Mokwa are the non-repellent *Crematogaster* sp.C (6-methyl-3-octanol dominant) and sp.B (3-octanol dominant).

In the tribe Tetramoriini, most *Tetramorium* species have a mandibular gland secretion akin to *Crematogaster* with 3-octanone dominant. An exception is the lestobiotic *T. termitobium* in which 2-undecanol predominates. Repellent *Tetramorium* species contain large quantities of perillen, a compound previously found in the formicine *Lasius fuliginosus*[8]. *D. uelense* has 3-octanol as the major component.

A series of bioassays was carried out on synthetic samples and natural fractions of ant mandibular gland components and related compounds. Aliphatic alcohols were not repellent to the termites, but repellency was observed with aliphatic ketones and aldehydes (table 2).

It appears that those myrmicines which are specialized or semi-specialized termite predators have aliphatic alcohols in the mandibular glands (possibly for the communication of alarm or other behaviour) instead of the more repellent carbonyl and other compounds which are usually present in other members of the same genera[7,9]. This enables the ants to move among the termites without being detected.

Bioassays performed on extracts of ants indicated that all parts were repellent, but that this repellency originated from exocrine glands in the head. In *Apis mellifera*, it was found that trans-9-keto-decenoic acid, part of the 'queen substance' from the mandibular glands could be found all over the cuticle[10]. A similar spreading of the compounds from the mandibular glands may also explain the repellency of some ants to termites.

We conclude that small quantities of glandular compounds, released on to the cuticles of ants, may form cues by which termites recognise their predators. Those ants which have been able to emphasise components of their secretions which termites cannot detect are able to become successful termite predators by virtue of this 'chemical crypsis'. These findings may help to explain the great diversity of exocrine secretions found in the Formicidae[9].

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Cerebral ammonia production during hypoglycaemia in the newborn calf[1]

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Summary. The effect of insulin hypoglycaemia on cerebral blood flow, and cerebral metabolic rates of glucose, oxygen and ammonia was investigated in the unanaesthetized newborn calf. A net loss of ammonia from the brain occurred during hypoglycaemia, and was greater in convulsing than in comatose animals.

Ammonia accumulates in brain tissue deprived of an adequate supply of glucose. This has been demonstrated both in vitro[3,4] and in vivo[5,6] by the measurement of brain tissue ammonia concentrations. In addition, a net loss of ammonia by the brain to the circulation has been observed during insulin hypoglycaemia in the anaesthetized dog[5], but could not be quantified as cerebral blood flow was not measured. These experiments were undertaken to quantify cerebral ammonia production during insulin-induced hypoglycaemia in the unanaesthetized newborn calf.

Cerebral metabolism was determined by the simultaneous measurement of cerebral blood flow and arterio-cerebral venous concentration differences of oxygen, glucose and ammonia. Cerebral blood flow was measured by an inert-gas technique[7] using molecular hydrogen gas and intravascular catheter mounted platinum electrodes[8,9]. Metabolite estimations were performed in duplicate on paired samples withdrawn simultaneously from the aorta and sagittal dural sinus at the beginning and end of each flow determination. Sampling catheters and electrodes were inserted under general anaesthesia (halothane) on the day prior to the experiment. Ammonia was estimated within 2 h of collection using a specific enzymatic technique[10]. Glucose was measured with a Beckman Glucose Analyser (Mark 2) and whole blood oxygen content by a polarographic technique[11]. Arterial blood gas tensions, pH and blood pressure were also monitored. The values for flow and metabolism refer to tissue drained by the sagittal dural sinus, which is predominantly cerebral cortex.

The experiments were carried out on 15 pedigree Jersey calves aged between 1 and 22 days. Measurements were made in unanaesthetized, unrestrained animals during normoglycaemia and following i.v. administration of insulin (Soluble Insulin B.P. Wellcome, 4 IU/kg b.wt).

