decrease of the fluorescence obtained. In contrast, *erythro*-α-methylnoradrenaline after boiling gained a fluorescence of the same intensity as the boiled *threo* form. It thus appears that under the present conditions an equilibrium between the two diastereoisomers is reached. The fluorescence from noradrenaline was but slightly reduced after boiling in an acid medium (Table 1). So it would be possible to make differential estimations of *erythro*-α-methylnoradrenaline and noradrenaline.

TABLE 1. THE RELATIVE FLUORESCENCE INTENSITY OF *threo*-α-METHYLNORADRENALINE (α-Me-NA), *erythro*-α-Me-NA AND NORADRENALINE (NA) WHEN TREATED ACCORDING TO THE TRIHYDROXYINDOLE METHOD WITH AND WITHOUT PREVIOUS BOILING IN AN ACID MEDIUM (SEE TEXT). ACTIVATING AND FLUORESCENCE PEAKS: 400 AND 510 mμ, RESPECTIVELY

	threo-α-Me-NA	*erythro*-α-Me-NA	NA
Unboiled ..	100 ± 5	6 ± 1	103 ± 5
Boiled	69 ± 1	69 ± 2	92 ± 2

Mean ± s.e.m. of 3–5 determinations.

Experiments are now in progress to apply this principle on eluates from tissue extracts.

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Inhibition of ethanol-induced glycogenolysis in brain and liver by adrenergic β-blockade

SIR,—Ethanol is known to increase lipolysis in adipose tissue (Brodie, Butler & others, 1961; Estler & Ammon, 1967; Ammon, Estler & others, 1966) and glycogenolysis in brain and liver (Ammon, Estler & Heim, 1965; Ammon, Estler & Heim, 1966; Estler & Ammon, 1965). The increased lipolysis is supposed to be due to the action of catecholamines which are released from the adrenal medulla by ethanol (Klingman & McC. Goodall, 1957; Abelin, Herren & Berli, 1958; Perman, 1961; Wartburg, Berli & Aebi, 1961) because it can be prevented by adrenalectomy (Mallov & Gierke, 1957), α-adrenergic blocking agents (Brodie & others, 1961) and β-adrenergic blocking agents (Estler & Ammon, 1967). Since catecholamines increase glycogenolysis by stimulating the adenylcyclase system, which activates not only the hormone-sensitive lipase in adipose tissue but also phosphorylase, we examined whether the glycogenolytic action of ethanol in brain and liver is mediated by catecholamines which

stimulate the adrenergic β-receptors. Female white NMRI mice, kept at 24° and fed with a standard diet (Altromin, Altromin G.m.b.H. Lage/Lippe, Germany) were given 50 μg/g of the β-blocking agent Kö 592 [1-(isopropyl-amino)-3-(*m*-toloxy)-2-propanol HCl] subcutaneously 30 min before the intravenous injection of 1·5 mg/g ethanol. At 10 and 30 min after the ethanol injection the animals were killed by immersion in liquid air and brain and liver tissue were prepared frozen. The glycogen content was estimated (Kemp & Kits van Heijningen, 1954).

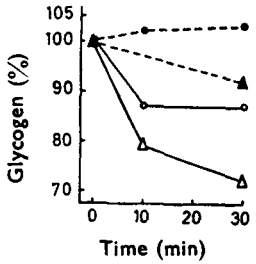

FIG. 1. Glycogen in brain (circles: 100% = 5·1 μmole glucose/g) and liver (triangles: 100% = 352 μmole glucose/g) of female white mice. —— after i.v. injection of 1·5 mg/g ethanol. – – – after i.v. injection of 1·5 mg/g ethanol in animals which had received 50 μg/g Kö 592 30 min before the ethanol. 100% = control value; ○ = $P \leq 0{\cdot}05$.

The intravenous injection of ethanol was followed by a decrease of glycogen in brain and liver (Fig. 1). But neither in the brain nor in the liver was there a significant decrease of glycogen after ethanol when the animals were pretreated with Kö 592.

It seems that, similarly to lipolysis in adipose tissue, the enhanced glycogenolysis in brain and liver which follows the administration of ethanol is due to the action of catecholamines, which it releases. These results are in accordance with earlier findings from this laboratory (Estler & Ammon 1965) that ethanol-induced glycogenolysis in the brain could be prevented by adrenalectomy.

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