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NL-339 (r 82/08)

Canada

Interhemispheric Communication and Binocular Vision

Submitted in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy
at Dalhousie University

October 1981

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ABSTRACT

It is widely accepted that the neural mechanism for stereoscopic depth perception can be found in the disparity-sensitive response of single visual neurons. The present study was undertaken to characterize the disparity-sensitive neuron, to elucidate its mechanisms and to investigate the transfer of depth-specific visual information between the two sides of the brain. Binocular visual interactions were examined in single units from the 17/18 border of normal cats and compared to responses from the 17/18 border of cats with large unilateral lesions of the opposite visual cortex. Units were activated with stimuli of varying disparity, moved in the same (sideways motion) and in opposite directions (motion in depth) on the two retinae. In normal cats, neurons showing substantial binocular interactions could be distinguished from disparity-insensitive units by cell type, ocular dominance, directional properties and cortical location. These data indicated clear dimensions in the organization of stereoscopic depth systems in cat visual cortex. Data from both normal and lesioned animals indicated that the critical mechanism of the disparity-sensitive response of single visual cells was binocular inhibition. Unilateral lesions of the visual cortex effected a specific subpopulation of neurons, rendering them unselective for stimulus disparity, and the location of these units, nicely mimicked the known distribution of callosal fibers in cat visual cortex. These data emphasize the role of intrinsic inhibitory circuits in the function of input from the two eyes and suggest that the corpus callosum plays a distinct role in the transmission of stereoscopic depth information between the two sides of the brain.

ABBREVIATIONS

VCL	- Visual cortex lesioned cats
Normal	- Normal cats
BI	- Binocular inhibition
BF	- Binocular facilitation
OD	- Ocular dominance
IN	- In-phase dynamic range
AN	- Antiphase dynamic range
COM	- Combined dynamic range
MIN	- Medial interlaminar nucleus of the thalamus
LGNd	- Dorsal lateral geniculate nucleus of the thalamus
LSVA	- Lateral suprasylvian visual area
AP	- Anterior-posterior
deg/sec	- degrees per second
mm	- millimeter
mg	- milligram
kg	- kilogram
CO_2	- carbon dioxide
O_2	- oxygen

ACKNOWLEDGEMENTS

A most sincere thank you to the members of my examining committee, Drs. Max Cynader, Ian Meinertzhagen, Donald Mitchell and Gian Poggio for giving this manuscript a careful reading, for making many useful comments and for taking the time to go over them with me. I've really appreciated the help. Thank you, Dr. Poggio, for making the trip to Halifax, despite your busy schedule and despite the difficulty in getting here. Thank you Ian and Donald, not only for the feedback you've provided on this thesis, but for contributing so much , and in so many different ways, to my years at Dalhousie. And Max, my thesis advisor, thank you for sharing with me--as you do with every one who comes into the lab-- your energy, enthusiasm, sense of humour, and your very creative view of scientific issues. To all of you, I hope our continued association is a long one.

INTRODUCTION

The horizontal offset of the two eyes in the head provides the geometric basis for binocular disparity and stereopsis. Since each eye views the same visual scene from a slightly differing vantage point, objects separated in depth fall on retinal coordinates which are not in perfect correspondence. This deviation from correspondence, called retinal disparity, was shown by Wheatstone (1838) to be a sufficient cue in the transformation of 2 dimensional retinal input into 3 dimensional visual scenes. Wheatstone's stereoscope, a simple device for producing controlled horizontal retinal disparities, is still in use today, and graphically illustrates the fact that horizontal disparity between the inputs from the two eyes is sufficient in and of itself to produce a vivid sensation of depth.

Presumably, the neural mechanism which appreciates these retinal disparities must be one which involves the convergence and combination of inputs from the two eyes. Since the visual cortex is the first point in the visual pathway where there is significant convergence of input from the two eyes onto single neural elements (Hubel and Wiesel, 1962), it was here that Barlow, Blakemore and Pettigrew (1967) sought the substrate for the neuronal mechanism of stereopsis. Recording from single neurons in cat visual cortex, they found neurons which responded differentially to binocular stimuli as a function of retinal disparity. Cells were encountered which had receptive fields on noncorresponding retinal coordinates, implying that at a fixed point of convergence, different cells would be optimally activated by stimuli of different depths. Other workers (Nikara, Bishop and

Pettigrew, 1968; Joshua and Bishop, 1970; Hubel and Wiesel, 1970; von der Heydt, Adorjani, Hanny and Baumgartner, 1978; Ferester, 1981), while differing with some of the conclusions of Barlow et al., provided confirmation of the essential idea--that visual cortical cells are sensitive to the retinal disparity of binocular stimuli-- a finding which has recently been extended to a variety of frontal-eyed species (Pettigrew and Konishi, 1976; Clark, Donaldson and Whitteridge, 1976; Poggio and Fischer, 1977; Fischer and Kruger, 1979).

The problem of midline stereopsis

Although disparity - sensitive neurons were only quite recently identified, the notion that stereoscopic processing involved the convergence of input from the two eyes onto a particular cortical locus was not a new one. As early as 1900, this idea was expressed by Heine (in Blakemore, 1970) in consideration of what may be called the problem of midline stereopsis. In light of the classic view of a strict nasotemporal division, Heine wondered how input from the two eyes subserving the region of visual space directly around the fixation point could converge onto a single cortical locus. Since it was believed that a partial decussation of fibers at the optic chiasm segregated the output of the nasal and temporal portions of the retina, it seemed that objects lying right in front of, or behind the fixation point, would be imaged respectively on the two temporal or nasal retinae, and that the input from each eye would be transmitted to opposite visual cortices. Thus, there would be no opportunity for information from the two eyes to converge upon a single cortical

locus. This arrangement must have seemed somewhat paradoxical, particularly in light of the knowledge (Helmholtz, 1867) that stereopsis was most acute in regions immediately surrounding the fixation point. Until relatively recently, it was not clear in this situation how or where the neural integration of information from the two eyes occurred.

In the last decade however, anatomical and physiological investigations have identified two independent routes for the transfer of information from the midline of the visual field: (1) retinal fibers from a zone of nasotemporal overlap which project to both optic tracts and (2) the corpus callosum.

The inexactitude of the nastemporal division

One of the first to challenge the widely accepted view of a strict nasotemporal division appears to have been Linksz (1952). He did so in an effort to account for the clinical phenomenon of "macular sparing". Macular or "foveal sparing" refers to a perceptual phenomenon observed in patients who have undergone removal of one occipital lobe. Not surprisingly, the lesion produces a homonymous hemianopsia--a loss of vision in the contralateral half of the visual field. In cases of macular sparing however, the separation between the blind and the normal half of the visual field occurs about 1° from the midline, toward the blind half of the visual field. Since the removal of one visual hemisphere functionally eliminates the callosal pathway, any vision beyond the midline must be attributable to other mechanisms. Linksz thought that the most likely explanation was an inexactitude of the nasotemporal division. He felt that hemidecussation at the optic chiasm was a statistical rather than an

absolute process and suspected that there must be a projection of at least some temporal retinal cells toward the opposite side of the brain. The size of the area of spared macular vision and the phenomenon itself would suggest that these fibers represent at least 1^{o} of binocular overlap and that they alone are sufficient to subserve midline vision.

