in Table 4. Response is estimated by substituting the estimate of Φ into equation 4.

The estimates of the coefficients of Φ obtained this way are simpler than those obtained from the longitudinal analysis. For SBP there are no sex or atoll differences. SBP decreases with age and increases with body mass. For DBP, there are minor sex and atoll differences. DBP increases with age and with body mass.

Estimating the Relationship Between Response and the Concomitants

The relationship between response and the original concomitants in the postmigrant environment is expressed by the function θ . To obtain an estimate of θ from the longitudinal data, the longitudinal responses were regressed on postmigration values of the concomitants. (For male DBP, the "response" was

the same as raw BP.) Table 5 summarizes the significant contributions to R^2 , partialled in the order given. The R^2 values are small. The major association, for migrant men, and for nonmigrant women, was with weight. Age was significant for nonmigrant men.

To obtain an estimate of θ from the cross-sectional data, we regressed the nonmigrant and migrant cross-sectional responses on the concomitants. For ease of interpretation, men and women were analyzed separately, and all subsets regression was used to obtain a reduced set of concomitants which was the same for all populations. Table 6 summarizes the significant contributions to R^2 , partialled in the order given. The overall R^2 are small, but significant, for all groups but nonmigrant women. The major contributions are made by obesity, age and atoll differences.

Discussion

The "response" model has been proposed as a means of recovering longitudinal information from a population that has been sampled cross-sectionally at several points in time. The response model has been used to examine the changes that occur when a single homogeneous population splits into two contingents, one remaining in the home environment, the other migrating to a radically different environment. In this case, the "response" indicates how the relationship between BP and a set of concomitants has changed in each sub-population. If a single population has been sampled repeatedly over time, the response model can be used to trace the change in relationship between a set of dependent variables and a set of concomitants in successive time periods.

To test the effectiveness of the response model, the estimates of longitudinal response obtained from the cross-sectional data were compared to those obtained from the longitudinal data. Scatter plots and t-tests showed no significant differences. The correlations between the cross-sectionally and longitudinally derived variables are displayed in Table 7. The values range from .77 to .99 and most are over .95. This excellent degree of correspondence displays the efficacy of the response variable approach for mixed cross-sectional and longitudinal data.

It should be noted that the F-test for significance of the regression of response on the concomitants is only approximate. Two important violations occur. First, the residuals have deterministic as well as a random component. This inflates the residual sums of squares by the addition of a squared bias component. As well, the error components of different measurements are correlated, longitudinally, because the same individuals are measured in different time periods, and cross-sectionally, because of familial correlations. In general the bias effects will be larger than the correlation effects, leading to conservative tests.

Such violations of assumptions affect all statistical procedures commonly used with epidemiological data.

Although we have used a linear model to determine response, the response model approach may be used with any appropriate functional form for Φ , which defines the relationship between the risk factor and concomitants in the original environment. A non-zero value of θ , which measures the change of relationship in the new environment, may be interpreted as an interaction between the original concomitants and some unmeasured concomitants. However, if the distributions of the concomitants differ widely between the home and postmigration environments, a non-zero estimate of θ , may also arise from an incorrect specification of Φ in the model. For example, if a linear function for Φ was specified when a nonlinear form would be more appropriate, θ would estimate the departure from linearity when the values of the concomitants change. The only concomitant associated with response in the Tokelau data is obesity. The magnitude of change in obesity makes a nonlinear effect unlikely, so the association is most likely due to interaction.

Overall, for the Tokelau populations, both response and change in BP are negative for nonmigrants, and positive for migrant men. For migrant women DBP shows little change or response, and SBP shows positive change but negative response. Both the longitudinal and cross-sectional analyses reveal atoll differences in the premigration associations of BP with the concomitants, and in response to the changing environment. Although the magnitude of association is smaller than with the raw values, the concomitants are associated with response in the follow-up time period, indicating either a non-linear relationship between BP and the concomitants, or, more likely, interaction with other, unidentified concomitants in the changed environment. In the following paper [7] the association between response and a set of physiological and socio-cultural factors is explored. These associations explain some of the observed patterns of response.

The response model yields highly consistent results between longitudinal and cross-sectional studies of the Tokelau populations, and reveals some interesting patterns of BP change. We interpret the estimate of BP response as a measure of that component of BP in the new environment which is not simply due to the changed distributions of concomitants identified in the original environment.

