

Hyperlipidemia A huge prospective study conducted in France indicates that there was no connection between the Rh blood type and risk of type 2 diabetes mellitus (T2DM). Further comparisons were made between the diabetics and controls in the group B and O secretors: mostly, fasting I-ALP activity was greater in the diabetics than in the controls; both types of diabetics had considerably greater liver ALP activity than the controls; type 2 diabetics had greater liver ALP than type 1 diabetics; and type 2 diabetics also had additional abnormal ALT and GGT values than type 1 diabetics [7, 33]. Undoubted confirmation has described a genetic relationship in those of European ancestry between nonsecretor status (se/se; homozygous for the A/A alleles of the FUT2 gene) and insulin-dependent type 1 diabetes mellitus (T1DM) [7]. The I-ALP findings by blood type for diabetes were the same as the findings for hyperlipidemia, with considerably greater serum I-ALP and entire ALP levels in blood type B and O secretors (together with controls) when compared to A secretors or ABO nonsecretors, but no substantial variation in serum I-ALP or entire ALP between A secretors and ABO nonsecretors (together with controls) [7, 33]. When adjusted for metabolic covariates (fasting blood glucose and lipids), blood type AB persons showed the maximum risk of T2DM (odds ratio, 1.95), followed by type B (odds ratio, 1.26) and type A (odds ratio, 1.21) as compared with blood type O persons, who had the lowest risk [61]. However, when comparing blood type B and O secretor diabetics to controls, I-ALP activity was similar between type 1 and type 2 diabetics but considerably greater in both types than in the controls; likewise, there was insignificant variation in I-ALP activity between type 1 and type 2 diabetics in the A secretor and ABO nonsecretor groups, but both types of diabetics had greater I-ALP activity than the controls in these groups [7, 33]. Other findings also indicated inconsistent results: a study in Yemen indicated that the maximum arbitrary blood sugar and insulin levels were found in blood type A, whereas blood type AB showed a defensive effect [56]. The odds ratio for nonsecretor status was 1.29 ($p = 7.3 \times 10^{-14}$) in the case of the control population, while the relative risk for nonsecretor status was 1.22 ($p = 6.8 \times 10^{-6}$) in the diabetic family population and the combined results clearly indicated a locus for T1DM in the FUT2 gene ($p = 4.3 \times 10^{-18}$) [7]. However, individuals with blood type O shows the lowermost risk of T2DM, whereas those with blood type B were at the uppermost risk, followed by type AB and type A individuals; nevertheless, the risk for type AB people did not have statistical implication [7, 61]. Troubled liver function could weaken the clearance of I-ALP by the liver and would elucidate the greater ALP levels found in the diabetics; high I-ALP has been described in victims with cirrhosis of the liver [7]. A research in Iraqi persons indicated greater blood glucose, total cholesterol, and blood pressure in blood type O persons, followed by lesser risk in type A, B, and AB persons, who showed the bottommost risk [7, 61]. When Rh and ABO types were assessed together, blood type B+ persons showed the uppermost risk, followed by type AB+, A-, and A+ persons, but similar risk was seen for the other types [7, 61]. A large study in Bangladesh indicated that there was no relationship between ABO blood types and T2DM. A study in Malaysia also shows lesser risk of T2DM in blood types A and O [62].