Observations made during hypoglycaemia are subdivided into 2 groups according to the clinical state of the animal. 18 observations were made in animals showing signs of somnolence and lethargy (comatose), and 7 during generalized seizures. No significant arterio-cerebral venous concentration difference for ammonia was observed during normoglycaemia, but a statistically significant net loss of ammonia from the brain was found during hypoglycaemia. Comparison of measurements made during hypoglycaemic seizures, with those made in comatose hypoglycaemic animals, showed a significantly raised cerebral blood flow and oxygen consumption ($p < 0.001$), a lower molar ratio of glucose uptake to oxygen consumption ($6 \times CMR_{Gl}/CMR_{O_2}$) ($p > 0.1$), and a higher rate of cerebral ammonia production ($p < 0.01$) (table).

Comparison of measurements made during normoglycaemia and hypoglycaemia in the unanaesthetized newborn calf

	No. of observations	MABP mm Hg	Pa_{O_2} mm Hg	Pa_{CO_2} mm Hg	pH	Arterial plasma glucose mmole/l	Sagittal sinus plasma glucose mmole/l	Arterial plasma NH_4^+ μmole/l	Sagittal sinus plasma NH_4^+ μmole/l	PCV	CBF ml · 100 g^{-1} min^{-1}	CMR_{Gl} μm · 100 g^{-1} min^{-1}	CMR_{O_2} μm · 100 g^{-1} min^{-1}	Glucose-oxygen index	CMR_{NH_4} μm · 100 g^{-1} min^{-1}
Normoglycaemia	35	92 ±3	66 ±2	41 ±1	7.41 ±8.01	4.72 ±0.38	4.26 ±0.40	42 ±5	42 ±4	33 ±1	45 ±2	14 ±1	92 ±4	0.94 ±0.03	0.00
Hypoglycaemia Comatose	18	84 ±2	64 ±3	40 ±1	7.40 ±0.01	1.06 ±0.10	0.67 ±0.10	48 ±7	56 ±7	36 ±3	44 ±5	11 ±1	79 ±7	0.80 ±0.08	−0.25 ±0.06
Seizures	7	88 ±4	67 ±5	41 ±2	7.39 ±0.01	0.87 ±0.16	0.66 ±0.15	42 ±8	60 ±8	35 ±3	112 ±13	15 ±4	124 ±16	0.60 ±0.14	−0.87 ±0.16

The values are means ± SEM. MABP: mean arterial blood pressure, CBF: cerebral blood flow, CMR: cerebral metabolic rate, Glucose-oxygen index: $6 \times CMR_{Gl}/CMR_{O_2}$. PCV = Packed cell volume.

$CMR_{NH_4}+$ during hypoglycaemia and following restoration of normoglycaemia.

On 4 occasions measurements were repeated at least 30 min after the start of i.v. glucose infusions (5–15 g) which raised the mean arterial plasma glucose concentration from 0.88±0.09 mmole/l to 2.67±0.47 mmole/l. The mean $CMR_{NH_4^+}$ was found to have fallen from −0.65±0.10 to −0.09±0.05 μmoles. 100 g^{-1} · min^{-1} ($p < 0.01$), (figure). These results provide confirmatory evidence that a rise in cerebral ammonia concentration accompanies hypoglycaemia, and show that this occurs in the newborn animal. They also represent the first quantification of net loss of ammonia from the brain during hypoglycaemia. Accumulation of ammonia may be an unavoidable consequence of utilization of endogenous amino acids as alternative substrates during hypoglycaemia[12]. The glucose-oxygen index fell below one during hypoglycaemia in these animals, indicating that alternative substrates were being oxidized. The simultaneous production of ammonia strongly suggests that these included endogenous amino acids. The precise mechanism of the functional central nervous system disturbances which accompany hypoglycaemia is not known. Ammonia is well known to have toxic effects on the central nervous system, and might well have a role in hypoglycaemic encephalopathy. Although ammonia production was significantly higher in convulsing animals in these experiments, a causative role for ammonia in seizure initiation cannot be inferred as seizure activity is itself associated with increased cerebral ammonia production[13].

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