Linkz's suspicion, that hemidecussation was inexact, has since been born out by a a number of anatomical studies in both cat (Stone, 1966; Stone and Fukuda, 1974; Kirk, Levick, Cleland and Wassle, 1976; Kirk, Levick and Cleland, 1976) and monkey (Stone, Leicester and Sherman, 1973; Bunt, Minckler and Johanson, 1977). In primates a 1^{o} strip of retina has been found which straddles the vertical meridian and projects to both optic tracts. A similar amount of overlap, about 1.2^{o}, has also been seen in the cat retina, among brisk-sustained units (Kirk, Levick, Cleland and Wassle, 1976) and X-cells (Stone and Fukuda, 1974). Larger amounts of overlap have been observed in brisk-transient units (Kirk, Levick, Cleland and Wassle, 1976) Y-cells (Stone and Fukuda, 1974) and in cells with slowly conducting axons (Stone and Fukuda 1974; Kirk, Levick and Cleland; 1976). Fibers from this zone of nasotemporal overlap have been found to project to the medial edge of the cat dorsal lateral geniculate nucleus (LGNd) whence the thalamic fibers project to the border between areas 17 and 18. (Sanderson and Sherman, 1971; Kinston, Vadas and Bishop, 1969). Fibers terminate in all main laminae of both LGN's and in the adjoining region of the medial interlaminar nucleus (MIN). In general, larger amounts of overlap have been found in the thalamus and visual cortex (Hubel and Wiesel, 1967; Nikara, Bishop and Pettigrew,

1968; Blakemore, 1969; Joshua and Bishop, 1970) than in the retina.

An electrophysiological demonstration that the input from these overlapping retinal fibers could influence neuronal responses at the level of the visual cortex was provided by Leicester in 1968. Mapping the location of receptive fields along the cat 17/18 border, he found a centrally located strip of bilaterally represented receptive fields which extended .5 to 1° into the ipsilateral hemifield. Sectioning the corpus callosum had no effect on the amount of overlap which was observed. Since any possible influence from the callosum was eliminated with the lesion, the ipsilateral representation of visual fields was attributed to a retino-thalamo-cortical projection. Recently, neurons in the lateral suprasylvian visual area (LSVA) have also been shown to receive ipsilateral activation via a similar projection. In a study by Marzi, Antonini and Legg, (1980) contralateral eye receptive fields in the LSVA extended up to 10° into the ipsilateral half-field after lesions of the corpus callosum. That a greater degree of spared ipsilateral overlap was seen in the LSVA than in the visual cortex corresponds well with the observation that a larger amount of ipsilateral representation can be found in the MIN, the thalamic nucleus which projects to the LSVA, than in the LGNd, the nucleus projecting to the visual cortex (Kingston, Vadas, Bishop, 1969; Sanderson, 1971; Kratz, Webb and Sherman, 1978).

The contribution of the corpus callosum

In addition to a thalamo-cortical projection, a second route for the transmission of input from the central visual fields is, of course, the corpus callosum. Numerous investigations have demonstrated that this commissural pathway is in fact a viable and functionally

efficacious route for the transfer of visual information between the two cerebral hemispheres. One of the first such demonstrations was a study by Choudhury, Whitteridge and Wilson (1965), who, after establishing that fibers ran from the margin of area 17 to their corresponding points in the opposite cortex, isolated the visual input to a single hemisphere by severing one optic tract. They found that in the deafferented visual cortex, responses could be obtained only from cells which had receptive fields located along the vertical meridian. This study was one of the first to show that the influence of this pathway was restricted to the central visual fields and also, that cortical neurons could be activated by input received exclusively via the corpus callosum. A similar experimental approach, was applied in a study by Berlucchi and Rizzolatti (1968) who, in spliting the optic chiasms of cats, restricted the input to each hemisphere to the ipsilateral retinal projection. Recording from units along the 17/18 border, they found neurons which had clearly-defined visual receptive fields in both eyes. Presumably, responses through the ipsilateral eye were mediated by thalamo-cortical connections while responses through the contralateral eye were due to cortico-cortico, callosal connections. Recently, a study by Cynader et al. (1979) has shown that the corpus callosum not only contributes an excitatory input to cells along the opposite 17/18 border, but also, that it specifically mediates disparity-sensitive responses. In these experiments, binocular interactions were measured in cats which had undergone a surgical section of the optic chiasm, and thus again, the only possible route for convergence of input from the two eyes was via the corpus callosum. Binocular interactions in these animals were

reduced relative to normal cats, but there was clear evidence for extensive binocular convergence and of disparity sensitive interactions.

The possible role of the corpus callosum in the transmission of disparity specific information and the relevance of this pathway to the problem of midline stereopsis was an issue considered by Blakemore (1970) in a study of a human patient who had a saggital section of the optic chiasm. In testing this patient's stereoscopic function, Blakemore predicted that since only the temporal retinal pathways remained intact, the subject should be able to discern the depth of stimuli lying immediately in front of the fixation point (crossed disparities) while at the same time being completely blind to objects immediately beyond the fixation point (uncrossed disparities). When measured with stimulus disparities of $.5^{o}$ to 6^{o}, this prediction was confirmed. The data indicated that the callosal pathway integrated information up to 3^{o} within the temporal retina of each eye. Since there was no evidence of stereopsis for uncrossed disparities, and since there was no sign of macular sparing, Blakemore concluded that it was the corpus callosum exclusively which was mediating this residual stereoscopic function.

In the patient described above, section of the optic chiasm did not disrupt convergent, fusional eye movements to a crossed disparate stimulus, and it thus appeared that the corpus callosum was also involved in the mediation of vergence eye movements. Further support for this association came from Westheimer and Mitchell (1969) and Mitchell and Blakemore (1970) who, when testing a subject who had had a surgical division of the callosum, found both a lack of depth perception and a lack of vergence eye movements to centrally located

targets. The subject's stereopsis and vergence eye movements were normal when tested with a target located 5° into the peripheral visual field, but were absent in midline vision for both convergent and divergent disparities of 2°. These data suggested that the corpus callosum enjoys a dual function, being involved not only in the mediation of midline stereopsis, but also, in the generation of vergence eye movements elicited by binocularly disparate stimuli.

Fine and coarse stereopsis

The studies of Blakemore (1970) and Mitchell and Blakemore (1970) suggested that midline stereopsis was principally mediated by the corpus callosum rather than by retinal fibers of the nasotemporal overlap. This is a conclusion however, which has been vigorously criticized by Bishop and Henry (1971) and Bishop (1981). These authors have pointed to the distinction between what appears to be two different stereoscopic subsystems (Ogle, 1950), one for "fine" and the other for "coarse" stereopsis, and they claim that the above studies tested only for coarse stereopsis . They feel that coarse stereopsis may in fact be mediated by the corpus callosum, but that fine stereopsis relies on the direct retinal projection. Since the disparities used for testing in in the above studies were too large to measure fine stereopsis, their conclusion was that Blakemore's claims were too sweeping and that his results indicated only the preservation of a relatively coarse stereoscopic system.

According to the formulation of Bishop and Henry (1971) and Bishop (1981), stereopsis is a dual system composed of separate mechanisms for fine and coarse stereoacuity and fusion which can operate, at least in part, independently of one another. Fine

st reopsis and single vision operates only within a very narrow range of stimulus disparities--probably less than .5°, provides for high-resolution stereoacuity and is always accompanied by coarse fusion. It requires very close similarity between the visual images in the two eyes or else retinal rivalry and suppression of one monocular input occurs. Coarse stereopsis, on the other hand, can operate when there is considerable difference between the two retinal images in form, luminance and the temporal onset of stimuli in the two eyes, and can tolerate up to 7-10° of retinal image disparity. Coarse single vision requires some degree of similarity between the two retinal images, but again, can operate with retinal image disparities of up to 2° and can occur in the absence of fine fusion. Measured clinically, the sensation of depth elicited with large stimulus disparities, presumably activating only the coarse stereoscopic system, is qualitatively different from that obtained with the measurement of fine stereoacuity and single vision.