References

1. G. Onesti and C. R. K. (ed.), in *Hypertension: Determinants Complications and Intervention*, Grune and Stratton, NY, 1979.
2. M. Szklo, Epidemiological patterns of blood pressure in children, *Epidemiological Reviews* 1, (1979), pp143-169.
3. J. Stamper, Improved Life Styles: Their Potential for the Primary Prevention of Atherosclerosis and Hypertension in Childhood, in *Childhood Prevention of Atherosclerosis and Hypertension*, R. M. Lauer and R. B. Shekelle (ed.), Raven Press, NY, 1980, 3-36.
4. R. H. Ward, P. G. Chin and I. A. M. Prior, Genetic Epidemiology of BP in a Migrating Isolate: Prospectus, in *Genetic Analysis of Common Diseases: Applications to Predictive Factors in Coronary Disease*, C. F. Sing and M. Skolnick (ed.), Alan Liss Inc, NY, 1979, 675-710.
5. R. H. Ward, P. G. Chin and I. A. M. Prior, Effect of Migration on the Familial Aggregation of Blood Pressure, *Supp 1 Hypertension* 2, 4 (1980), I-43 - I-54.
6. R. H. Ward, Genetic Epidemiology in an anthropological context, *Medical Anthropology* 4, (1980), 293-306.
7. N. S. Altman, R. H. Ward and I. A. M. Prior, Patterns of blood pressure response in migrant and nonmigrant Tokelauans II: Physiological and socio-cultural correlates of BP response, Biometrics Unit Technical Report BU-995-M, Cornell University, (1990)..
8. N. S. Altman, R. H. Ward and I. A. M. Prior, Patterns of blood pressure response in migrant and nonmigrant Tokelauans III: response in Tokelauan children, Biometrics Unit Technical Report BU-996-M, Cornell University, (1990).
9. I. Prior, Migration and Physical Illness, *Advances in Psychosomatic Medicine* 9, (1977), 105-131.
10. I. A. M. Prior, J. M. Stanhope, J. G. Evans and C. E. Salmond, The Tokelau Island Migrant Study, *International Journal of Epidemiology* 3, (1974), pp225-232.
11. J. M. Stanhope and I. A. M. Prior, The Tokelau Island Migrant Study: Prevalence of Various Conditions Before Migration, *International Journal*

of Epidemiology 5, (1976), pp259-266.

12. I. A. M. Prior, A. Hooper, J. W. Huntsman, J. M. Stanhope and C. E. Salmond, The Tokelau Island Migrant Study, in *Population Structure and Human Variation*, G. A. Harrison (ed.), Cambridge University Press, Cambridge, 1977, 165.
13. R. H. Ward, P. D. Raspe, M. E. Ramirez, R. L. Kirk and I. A. M. Prior, Genetic Structure and Epidemiology: The Tokelau Study, in *Population Genetic Studies on Isolates*, A. W. Eriksson, A. Forsius and H. Nevalinna (ed.), Academic Press, London, 1979.
14. BMDP-79: *Biomedical Computer Programs P-Series*, W. J. Dixon and M. B. Brown (ed.), University of California Press, Berkeley, 1979.
15. D. J. Fox and K. E. Guire, *Documentation for MIDAS*, Statistical Research Laboratory, University of Michigan, 1976.

FIGURES AND TABLES

TABLE 1
DISTRIBUTION OF SAMPLE SIZE IN THE VARIOUS SURVEYS

TYPE OF DATA	CROSS-SECTIONAL			LONGITUDINAL	
	Survey	Premigrant	Nonmigrant	Migrant	Nonmigrant
Female	488	381	443	301	153
Male	374	271	534	208	112

TABLE 2
LONGITUDINAL ANALYSIS OF BP CHANGE:
R² VALUES (IN PERCENTAGE) DERIVED FROM STEPWISE REGRESSION.
VARIABLES ENTERED IN THE ORDER GIVEN.

	INITIAL BP	CHANGE IN AGE ^f	CHANGE WEIGHT ^a	CHANGE HEIGHT	INITIAL AGE	INITIAL AGE ³	INITIAL FAT INDEX	INITIAL BODY MASS ^a	TOTAL
MALE									
Nonmigrant SBP	18.9*	0.9	3.1*	2.8**	0.1	0.8	0.3	1.9	28.8**
Nonmigrant DBP	21.2**	0.2	3.0**		0.0	6.2**	1.1	0.2	31.9**
Migrant SBP	1.2	12.8**	3.8	0.6	13.0**	2.9	2.8	3.1	40.2**
Migrant DBP	9.7*	4.5	2.7		4.9*	0.0	3.0	14.3**	39.1**
FEMALE									
Nonmigrant SBP	4.6**	0.1	6.0**	0.4	6.5**	0.0	0.8	8.9**	27.3**
Nonmigrant DBP	10.4**	0.0	8.5**		4.7**	2.9**	0.0	8.1**	34.6**
Migrant SBP	2.3	3.5*	7.6	0.2	21.0*	0.6	0.1	5.9**	41.2**
Migrant DBP	10.5**	0.9	10.0**		15.1**	0.0	0.7	4.3*	41.5**

* .01 < p < .5
 ** p < .01
 f Fakaofu only (not significant for other atolls)
 a Includes atoll interaction terms

TABLE 3
CONTRIBUTION TO R² (IN PERCENTAGE) OF THE CHANGE IN BP BY THE CHANGE IN CONCOMITANTS.
VARIABLES ENTERED IN THE ORDER GIVEN.

