1982). The fatty acid pattern of RBCs in post MI of Black Africans is very similar to a normal urban black control group. Only 20:5 w-3 and 22:5 w-3 demonstrated a small but significant difference between the two groups and they were decreased in the patients. It is not likely that the patients altered their diet following the MI because no patient professed to receiving medical counseling in this regard, and in the black community, awareness of risk factors is very low. In healthy white populations with differing diets, the saturated and monounsaturated fatty acid content of RBCs and platelets mirror variations in dietary fat, while PUFA levels in platelets tend to resist change by dietary intervention (Dougherty et al., 1987). The finding of high levels of saturated fatty acids in platelets but not RBCs is rather difficult to evaluate given the lack of comparative data in this ethnic population. The fatty acid pattern of platelets however, may be effected by a number of factors which are independent of diet. Among these are hormonal control of endogenous fatty acid synthesis and desaturation (Brenner 1982), activation of fatty acid utilizing enzymes (deBoer et al., 1982), and affinity for uptake of exogenous fatty acids (Neufeld et al., 1983).

The findings of significantly decreased levels of 20:5 w-3 in both platelets, plasma and RBCs indicate

that this may be an important risk factor determinant in this and other populations (Lea et al., 1982; Aursnes et al., 1986). Variations of omega-3 fatty acids in RBCs suggests that this is dietary related. A study conducted over 10 years ago however, determined that South African urban Blacks consumed very little fish due to lack of electricity for refrigeration (Manning et al., 1974). While no recent work has been done on the dietary patterns of urban blacks in Soweto, the majority of the township is now electrified. Also, outlets for precooked fish are very common, and discussions with owners of retail outlets indicate a large turnover of tinned pilchards in tomato sauce. While it is difficult to envision the control group in this study maintaining a larger intake of omega-3 fatty acids based on fast food outlets and tinned pilchard, the availability is none the less present.

An interesting correlation to the increased levels of EPA in the control group is the finding that this group also had a higher platelet membrane cholesterol to protein ratio. In a study conducted by Goodnight et al., (1981), normal individuals were given concentrated fish fatty acid extracts for eight weeks. After the eight week period it was found that the group experienced a decrease in platelet responsiveness to ADP and an increase in platelet membrane cholesterol levels. The possibility may exist that even slightly

increased levels of EPA may increase the phase solubility of cholesterol in the membrane. Given the platelet aggregation findings by Goodnight, this may occur without the adverse changes in platelet aggregation which have been demonstrated when platelets are incubated with cholesterol rich liposomes (Kramer et al., 1982). Whether or not this is actually the case, and whether it occurs in cells such as macrophages which have a tendency to degenerate into foam cells when overloaded with cholesterol, may be a topic worthy of further investigation.

7.1.2. *Serum Lipids.*

Hypercholesterolemia is a well established risk factor for CHD. This has been demonstrated in many epidemiologic studies, both within and between white populations. While comparative studies in America have indicated that absolute levels of serum cholesterol are similar in black and white males, blacks have more cholesterol associated with HDL and less associated with LDL. Furthermore, rural black Africans on a high cholesterol diet have lower total serum cholesterol and LDL cholesterol and higher HDL cholesterol than that reported for normal urban Black Americans (Vorster et al., 1987; Cooper et al., 1985). Given these racial differences in lipogram profile, studies are warranted to determine the specifics for CAD risk in Black African populations. In the current study, total serum

cholesterol was significantly elevated in the cases and this elevation was almost entirely reflected by significantly elevated levels of LDL cholesterol. From what is known about the cellular metabolism of cholesterol, LDL is the most atherogenic of the lipoproteins (Brown and Goldstein 1984). Also, serum apolipoprotein B (apo B) was significantly elevated in the cases. Durrington et al., (1986) using discriminate analysis in a White British population determined that out of all the lipid parameters, apo B was the best single discriminating variable in men who had previously had a myocardial infarction.

As indicated by the similarities in total saturated and total polyunsaturated fatty acids in RBCs and platelet poor plasma, it is unlikely that the elevated levels of cholesterol are related to differences in diet. In white populations, a very small percentage of hypercholesterolemia is caused by inherited defects of the LDL receptor (Vega et al., 1987). Familial hypercholesterolemia is rarely detected in black populations (Cooper et al., 1985). Other causes of increased circulating LDL-C could be overproduction of lipoproteins by the liver, or low affinity of circulating LDL-C for receptors caused by a primary defect in apo B-100. Answers to these hypotheses await further studies on the fractional clearance rates of LDL-C, and the structural diversity of apo B-100 in black populations.

Decreased levels of HDL-C and apo A1 have been shown to correlate positively with the occurrence of MI in white populations (Durrington et al., 1986; Salonen et al., 1985). Both HDL-C and serum Apo A1 levels were very similar among the two groups in this study. Other investigations which have examined lipoproteins and apolipoproteins in normal white and black populations have found HDL-C and serum apo A1 levels to be elevated in the blacks (Freedman et al., 1987). It is known that apo A1 is cleared from circulation by the kidneys (Patch and Gotto 1987). It is also known that there are differences in renal function between the two populations (Levy et al., 1978) however, studies have not been carried out to determine if this is related to the elevated levels of these substances in blacks. Whatever the reason, HDL-C and serum Apo A1 do not appear to be a risk factor determinants for MI in black populations, and may have some influence on the overall lower levels of CAD observed in these populations.