Studies of disjunctive eye movements have lent support to the notion of dual stereoscopic subsystems and have suggested that the operation of these two systems is complemented by a dual control system for vergence eye movements-one system which initiates such movements and the other which "carries them through to completion" and underlies fusional control (Westheimer and Mitchell, 1969; Mitchell, 1970). As Westheimer and Mitchell (1969) have demonstrated, stimuli which are presented on non-corresponding retinal coordinates elicit disjunctive eye movements, convergent or divergent, which are always appropriate to the sign of the stimulating disparity. For the initiation of vergence movements, retinal disparities can be very

large, up to 5-10°, and the visual images in the two eyes can be significantly different. They can be remarkedly dissimilar in shape, luminance, contrast or in their temporal onset in the two eyes, and still elicit the appropriate vergence eye movement. Nevertheless, although being sufficient for the initiation of a disjunctive eye movement, stimuli differing greatly in image similarity, do not permit its completion. Dissimilar stimuli, adequate for the initiation of eye movements, permit the subjective localization of objects in depth although they do not allow for the images to be subjectively fused. The mechanisms which underlie the initiation of vergence eye movements evoked with large stimulus disparities thus appear more closely associated with the system for coarse than for fine stereopsis.

Although maintaining the distinction between mechanisms for fine and coarse stereopsis, the data of Richards (1970) and Jones (1977) suggested a further subdivision of the coarse stereoscopic system into mechanisms for "near" and for "far" vision. In a psychophysical study, Richards tested the abilities of individuals to distinguish between targets presented at zero disparity ("the same depth as") and from .5° of crossed ("nearer than") and uncrossed ("further than") disparities. He found that a strikingly large proportion , about 30%, of randomly chosen, and apparently normal human subjects, were deficient in at least one of the 3 tasks. All combinations of stereoanomaly were detected and it was found that a person could, for example, have normal abilities for distinguishing crossed or uncrossed disparities, while at the same time be very poor at detecting opposite disparities. With a similar experimental design, these findings were later replicated by Jones (1977) who concurred with Richards on the frequency of stereoanomaly found in the population. However, in a

significant extension to the previous experiment, Jones additionally measured stereopsis in his subjects for retinal disparities of less than .5°--demonstrating that all of his subjects had normal stereoacuity when tested with standard clinical procedures. These data indicated that the systems for fine and coarse stereopsis were dissociable from one another and suggest that the stereoanomalies first described by Richards affected the coarse stereoscopic system only. Jones also examined the vergence movements of his subjects and found an incidence of oculomotor anomaly (20%) only slightly less frequent than perceptual stereoanomalies. Although the converse was not always true, a perceptual stereoanomaly was always found to be accompanied by a vergence anomaly. Not infrequently, vergence anomalies were present in a single dimension only, so that a subject could have normal divergence and anomalous convergence or vice versa. These data thus suggested that the 2 types of eye movements, divergence and convergence, were guided by independent control systems and that deficits could selectively effect only one of these components.

Disparity-sensitive neurons

In recent years neurophysiological investigations (Poggio and Fischer, 1977; Poggio and Talbot, 1981; Fischer and Kruger, 1979; Ferrester, 1981) have focussed on the identifiction of a neural correlate for the psychophysical effects described above. If indeed these observations can be attributed to the response characteristics of binocular visual neurons then there should be at least 3 distinct classes of disparity selective cells: one each for fine stereopsis,

crossed and uncrossed coarse stereopsis, and their associated vergence eye movements. This notion has been supported by the identification in both cat (Fischer and Kruger, 1978; Ferester, 1981) and primates (Poggio and Fischer, 1978; Poggio and Talbot, 1981) of cells which appear functionally capable of providing the substrate for the mechanisms of fine and coarse stereopsis.

The first to describe such cells were Poggio and Fisher (1977), in an experiment involving the use of awake, behaving monkeys, under conditions of normal binocular vision. The procedures utilized in this study had not only the virtue of approaching a natural visual situation, but also permitted a resolution in measurement which was not only far better than had previously been obtained but, was sufficient to reveal that stereoacuity in the non-human primate closely corresponds with that of its human counterpart. Additionally, this experiment indicated, in contrast to a previous study (Hubel and Wiesel, 1970), that disparity sensitive cells can be found in the primary visual cortex of the rhesus monkey. Recording from single neurons in both the striate and parastriate cortex, Poggio and Fischer found 2 major classes of disparity sensitive units. Cells in one group (tuned excitatory and tuned inhibitory neurons) were selective for very small stimulus disparities, averaging .2° around the fixation point, had symmetrical tuning curves and properties which would make them suitable for a system of fine stereopsis. The other group (near and far cells) responded over a broader range of stimulus disparities, had asymmetric tuning curves and were selective for stimuli either in front of or behind the fixation point. These units, with their less specific stimulus demands could provide for a mechanism of coarse stereopsis.

In a previous study of binocular interactions in the cat 17/18 border (Cynader et al., 1979) we have found that animals which had a section of the optic chiasm, and therefore received only ipsilateral input to each hemisphere, had units which showed substantial binocular activation, as well as disparity specific binocular interactions. It was obvious to us that the corpus callosum was an effective route for communication between the two visual hemispheres. However, examining the binocular interactions in split-chiasm cats has at least two serious difficulties. Firstly, since chiasm section alters the nature of binocular input to the lateral geniculate nucleus and the cortex on each side of the brain, the properties of callosal projection neurons are unlikely to be the same in split-chiasm cats as in normal cats. Secondly, studies of this type can only reveal those aspects of visual function for which the callosum is sufficient, rather than those for which it is necessary, and thus it was not clear from these data what the role of this projection would be in a relatively intact cortex. A recent approach to this question was that of Payne et al. (1980) who showed that after section of the corpus callosum, there was a dramatic drop in the number of units which could be driven equally by the two eyes, as well as a striking increase in the number of units (OD 1 and 7) which received excitatory input from exclusively one eye. These data suggested that the role of the corpus callosum for binocular connectivity in the opposite visual hemisphere was both substantial and necessary.

In the study of Payne et al., the responses of visual neurons were examined only under conditions of monocular stimulation. The present study was undertaken to examine the contribution of the

callosal projection to binocular interactions in cells along the vertical midline, and to determine if, in addition to the corpus callosum, there was evidence for other mechanisms of binocular convergence in cells with receptive fields located along the vertical midline. Binocular visual interactions were examined in single units from the border of area 17 and area 18 of cat visual cortex, and compared to responses from the 17/18 border of cats with unilateral lesions of the opposite visual cortex. Responses were examined with stimuli which moved in both the same (in-phase movement) and in opposite (antiphase) directions on the two retinae, movement which simulated motion toward or away from the animal or "motion in depth" (Cynader and Reagan, 1978; Poggio and Talbot, 1981). The results showed that stereoscopic processing depends on binocular inhibition in "monocular" neurons and that the corpus callosum plays an active role.

METHODS

In all experiments, subjects were normally-reared adult cats weighing 3-4 kilograms. For single unit recording, animals were initially anesthetized with intravenous Pentobarbital sodium, an endotracheal tube was inserted and paralysis was induced with intravenous Gallamine triethiodide. The skull was exposed and a small bone flap was removed over that part of the visual cortex representing the border between areas 17 and 18. Pentothal was discontinued at this point, Neosynephrine was applied to retract the nictitating membranes and the pupils were dilated with atropine. Contact lenses were chosen by retinoscopy to focus the eyes on a tangent screen 145 cm distant; the lenses contained 4 mm artificial pupils to decrease scattered light and increase depth of focus. A reversing ophthalmoscope was used to plot the two optic discs and areae centrales on the tangent screen. The vertical meridian for each eye was estimated to run through the center of the visual field perpendicular to the floor (Cooper and Pettigrew, 1980). Animals were initially paralyzed with a high dose (.5 cc/kg) of intravenous Flaxedil (Gallamine triethiodide) and then infused continuously with a mixture of Flaxedil (5 mg/kg/hr), D-tubocurarine hydrochloride (.5/mg/kg/hr) and 5% Dextrose in lactated Ringers (1cc/hr). During single unit recording, a level of light anesthesia was maintained by artificially ventilating the animal with a mixture of N_2O and O_2 (70:30) and intravenous anesthesia was discontinued. The animal's body temperature was held near 38° with a feedback-controlled heating pad, and end-tidal CO_2 was monitored continuously and kept near 4.2% by varying the rate of an artificial respiration pump. The cats were

usually maintained for a three day period. At the end of the experimental session, animals were perfused intracardiacally with saline, followed by a mixture of 10% formalin in a .9% saline solution. Brains were blocked in the electrode plane, removed from the skull and allowed to sink in 30% sucrose formalin. Forty micron sections were cut on a freezing microtome and stained with cresyl violet.

