close examination of the sections showed that only the motor nucleus of the 7th nerve was filled, and no portion of the motor nucleus of trigeminal was filled - thus indicating that the 7th nerve was the only nerve filled with cobalt.

In the mouse, the mesencephalic nucleus of trigeminal is located in the mesencephalon at the level of the oculomotor nucleus and lateral to the 3rd ventricle. As a result of filling of the facial nerve in the mouse, scattered cells within the ipsilateral mesencephalic nucleus were identified as giving rise to afferent fibres which enter the facial nerve (figure 2). The fibres course ventrally from the nucleus and pass medial to the lateral lemniscus on the ipsilateral side of the brain. Passing caudally through the brainstem, the fibres pass lateral to nucleus locus coeruleus and lateral to the motor nucleus of trigeminal. Further caudally they exit the brainstem with the motor fibres of the facial nerve.

To insure that only the afferent fibres from the facial muscles of the mouse were filled, the 7th nerve was isolated at its exit from the skull at the stylomastoid foramen, and filled with cobalt at this point. Therefore, the labelling of the cells within the mesencephalic nucleus of trigeminal are from afferent fibres which probably arise from the facial musculature.

We have been able to demonstrate that the facial nerve, in at least 2 species, contains an afferent component which arises from the ipsilateral mesencephalic nucleus of the trigeminal complex, and that the fibres pass caudally through the brainstem to exit with the motor branch of the facial nerve. It is quite possible that this afferent component may indeed be the proprioceptive fibres of the facial nerve.

1 This work was supported by National Aeronautics and Space Administration Grant No. NAS 9-15213.
2 R.D. Smith and H.Q. Marcarian, J. dent. Res. *47*, 616 (1968).
3 J.J. Baumel, Archs. Otolar. *99*, 34 (1974).
4 A. Tokunaga, M. Oka, T. Murao, H. Yokoi, T. Okumura, T. Hirata, Y. Miyshita and S. Yoshitatsu, Med. J. Osaka Univ. *9*, 397 (1958).
5 N. Iwata, S.T. Kitai and S. Olson, Brain Res. *43*, 662 (1972).
6 D.J. Prior and P.M. Fuller, Brain Res. *64*, 472 (1973).
7 P.M. Fuller and D.J. Prior, Brain Res. *88*, 211 (1975).
8 M.R. Martin and C.A. Mason, Brain Res. *121*, 21 (1974).
9 N.M. Tyrer and E.M. Bell, Brain Res. *73*, 151 (1974).
10 E. Weinberg, J. comp. Neurol. *46*, 249 (1928).
11 K. Rubinson, Brain Res. *19*, 3 (1970).

Coffee and cola beverage consumption as heart disease risk factors in men[1,2]

Marge F. Stanton, R.A. Ahrens and L.W. Douglass

Food, Nutrition and Institution Administration Department, College of Human Ecology and Dairy Science Department, College of Agriculture, University of Maryland, College Park (Maryland 20742, USA), 26 January 1978

Summary. The relationship of coffee drinking and cola beverage consumption to recognized heart disease risk factors was studied in 365 adult men. Cola beverage consumption was not positively related to any risk factor. Heavy coffee drinking seemed to reflect a life style characterized by infrequent eating, which was significantly correlated to all the heart disease risk factors studied.

The possible involvement of coffee drinking as a risk factor in heart disease has been controversial. In 1973, the 'Boston collaborative drug surveillance program' reported that patients hospitalized following a heart attack had drunk more coffee prior to hospitalization than patients hospitalized for other reasons[3]. Their analysis showed that this effect of coffee was independent of cigarette smoking. Analysis of the Framingham[4] and Kaiser-Permanente[5] epidemiological studies failed to find any relationship between coffee drinking and heart disease death rate, except for that connected with associated cigarette smoking. Maugh has suggested that all 3 studies are invalid because they failed to take into account the consumption of cola beverages, which supply more caffein to many people than does coffee[6]. The present study specifically measured cola beverage consumption in men, 17-70 years of age, and compared its association with known heart disease risk factors to that of coffee drinking.