	ATOLL	HEIGHT	WEIGHT	BODY MASS	FAT INDEX	CHOLESTROL.	AGE	AGE ²	AGE ³	TOTAL
MALE										
Nonmigrant SBP		1.6n	4.6**f							6.2
Nonmigrant DBP				4.4**f				4.8**	0.9#	10.1**
Migrant SBP	18.0**f	0.9	0.9		4.5*#f		13.1**p	14.4**		51.8**
FEMALE										
Nonmigrant SBP				9.2**			0.6n		1.7*f	11.5**
Nonmigrant DBP				1.37**				1.8*f		14.8**
Migrant SBP				7.7**		11.5**			8.2**	27.4**
Migrant DBP			1.8f		7.7**	6.3*p	7.3**		7.6**	30.7**

* .01 < p < .5
 ** p < .01
 f Fakaofu only (not significant for other atolls)
 p Fakaofu negative, other atolls positive
 n Nukunonu only
 # negative

TABLE 4
ESTIMATE OF ADDITIONAL CONTRIBUTIONS TO R² (IN PERCENTAGE) OF THE CONCOMITANTS IN THE LONGITUDINAL
DATA SET ONCE THE BASELINE RELATIONSHIP HAS BEEN ACCOUNTED FOR.
VARIABLES ENTERED IN THE ORDER GIVEN.

	WEIGHT	BODY MASS	AGE ³	TOTAL
MALE				
Nonmigrant SBP	.02	.01	3.2*	3.5
Nonmigrant DBP	0.0	0.0	0.0	0.0
<hr/>				
Migrant SBP	2.8	0.7	0.0	3.5
Migrant DBP	9.6**	0.7	0.1	10.4**
<hr/>				
FEMALE				
Nonmigrant SBP	3.2**	0.4	0.4	4.0*
Nonmigrant DBP	2.1*	0.0	0.1	2.2
<hr/>				
Migrant SBP	0.2	0.0	1.4	1.6
Migrant DBP	0.0	0.0	0.0	0.0

* .01 < p < .5

** p < .01

TABLE 5
CONTRIBUTION TO R² OF BP (IN PERCENTAGE) BY THE CONCOMITANTS IN THE ORIGINAL ENVIRONMENT.
VARIABLES WERE ENTERED IN THE ORDER INDICATED.

	BODY MASS	FAT INDEX	AGE	AGE ²	AGE ³	TOTAL
SBP	12.1**		23.8**	4.2**	0.7**	40.8**
DBP	17.3**	1.8**f		10.5**	0.4*n	30.0**

* .01 < p < .5
 ** p < .01

f Fakaofu only
 n Nukunonu only

TABLE 6

ESTIMATE OF ADDITIONAL CONTRIBUTIONS TO R^2 (IN PERCENTAGE) OF THE CONCOMITANTS FOR THE POSTMIGRANTS IN THE CROSS-SECTIONAL DATA SET ONCE THE BASELINE RELATIONSHIP DERIVED FROM THE PREMIGRANT DATA HAS BEEN ACCOUNTED FOR. VARIABLES WERE ENTERED IN THE ORDER INDICATED.

	WEIGHT	BODY MASS	CHOLESTROL.	ATOLL* AGE	AGE ²	AGE ³	TOTAL
MALE							
Nonmigrant SBP	0.3	6.8**	1.1	12.0**	3.2**	1.8*	25.2**
Nonmigrant DBP	0.4	2.9**	2.4*	3.0*	4.9**	0.4	14.0**
Migrant SBP	0.0	2.9**	0.7	2.3**	3.5**	2.9**	12.3**
Migrant DBP	0.2	0.1	0.9*	1.4**	2.3**	2.2**	7.1**
FEMALE							
Nonmigrant SBP	0.2	0.0	0.0	2.2**	0.0	0.1	2.5
Nonmigrant DBP	0.7	0.1	0.1	1.1	0.1	1.4*	3.5
Migrant SBP	1.6**	0.2	3.5**	6.0**	2.0**	0.1	13.4**
Migrant DBP	1.3*	0.2	0.4	2.1**	2.8**	0.2	7.0**

* .01 < p < .5

** p < .01

TABLE 7
CORRELATION BETWEEN LONGITUDINAL AND CROSS-SECTIONAL RESPONSE

	NONMIGRANT	MIGRANT
SBP		
Male	.96	.79
Female	.99	.94
DBP		
Male	.96	.96
Female	.98	.86

Fig. 1. Plots of blood pressure with age.

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Fig. 2. Plots of concomitants with age. There is no significant difference in distribution between premigrants and nonmigrants for any variable but cholesterol. As a result, the lines have been combined.

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