Approxminately one month prior to single unit recording, extensive lesions were made of the visual cortex in 5 animals. For surgery, cats were anesthetized with intravenous Alfathesin, fixed in a stereotaxic frame and a bone flap, 3 cm x 2 cm was cut through the skull. Cortex was removed by subpial aspiration, the bone flap was replaced, animals were administered subcutaneous Chloromycetin and returned to a cage for recovery. The lesions (see figure 9) included all of areas 17, 18 and 19 and extended laterally to include the crown of the suprasylvian gyrus and the Clare Bishop area.

Recording and unit classification

In normal cat experiments, a bone flap of approximately 5 mm^2 was removed with bone cutters under direct visual conrol. In an attempt to minimize the extent of dura left exposed after the craniotomy, a different procedure was used on the later-recorded decorticate cats. In these experiments, a small hole was drilled through the skull with a aid of dissecting microscope and less than 1 mm diameter of dura was exposed. In both cases, platinum iridium electrodes were advanced through the unopened dura with a hydraulic microdrive and responses of single units recorded from the 17/18 border. Action potentials were

amplified by conventional methods, monitored over a loudspeaker and displayed on a Tektronix D13 oscilloscope. Following isolation of a single unit, the receptive field was plotted with a hand projector and the following characteristics were noted; 1) the range of orientations over which the unit would respond 2) preferred orientation 3) direction selectivity 4) velocity preference 5) receptive field size 6) level of spontaneous activity 7) ocular dominance and 8) unit type. Moving and flashed stimuli, which included edges and light or dark bars of varied lengths and widths were used to plot receptive fields including edges and light or dark bars. Qualitative methods were generally employed to assess these response properties and quantitative analysis (see below) was reserved for the measurement of disparity sensitivity.

Simple and complex units were classified on the basis of subfield organization as originally described by Hubel and Wiesel (1962). Units were classed <u>simple cells</u> if their receptive fields could be divided into separate 'on' and 'off' areas and/or if responses to leading and trailing edges of moving light stimuli were evoked at different points in the visual field. Cells were classified as <u>complex</u> if both on and off regions and leading and trailing edge discharge regions were intermingled. Four other unit types were distinguished. A cell was classified as <u>hypercomplex</u> if it was selective for the length of a bar positioned along its preferred axis of orientation (Hubel and Wiesel, 1965). If a unit responded poorly or not at all to monocular stimulation but gave a vigorous response to binocularly presented stimuli it was called <u>binocular only</u>. A population of cells encountered gave only on or off responses throughout their receptive fields and these units were considered as

one type, on/off. Some cells did not fit clearly into any of the above categories, or had receptive fields which were difficult to plot, and such units were termed unclassified.

The ocular dominance (OD) of a unit was determined qualitatively and rated on a scale of one to seven according to the scheme of Hubel and Wiesel (1962). Units in OD group 1 receive excitation exclusively through the eye contralateral to the hemisphere under study, and units in higher OD groups receive successively more excitatory input from the ipsilateral eye. Units in group 4 were driven equally through the two eyes, and units in group 7 were excited exclusively by the ipsilateral eye.

Elongated stimuli of the optimum orientation presented at a velocity which evoked vigorous responses from the unit were employed for the assessment of direction selectivity. A unit was defined as "direction selective" if one direction of stimulus movement produced four times as many action potentials as movement in the opposite direction. If twice as many spikes were elicited by one direction of movement than the other, a unit was considered to have a directional preference. Non-directional cells responded with approximately the same number of spikes to either direction of stimulus movement.

Presentation of stimuli for quantitative analysis

Visual stimuli were projected from two similar but independent folded optical systems, each of which was arranged as follows. A slit of variable length and width was positioned in front of a condenser and illuminated by a 300 W tungsten lamp. A 9 cm achromat lens projected an image of the slit onto the tangent screen in front of the

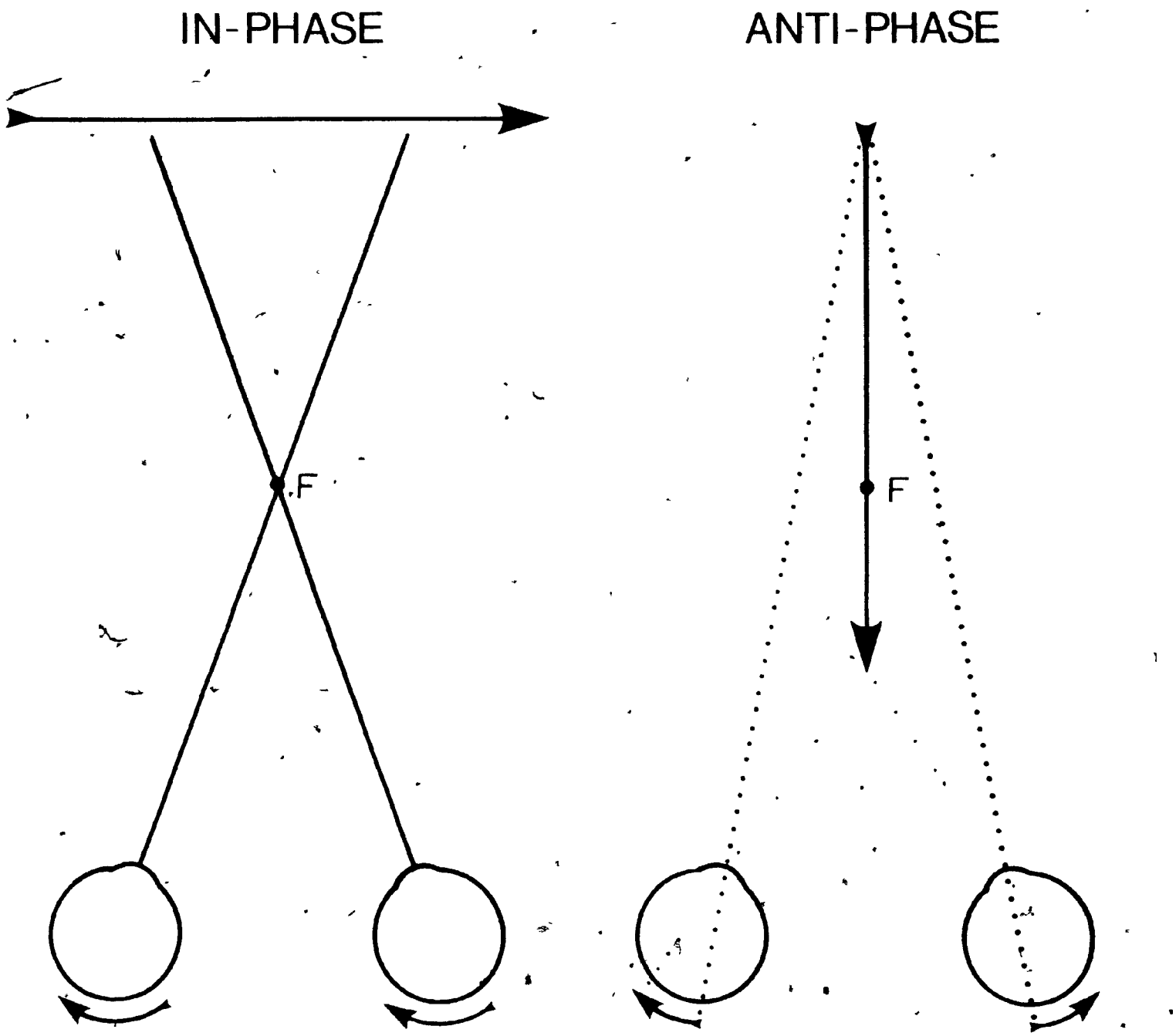
cat's eyes. Before reaching the screen, the beam was first reflected through 90° by a small front-surface plane mirror mounted on a galvanometer motor (General Scanning, type 300 PDT), then passed through a computer-controlled rotator, was again reflected through 90° by a large front-surface plane mirror and finally projected onto the tangent screen. By separating the receptive fields of the two eyes widely with a Risley prism, it was ensured that the receptive field of the left eye could be stimulated by only one of the two projected slits and that of the right eye by the other slit. The luminance of the stimuli was about 2.5 cd/m^2. Stimulus length, width, orientation and velocity were adjusted to match the preferences of the unit under study. The room and projection screen were diffusely illuminated by low-level tungsten light (0.34 cd/m^2). Computer-generated signals fed to the two galvanometer motors oscillated the small mirrors so as to move the bar images from side to side with a ramp wave motion and the positions of the bars were stabilized by positional feedback from the galvanometer. The image rotators were used to vary the orientation of the bars: the direction of movement was always perpendicular to the bars' orientation. The relative speeds and directions of motion could be controlled electrically as could their absolute speeds and repetition frequency.