Methods and materials. Records were obtained from men who attended a 'heart screening' project of the Heart Association of Southern Maryland. Of these, only complete records from men who were taking no prescription medication were analyzed. That left 365 subjects, aged 17-70 years, who completed a medical history and a dietary questionnaire. Screening personnel recorded sex, age, height, weight and measured blood pressure by arm cuff and sphygmomanometer. A 12-h fasting blood sample was analyzed for serum cholesterol[7] and triglyceride[8] levels. The dietary questionnaire was a 7-day recall that attempted to get an estimate of average weekly consumption by the subject. The short method of dietary analysis[9] was used to judge serving sizes and total food intakes, which were then adjusted to a daily basis. Body fatness was estimated by calculating the ponderal index (height divided by cube root of weight) of each subject. Subjects with the smallest ponderal index tended to be most obese. Frequency of eating was estimated by adding the meals and snacks reported by each subject and dividing by 7 to get a daily average. Smoking refers to cigarette smoking. Several biomedical data description and step-up linear regression computer programs were used to select variables that were closely related to one another.

Results and discussion. The correlation coefficients of selected risk factors (cigarette smoking, body fatness, blood pressure, serum cholesterol and triglyceride levels) with selected dietary components are presented in the table. Cola beverage consumption was not positively correlated with any of the heart disease risk factors measured. There were significant negative correlations with blood pressure and body fatness ($p < 0.05$). However, it seems probable that this is a reflection of the strong negative correlation ($r = -0.29^{**}$) between age and cola beverage consumption. Coffee drinking, on the other hand, was positively correlated with age ($r = 0.14^{**}$), cigarette smoking ($r = 0.32^{**}$) serum cholesterol ($r = 0.18^{**}$) and triglyceride ($r = 0.15^{**}$) levels. This would tend to indicate that any correlation between coffee drinking and recognized heart disease risk factors is unrelated to the caffein content of the coffee. Rather, the relationship of coffee drinking to heart disease risk factors seems to be indirect and a reflection of the life style associated with coffee drinking. To that extent, our findings confirm the earlier reports from the Framingham[4] and Kaiser-Permanente[5] epidemiological studies that any effect of coffee drinking on heart disease risk

Simple correlation matrix for variables descriptive of eating pattern, food intake, and heart disease risk factors in Maryland men

Variable identification	Sys	Dias	Chol	Tri	Cal	Fat	Suc	Alc	Smo	PI	Cof	Col	E.F.
Age	0.26[b]	0.17[b]	0.36[b]	0.15[b]	−0.27[b]	−0.05	−0.20[b]	0.00	0.07	−0.22[b]	0.14[b]	−0.29[b]	−0.06
Systolic blood pressure (Sys)		0.57[b]	0.19[b]	0.14[b]	−0.18[b]	0.00	−0.10[a]	0.14[b]	0.02	−0.28[b]	0.05	−0.12[a]	−0.22[b]
Diastolic blood pressure (Dias)			0.12[a]	0.07	−0.15[b]	0.02	−0.07	0.14[b]	0.02	−0.24[b]	0.04	−0.11[a]	−0.16[b]
Cholesterol level (Chol)				0.45[b]	−0.08	0.00	−0.06	0.08	0.16[b]	−0.23[b]	0.18[b]	−0.03	−0.14[b]
Triglyceride level (Tri)					−0.06	−0.08	−0.01	0.10[a]	0.19[b]	−0.22[b]	0.15[b]	−0.02	−0.12[a]
Caloric intake (Cal)						−0.06	0.26[b]	−0.18[b]	0.03	0.22[b]	0.03	0.53[b]	0.15[b]
% Fat calories (Fat)							−0.60[b]	−0.06	0.06	0.05	0.07	−0.38[b]	0.03
% Sucrose calories (Suc)								−0.25[b]	0.00	0.06	−0.05	0.78[b]	0.01
% Alcohol calories (Alc)									0.14[b]	−0.07	0.11[a]	−0.12[a]	−0.17[b]
Smoking (Smo)										−0.06	0.32[b]	0.10[a]	−0.19[b]
Ponderal index (PI)											−0.07	0.11[a]	0.12[a]
Coffee drinking (Cof)												−0.03	−0.11[a]
Cola drinking (Col)													0.05
Eating frequency (E.F.)													

