Different contribution of aldehyde dehydrogenase type 2 (ALDH2) to metabolic activation between glyceryl trinitrate (GTN) and isosorbide dinitrate (ISDN) has been suggested. However, systematic mechanical evaluation has not been presented. Therefore, we examined degree of contribution of ALDH2 to vasodilatory effect of GTN, ISDN, sodium nitroprusside (SNP) and NaNO$_2$ on excised aortic ring preparation suspended in organ bath. Phenylephrine was used to obtain sustained contraction of the preparation. GTN-, ISDN- and NaNO$_2$-induced relaxations were significantly attenuated by ALDH2 inhibitors, cyanamide (3x10$^{-4}$M) and chloral hydrate (5x10$^{-4}$M). Rank order of the susceptibility (ratio of pD$_2$ in the presence of a inhibitor to that in the absence of the agent) was GTN > ISDN > NaNO$_2$ by both inhibitors. Vasodilatory effects of above relaxants, except for SNP, were significantly inhibited by methylene blue (MB: 10$^{-5}$M), a guanylate cyclase inhibitor. In guinea-pig aortic ring SNP-induced relaxation was susceptible to MB, where similar results (rank order of susceptibility to the inhibitors was GTN > ISDN > NaNO$_2$) were obtained as well. These results indicated that ALDH2 is involved in activation of GTN and also, in smaller degree, ISDN and NaNO$_2$, but not SNP.

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