Computer control of stimulation and recording

Stimulus parameters were set, and stimulus sequences initiated by typing appropriate instructions into a Tektronix model 4010 graphics terminal, which communicted with a PDP 11/34 computer. The terminal provided an on-line display of accumulated spike counts for each

-timulus condition. The time of each response after the initiation of stimulus movement was recorded for a fixed interval, the length of which depended stimulus velocity, and the data were recorded on DEC RKO 5 disks for later analysis. In any given experimental run, stimulus velocity was held constant in the dominant eye. The velocity, 5 deg/sec , 10 deg/sec, 20 deg/sec, 40 deg/sec, or 80 deg/sec which gave the best response from that eye was selected. Stimulus excursion was always sufficient to allow the stimuli to start and stop outside the receptive fields. Responses to stimuli moving in the same direction and the same speed in the two eyes (called in-phase motion) were compared with responses to stimuli moving in the opposite directions at the same speeds in the two eyes (called antiphase motion). The direction of stimulus motion was always the preferred direction in the dominant eye and was varied in the nondominant eye. As illustrated in figure 1, in-phase motion on the two retinae simulated sideways movement in the external world and antiphase motion simulated movement toward or away from the animal's nose. This comparison was carried out at seven different disparities separated by 1^{o} or 2^{o} intervals. Responses to 16 or 32 sweeps at each of the seven disparities were summed. Responses through the dominant eye alone were also measured as was the response evoked by stimulation of the nondominant eye alone in both directions of motion. This resulted in a total of 17 stimulus conditions which were individually interleaved. The relative speed with which these data could be collected (5 to 15 min) helped control response variability due to residual eye movement and fluctuations in response rate which occur over time. In the plots presented below, the disparities represented refer to relative disparities between the two receptive fields, and a value of 0

FIGURE ONE In-phase and antiphase stimulus motion. Disparity specific binocular interactions were measured with two types of stimulus motion. Stimuli presented in-phase moved across the two receptive fields in the same direction (figure 1, left), representing sideways motion in the external world. Stimuli presented in antiphase moved across the two receptive fields in opposite directions (figure 1, right), simulating motion toward or away from the animal's nose or motion in depth. Receptive fields were separated with a Risley prism and each eye was stimulated with independently controlled optical systems. In each of the two movement conditions, responses to zero disparity, 3 uncrossed and 3 crossed disparities were measured. Responses were also measured through the dominant eye alone in the preferred direction, and the nondominant eye alone in both directions, resulting in a total of 17 individually interleaved stimulus conditions. In figures 2,3 and 9, crossed disparities are represented with a plus sign, uncrossed disparities with a minus sign.

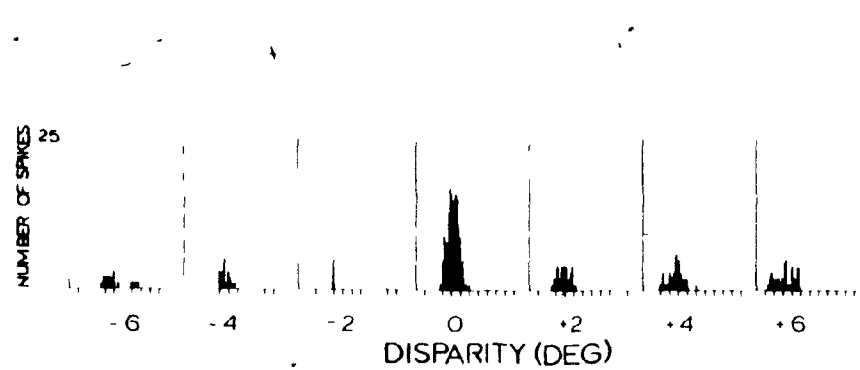

IN-PHASE
ANTI-PHASE
F
F

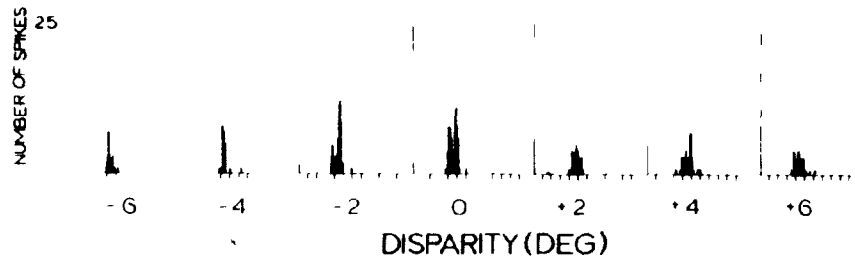
epresents the two centers. Since the use of moving stimuli confounds the variables of space and time (Gardner and Cynader, 1977; Cynader, Gardner and Douglas, 1978; see discussion) no distinction made will be made between "spatial" and "temporal" binocular disparities. The term "binocular interaction" refers to a nonlinear binocular response which is not presumed to be a response to any particular aspect of the binocular stimulus. Likewise, the terms "retinal disparity" and "disparity specific " are general terms which refer to either or both temporal and/or spatial disparities. Moving stimuli were chosen for the present experiment as they are more effective in driving many visual cells than are flashed stimuli, and it was important to sample at regular intervals from an unbiased population. Procedures for data reduction were chosen so that the responses of all units could be quantified and that comparisons could be made across as broad a population as possible. The principles derived from these data are believed to apply to both spatial and temporal mechanisms for stereoscopic depth perception.