Critical correlation coefficients. [a] $r = \pm 0.10$ ($p < 0.05$); [b] $r = \pm 0.14$ ($p < 0.01$).

involves a 3rd factor. Coffee drinking seems to act as an index of life style. Central to this life style seems to be infrequent eating. Infrequent eating correlated significantly with all of the heart disease risk factors studied. Moore et al.[10] have taken this relationship 1 step further and found raised coronary lesions in deceased persons who practiced an earlier life style that included infrequent eating.

1 This investigation was supported in part by the Computer Science Center of the University of Maryland.
2 An acknowledgment is due Michael W. Delaney and the Heart Association of Southern Maryland for providing the original data on which this analysis is based.

3 Boston Collaborative Drug Surveillance Program, Lancet *2*, 1278 (1973).
4 T.R. Dawber, W.B. Kannel and T. Gordon, N. Engl. J. Med. *291*, 871 (1974).
5 A.L. Klatsky, G.D. Friedman and A.B. Siegelaub, J. Am. Med. Ass. *226*, 540 (1973).
6 T.H. Maugh, Am. Heart J. *88*, 672 (1974).
7 G.J. Koval, J. Lipid Res. *2*, 419 (1961).
8 J.L. Geigel, A.B. Ham and W. Clema, Clin. Chem. *21*, 1575 (1975).
9 Vanderbilt University Hospital, Diet Manual, Vanderbilt University Press *2*, 112 (1961).
10 M.C. Moore, M.A. Guzman, P.E. Schilling and J.P. Strong, J. Am. diet. Ass. *67*, 22 (1975).

Evidence for cross bridge slippage in a stretched muscle fibre[1]

K. Güth, H.J. Kuhn, J.W. Herzig and J.C. Rüegg

Department of Physiology II, University of Heidelberg, Im Neuenheimer Feld 326, D-6900 Heidelberg (Federal Republic of Germany), 16 December 1977

Summary. Bundles of glycerol-extracted psoas fibres which were contracted by immersion in a saline containing 15 mM MgATP and 12 µM free Ca^{++} were subjected to up to 3 stretches (rise time 0.8 msec) each of amplitude 1% L_i at intervals of 10 msec. The elastic tension responses to these stretches were all of comparable size and the peak tensions reached during the stretches were in each case followed by a rapid tension decline almost to the tension values before the stretches. This indicates that stretch-induced detachment and reattachment of cross bridges to the actin filament occurred within 10 msec (slippage).

The Huxley and Simmons model (1971)[2] for muscle contraction attributes the quick phase of the tension transient, following a quick length change, to rotation of acute angled cross bridges into a perpendicular position[3,4]. The elastic elements of these rotated cross bridges are thereby discharged, bringing about a decline in tension. The Huxley and Simmons model[2] further postulates that, once a cross bridge is attached, it remains attached for at least several tens of msec, i.e. no appreciable attachment or detachment of cross bridges can take place within the quick phase.

According to Huxley[5] and Podolsky[6] on the other hand, rapid attachment and detachment processes may take place during the quick tension transients.

Major criteria for deciding between such models are provided by the analysis of force transients. In order to investigate whether fast attachment and detachment processes take place during the quick phase or not, we performed double stretch and triple stretch experiments.

Methods. A bundle of 5 glycerol-extracted[7] rabbit psoas fibres was bent as a loop around the pin of a RCA-5734 force transducer (resonance frequency 2 kHz), and the 2 free ends were glued to a glass rod attached to a servo controlled Ling Dynamics type 101 Vibrator[8]. Length steps performed within 0.8 msec were recorded by field plates. The preparation was first immersed in relaxing solution containing: 15 mM ATP, 15 mM $MgCl_2$, 20 mM imidazole 4 mM EGTA, pH 6.7, estimated free Ca^{++}-concentration of $< 10^{-8}$ M. The bundle was subsequently immersed in contraction solution. This was identical with the relaxing solution except that EGTA was replaced by 4 mM CaEGTA (Ca^{++}-concentration 1.2×10^{-5} M).

Results. In the contracting state (isometric tension 4.5 mN), 3 stretches and a subsequent release were performed. Each length change had an amplitude of 1% L_i of the initial length of the fibre. The corresponding force transient is shown in the figure.