7.1.3. *Uric Acid and Blood Glucose.*

Uric acid and blood glucose levels were measured in this study, and they were both significantly elevated in the cases. While it has been indicated that increased levels of these substances may be a risk determinant for CAD (Gertler and White 1976), the levels in this study

may not be meaningful, as the majority of the cases were receiving a furosemide diuretic. This medication causes increases in blood glucose and uric acid levels (Rastagar and Thier 1977). These values were reported, as the normal levels may be of some future benefit.

7.1.4. *Clotting Factors and Inhibitors.*

High fibrinogen concentrations have been associated with an increased risk of death from CAD (Meade et al., 1985), as have elevated levels of factor VII (Meade et al., 1980). Also, abnormalities of fibrinolytic factors such as plasminogen (Brommer and Brakman 1984) and protein C (Gonzales et al., 1985) have been shown to result in a thrombotic condition. In the current study, fibrinogen and plasminogen were elevated in the case group. Factor VII and protein C were not significantly different. Elevated fibrinogen levels have been reported in a normal Black African group on a high cholesterol diet indicating that this may be an important risk factor determinant in this population (Vorster, et al., 1987). Abnormalities in these variables in patients with preexisting CAD however, may either be due to atherosclerotic lesions, or to on going congenital abnormalities (Breddin 1986). Given this, a cause and effect relationship between these factors and risk for the occurrence of MI is difficult based on the data from this study.

7.2. *Conclusions*

In conclusion, the diet of normal urban Black Africans and urban Black Africans who have experienced an MI is very similar. This is in contrast to a number of other studies which have determined dietary differences retrospectively in white groups who have experienced an MI. The indication is that the increasing incidence of MI in a Black South African urban population may not be caused exclusively by changes in dietary habits. Other reasons for this increase may be found in socioeconomic changes resulting from urbanization and westernization. The platelets of Black Africans who have experienced an MI contain significantly greater amounts of saturated fatty acids and lower amounts of PUFA than a normal black group and this does not appear to be related to diet. This is contradictory to studies done in White European populations which have demonstrated dietary changes in platelet function in relation to MI. These variations cannot be readily explained at this time. Low levels of 20:5 w-3 in platelets and RBCs may be an important risk determinant for MI in this, and other populations. Elevated levels of total cholesterol and LDL-C appear to be important indicators of CAD risk in black populations. Reasons for this elevation in the absence of dietary variation await further studies. Finally, fibrinogen levels may be an important risk factor determinant in the Black population however further studies are required to confirm this.

CHAPTER 8

ABKIEWICZ, S.R., AHMED, A.S., ALLI, M.A., ALLY, R., AMANJEE, M.S., BAUM, D., BEALE, E., BENLOULOU, G.C., BERKOWITZ, I., BHAGA, H.R., BLUMENTHAL, E., BLUMSOHN, A., BLUMSOHN, D., BOOKATZ, B., BOOK, N., BRAUDE, M., BRAY, H.E., BROWN, J., BRUNING, A.H., CAPLIN, B.A., CASSIMJEE, F.G.H., CELLIERS, L., CHUNG, R., CHURCHYARD, G.J., DEMETRIOUS, A., DUBB, A., DURAO, H., EDELSTEIN, D.M., ESSA, S., FETTER, G.K., FRAME, G., FRANKLIN, D.S., FRIEDMAN, M., GAYTANAKIS, H., GELDARD, P., GHOOR, M.A., GLOCER, J., GORDON, A.I., GROBBELAAR, T.M., GUEST, S., HECKMANN, J.M., HODKINSON, H.J., HON, H., HUDDLE, K., ISMAIL, M.H.A., JAQUES, V., JIVHUHO, L., KAHN, H., KARSTAEDT, A.S., KENYON, M., KEW, J., KHUMALO, B.F., KLENOVSEK, M., KRUT, L.H., LATAKGOMO, L.L., LAZAR, A., LONG, J.E.H., MAKHANE, M.S., MANCLOVSKI, J.M., MARINOPOULOS, G.C., MARSH, D., MENDELSOHN, H., MENDOCA, R., MODI, D., MOHAMED, A.E., MOKGATLE, L., MZAMANE, D.V.A., NANABHAI, F., NGCOBO, N., NGCWABE, Z., NGWENYA, E., OSTERMEYER, C., PARBHOO, T., PATEL, B.A., PATEL, M., PEREIRA, L., PILLAY, M., PLANER, B.C., RAPEPORT, N., RAPHUNGA, M.M., RATHAKRISHNAN, S., RIEDEL, L., RUBIN, G., SACK, J., SAFFER, D., SALEY, Y., SEEDAT, M.A., SEGAL, K.D., SHAMLEY, D.J., SHAPIRO, M.H., SHIRES, R., SINGH, V., SINOFF, C., SIWINSKA, D., SOFIANOS, J., STANWIX, A., STAVROU, C., TIKLY, M., TRAUB, B., WEINER, S., WULFSOHN, A.H. Conditions at Baragwanath Hospital. S. Afr. Med. J. 72: 361, 1987.

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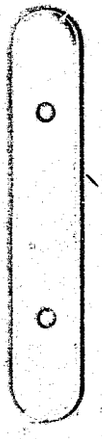

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