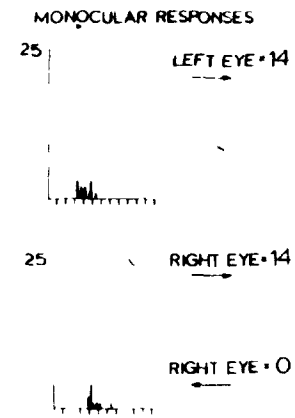
Data analysis

Responses to each of the 17 conditions of visual stimulation were summed and the summed responses and/or individual histograms were displayed on the graphics terminal. Hard copies were made using a Textronix 4610 hard copy unit or 4662 digital plotter. The plots were of the form presented in the top row of histograms in figure 2. In order to compare the degree of binocular interactions in the responses of single units, three indices, binocular inhibition, binocular facilitation and dynamic range, were constructed to indicate the

FIGURE TWO Data reduction. Method of data reduction is shown for a unit which displayed strong disparity specific binocular interactions to in-phase stimulus movement, and was relatively unselective for stimulus disparity with antiphase movement. This unit was recorded within 300 micra of the cortical surface, was direction selective and classified as binocular only. The two rows of post-stimulus time histograms illustrate the responses to 7 different disparities elicited with in-phase (top) and antiphase (bottom) stimulus motion. The number of spikes elicited at each disparity, in each movement condition, is shown in the summary histograms to the right. Responses through the dominant eye alone to the preferred direction and the nondominant eye alone to both directions of motion are also shown. As shown in the insert, the index of binocular facilitation (BF), binocular inhibition (BI) and dynamic range was calculated separately for each of the two movement conditions. There was one index each for combined binocular facilitation, combined binocular inhibition and combined dynamic range and its calculation considered responses across both in-phase and antiphase conditions. Although the procedures employed in quantifying the neuronal responses represent a considerable reduction in raw data, the results of the figures which follow show a high degree of internal consistency, and indicate that the observed effects are robust enough to withstand this degree of data reduction.







		IN-PHASE (IP)	ANTIPHASE (AP)
2-1	BINOCULAR FACILITATION (BF) $\frac{\text{MAXIMUM RESPONSE}}{\text{SUM OF MONOCULAR RESPONSES}}$	$\frac{214}{28}$ 7.6	$\frac{72}{14}$ 5.1
2-2	*BINOCULAR INHIBITION (BI) $\frac{\text{MINIMUM RESPONSE}}{\text{SUM OF MONOCULAR RESPONSES}}$	$\frac{6}{28}$ .2	$\frac{24}{14}$ 1.7
2-3	DYNAMIC RANGE $\frac{\text{BINOCULAR FACILITATION}}{\text{BINOCULAR INHIBITION}}$ 10	$\frac{7.6}{2} \cdot 38 \cdot 1 \cdot 37.0$	$\frac{5.1}{1.7} \cdot 3 \cdot 1 \cdot 2.0$
2-4	COMBINED BINOCULAR FACILITATION MAXIMUM INDEX OF BF, IP OR AP = 7.8		
2-5	COMBINED BINOCULAR INHIBITION MINIMUM INDEX OF BI, IP OR AP = .2		
2-6	COMBINED DYNAMIC RANGE $\frac{\text{COMBINED BF}}{\text{COMBINED BI}}$ $10 \cdot \frac{7.6}{2} \cdot 38 \cdot 1 = 37.0$		

degree by which the units' firing departed from that which would be expected on the basis of simple summation of the monocular responses.

The index of binocular inhibition (figure 2-1) for a given unit was derived separately for in-phase and antiphase stimulation by choosing the lowest value in the tuning curve and dividing that value by the sum of the monocular responses. For in-phase stimulation, the denominator of this ratio was the sum of the response evoked by stimulation of the dominant eye in the preferred direction and stimulation of the nondominant eye in the same direction. For antiphase stimulation, the denominator was the sum of the number of spikes evoked by stimulation of the dominant eye in the preferred direction and stimulation of the nondominant eye in the opposite direction. For a cell which shows little or no binocular inhibition this index will show a value of close to 1.0. Increasing degrees of binocular inhibition will result in successively lower values for this index. The index of binocular facilitation (figure 2-2) for a given unit was derived by choosing the maximum value of the disparity tuning curve and dividing it by the sum of the two appropriate monocular responses. This was done separately for in-phase and antiphase responses. Again, a cell showing little or no facilitation will display a value close to 1.0 according to this index, and cells with increasing degrees of binocular facilitation will display successively larger values.

In order to provide a measure of the degree to which the unit's firing could be modulated up or down by stimuli of different disparities, a measure caled the dynamic range was derived for each unit. This index represents the difference between the maximum and

minimum response observed over the 7 disparities tested, and was calculated by taking a ratio of the indices of binocular facilitation and inhibition described above (figure 2-3). As before, this was calculated separately for in-phase and antiphase stimulation. Thus, a cell showing substantial binocular facilitation (with a value of 4.0) and no inhibition (a value of 1.0) will achieve a dynamic range of 4.0, minus 1.0 for a total of 3.0. Likewise, a cell which lacks binocular facilitation (a value of 1.0) but displays marked inhibition (a value of .25) will also achieve a dynamic range of 3.0 , as will a cell which displays a moderate degree of both facilitation (a value of 2.0) and inhibition (a value of .5). The distribution of <u>combined</u> binocular facilitation (figure 2-4), binocular inhibition (figure 2-5) and dynamic range (figure 2-6) represents the minimum (inhibition) and maximum (facilitation) value obtained on these indices across the two movement conditions on these indices, and their ratio.

These measures are applied to the responses of a unit with very large binocular interactions in figure 2. In this figure, the 7 post-stimulus time histograms along the top show responses to different stimulus disparities tested with in-phase movement, and to their right, the summary histogram indicates the number of spikes elicited at each disparity. Beneath these, the monocular responses for each eye to the same direction of movement are also shown. To determine the degree of binocular facilitation, the maximum response, 214, was divided by the sum of the monocular responses, 28, to achieve an in-phase facilitatory value of 7.6. Binocular inhibition was calculated by dividing the minimum response, 6, again by the sum of the monocular responses, 28, for an in-phase inhibitory index of .21,

which was rounded to .2. In-phase dynamic range was determined by dividing 7.6 by .2 (max/min) leading to a value of 38, minus one, for a total of 37. In the second row of figure 2, the responses of the same unit to stimulation with antiphase motion at the same seven disparities as above are shown. For this cell, the degree of binocular interaction is less pronounced with antiphase motion than for in-phase motion. The value of the antiphase facilitatory index is 5.1, that for the antiphase inhibitory index is 1.7 and the antiphase dynamic range has a value of 3.0, minus 1, for a total of 2.0. To calculate the combined dynamic range for this unit, the larger of the two facilitatory values were divided by the smaller of the two inhibitory values for the cell. Since in this cell, these indices are both larger for in-phase motion than for antiphase motion, the combined dynamic range is equal in value (37) to the in-phase dynamic range.

RESULTS

I. Qualitative results in normal cats

In experiments on 10 normally-reared cats, 309 units with receptive fields along the 17/18 border were studied with qualitative methods. In 6 of these 10 animals, binocular interactions were examined quantitatively in 158 neurons. Electrode penetrations were perpendicular, or approximately so, to the cortical surface, and were confined to the region outlined in figure 9.' This area encompasses Horsely-Clarke stereotaxic coordinates, anterior 3.0 to posterior 3.0 and lateral 1.5 to 4.0 (Otsuka and Hassler, 1962). Most penetrations were made near AP 0.0, lateral 2-3, as previous experiments had shown that this region marked the 17/18 border. At the end of some representative penetrations, small electrolytic lesions were made (3 microamps for 3 sec, electrode negative) for histological reconstruction of electrode tracks.

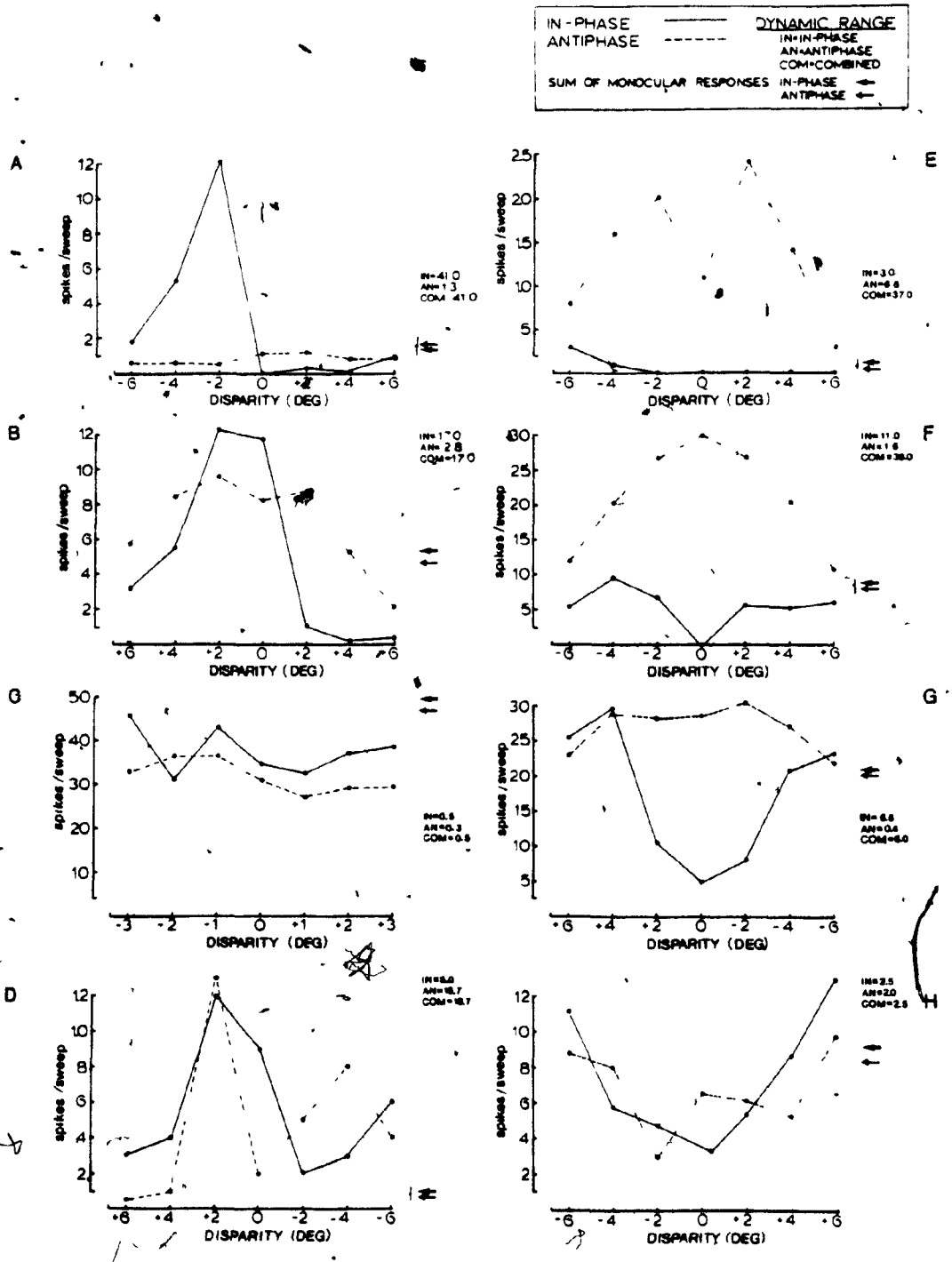
Quantitatively studied units had receptive fields which were usually located within 3^{o} of the vertical meridian and generally $5\text{-}10^{o}$ into the lower visual fields. In some penetrations, the response characteristics of the cells were similiar to those of area 18 units. Their receptive fields were relatively large ($5\text{-}8^{o}$), they responded with only a transient burst of impulses to a flashed stimulus, and they preferred very high stimulus velocities-frequently having no apparent high-end velocity cut off (Orban, 1977; Tretter, Cynader and Singer, 1975). Other units were more reminiscent of cells found in area 17, having smaller receptive fields, showing sustained responses to flashed stimuli and a preference for low stimulus velocities. Most

frequently however, penetrations near the 17/18 border contained units which showed a wide range of response characteristics. Some cells preferred low stimulus velocities, others very fast velocities, with a complement of sustained and transient responses to brief stimuli. Monocular receptive field sizes generally ranged between 2 and 5^{o} (86% of all units), while units with very small receptive fields (less than 1^{o}) such as those found often in the area centralis of area 17, and units with large receptive fields ($6-10^{o}$) were relatively uncommon (3% and 11% respectively). All six cell types described in the methods were represented in this sample. Nearly all cells recorded displayed orientation selectivity while 88% of this units showed direction selectivity or at least a directional preference. Many cells were binocularly driven as shown in the normal cat ocular dominance distribution of figure 8A.

Quantitative analysis of binocular interactions in normal cats

As described by previous investigtors, responses of cortical visual neurons to binocularly-presented stimuli vary with the disparity of the stimulus. Some units show binocular facilitation, others binocular inhibition, while others respond with facilitation at certain disparities and inhibition at others. In figure 3, a variety of such responses are shown. The response elicited at each of the 7 different disparities is illustrated for both in-phase (solid line) and antiphase (dotted line) movement, and can be compared with the 'predicted' binocular response (sum of monocular responses, arrow) for the two movement conditions. For reference, the value of the dynamic range index for each condition is indicated next to the graph.

FIGURE THREE Disparity tuning curves in normal cats. These disparity tuning curves illustrate the variety of binocular interactions seen among visual neurons of normal cats. Responses to both in-phase (solid line) and antiphase (broken line) stimulus movement are shown, and the arrows indicate the sum of the monocular responses appropriate to each stimulating condition. The responses of different units were characterized by binocular facilitation (A-in, D-in, E-an, F-an), binocular inhibition (F-in, G-in, H-in and an) or showed inhibition at particular disparities, and facilitation at others (B-in, D-an). Some units were insensitive to variations in stimulus disparity (A-an, C-in and an, G-an). As these tuning curves indicate, interactions to in-phase and antiphase stimulus movement could be similiar (B, C, D), different (A, E, G) or opposite in sign (F). The response characteristics of each of the units were as follows: (A) OD 6, direction selective, unclassified (B) OD 6, direction selective, simple (C) OD 4, direction selective, complex (D) OD 6, direction selective, on/off (E) binocular only, direction selective (F) OD 1, unclassified (G) OD 6, directionally preferential, complex (H) OD 5, direction selective, simple. The values of the in-phase, antiphase and combined dynamic range indices are noted to the right of each tuning curve.



III. Biology of Lobsters

A. Taxonomic Position

Homarus americanus is a decapod crustacean of the sub-order Reptantia, section Macrura. The Reptantia are generally adapted for a 'crawling' mode of existence, rather than swimming. The Macrurans however, have retained the 'large tail' which differentiates them from other Reptantians, and allows them to escape rapidly backward by flexing the abdomen ventrally (Barnes,1974; Cobb,1976).

B. Anatomy and Physiology

1. External Anatomy

The body of the lobster is divided superficially into 21 segments. The first 14 are fused into the cephalothorax, which carries the head, thorax, and first 5 of the 10 appendages. The last 7 segments are more clearly delineated externally from one another and form the abdomen. The body is covered overall by a chitinous exoskeleton, or cuticle, which forms a protective outer armour as well as providing a point of skeletal attachment and support for the muscles and other organs.

The head, although fused with the thorax, can be differentiated into 6 segments (which are representative of all arthropods), and their associated appendages. The first segment bears a pair of compound eyes set upon mobile,

jointed, stalks. The second carries a set of biramous antennae, the 'first antennae'. The 'second antennae', on the third segment are much longer and have only one filament. The fourth segment bears the toothed jaws (mandibles) that are required for crushing the lobster's food. On the fifth and sixth segments are the first and second maxillae, a pair of specialized appendages used to manipulate food and to 'drive' water out of the respiratory cavity.

The thorax also bears one pair of appendages per segment. The first three contain the first, second, and third maxillipeds, respectively; these appendages fulfill a primarily manipulatory function, however the third is sufficiently powerful to tear and mince food. The basal section of all three maxillipeds bears a thin flap of tissue (epipodite) which separates and protects the gills attached to the second and third. The fourth thoracic segment forms the point of attachment for the large pinching or crushing claws (chelipeds), which are diagnostic for Homarus species. As the animal grows these claws become asymmetrical, one developing into the larger crusher claw with the teeth fused into tubercles, and the other retaining a smaller, more slender appearance, and carrying a row of sharp teeth which are used in seizing prey. The next four segments all bear walking legs, each of which in turn bears a basal gill separator and a gill. The movements of the lobster serve to activate the gills and to stimulate the flow of water through the respiratory cavity located just beneath the carapace.

The abdomen bears a pair of appendages (pleopods) on all but the last segment. Those on the first segment are differentiated according to the sex of the lobster. In males the first pleopods form a troughlike structure adapted for the intromission of sperm during mating. The first pleopods in females are smaller and relatively undifferentiated. The second to fifth segments all bear the typical ancestral biramous appendages called swimmerets. Together with the modofied swimmerets of the sixth segment (uropods) and the dorsoventrally flattened posterior segment (telson), the swimmerets provide a tail fan which aids in aeration and rapid backward swimming.

The skeletal musculature of the lobster is characteristically striated, and therefore well suited for rapid movement of the body and appendages. The internal musculature is unstriated and adapted to slower, more rythmic contractions (Buchsbaum,1938; Barnes,1974).

2. Digestive System

The digestive system is composed of three main regions, only one of which is covered with an endodermal lining. The anterior and posterior regions develop as ingrowths of ectoderm, and are continuous with the cuticle which is shed when the animal moults. The anterior chamber is relatively unspecialized and serves largely for storage. The posterior is involved in sorting and straining. The stomach proper is equipped with a set of hard chitinous

teeth and numerous muscles which pulverize the food into minute particles. These particles are further reduced by the large digestive glands which are also specialized for absorption; this explains why a large and relatively active animal like the lobster can function so effectively with so short an intestine. (Buchsbaum,1938)

3. Circulatory System

The circulatory system is open, with a muscular heart suspended in a dorsally situated blood filled chamber (the pericardial sinus). Blood enters the heart through a series of ostia which are regulated by valves which prevent backflow during contraction. The blood leaves the heart through arteries which permeate the body tissue. The smallest branches dissolve into small blood cavities called sinuses. From here blood collects in the large ventral sinus where it is passed into the gills and back to the heart for recirculation. (Buchsbaum,1938)

4. Moulting Physiology

Although growth in lobsters is an ongoing process, size increase is restricted to periods of intermittent moulting or ecdysis. Ecdysis begins with a split in the connecting membrane between the abdomen and carapace. Muscles relax, fluid is withdrawn from the tissues, the animal flexes into a V position laterally, and extracts itself from the discarded cuticle. Water is reabsorbed once

the animal is free and the lobster expands to a size determined by the dimension of the new carapace. Hardening of the cuticle occurs with varying rapidity. (Cobb,1976)

The physiology of this process is complex and incompletely understood for Homarus americanus. It is thought to be a bihormonal process regulated by the antagonistic interaction of two major hormones (Aiken,1977). Moult-inhibiting hormone, produced in the neurosecretory centers of the eyestalk, suppresses the induction of premoult conditions (Cobb,1976). This is balanced by the release of some external or endogenous moulting cue which causes the suppression of moult-inhibiting hormone and allows the production of moult stimulating hormones (Aiken, 1977), including ecdysterone. Although ecdysterone does not induce all premoult conditions, it is thought to regulate cuticle formation (Cobb,1976).

The temporal components of the moult cycle have been narrowly categorized, however it is sufficient here to list the 5 major stages. These include: A, the newly moulted and soft shelled stage, covering approximately 1½ to 2 percent of the entire cycle, in which water is reabsorbed and the exoskeleton becomes mineralized; B, the paper shell stage, covering approximately 8 percent of the cycle, in which endocuticle secretion and new tissue growth are initiated; C, the hard stage, occupying roughly 65 percent of the cycle, during which the major tissue growth and accumulation of organic reserves occurs, and; D, the proecdysis or

premoult stage, covering approximately 25 percent of the cycle, and during which the major physiological changes associated with moult induction occur. The final stage, E, is the moult stage proper. (Aiken,1977)

5. Nervous System

The relatively large brain of the lobster is located anteriorly, above and just behind the eyes. A pair of large nerves pass ventrally, around the oesophagus and digestive tract, and unite below to form the suboesophageal ganglion. The paired nerve cord passes posteriorly in the ventral position, forming enlarged ganglia in most segments. (Buchsbaum,1938; Cobb,1976)

6. Sensory Physiology

"Sensory physiology cannot readily be isolated from behaviour." (Ache,1976) Behaviour depends on reception of external stimuli, endogenous rhythms, and neurobiological development and processing. Each of these factors governs how an individual will respond to its environment (Atema, 1976).

The sensory capabilities of the lobster are distributed among specialized sensory cells. These cells generally form aggregates, or distinct organs, which are peculiarly adapted for monitoring the animal's internal and external milieu. The organs are characteristically receptive to specific sensory modalities; this allows for the most efficient

stimulus transduction and signal integration in afferent neural pathways. The sensory capabilities are therefore considered here with respect to their mode of reception. (Ache,1976)

(a) Chemoreception

The principal 'known' sites of chemoreceptivity in the lobster are the two sets of antennular filaments, the dactyls of the walking legs, and the mouthparts. These appendages are further differentiated into low or high threshold receptors, according to whether the stimulus is distant or requires physical contact. This discussion is limited to behaviourally relevant stimuli, thus avoiding the gray area of membrane physiology, and pH and osmotic regulation (Ache,1976).

The lateral filament of the first antennae (or antennule) with the distinct distal tuft of aesthetasc hairs is regarded to be the principal site of low threshold chemoreception in the crustacea (Hazlett,1971). Each hair is basally innervated by a tight sheath of neuron somata. Molecules are thought to actually penetrate a pore at the distal end of the hair to achieve contact with dendritic receptors (Laverack & Ardill,1965) in Panulirus argus, but Homarus aesthetascs appear to be covered over the distal third of the shaft by a continuous 'spongy' cuticle, totally devoid of pores. Electrophysiological studies suggest that lateral filament receptors are sensitive to low molecular weight components, particularly of potential feeding stimuli

in aqueous solution (Ache et al,1976). The neural action potential appears to be proportionally related to the concentration gradient of amino acids and amines. It is thought that the 'flicking' action characteristic of lobster antennules is a device for monitoring changes in this stimulus concentration (Ache,1976). Primary antennular sensory neurons probably project to the olfactory lobe of the brain by way of the antennular nerve (Maynard,1966).

Higher threshold receptors are situated along the length of the filament of the second antennae. There is evidence to suggest that while antennal receptors are sensitive to osmotic variations in the ionic concentration of stimuli, these delineations are of a strictly quantitative rather than a qualitative nature (Tazaki,1975). Other high threshold or 'contact' chemoreceptors are clustered in the dactylopodites of the walking legs. Receptors occur in discrete rows of fine, branched hairs, starting some distance proximally to the dactyl's tip. They respond to simple chemical molecules such as trimethylamine and betaine (Laverack,1963a; Shelton & Laverack, 1970); these compounds induce the long latency and long duration neural response which is characteristic of chemoreceptors (Ache,1976).

The chelate first periopods are covered with branched hairs along the inner face. These hairs, which are thought to respond to similar stimuli to the dactyl hairs (Shelton & Laverack,1970), may account for the proposed potentiation of chemoreception by simultaneous mechanical stimulation

(Hazlett,1971). "The hair position subjects them to bending when an object is grasped in the chelae which exposes new faces of the hair to environmental stimuli, a phenomenon ... likely to aid in stimulus detection." (Ache,1976)

The third maxilliped and other mouthparts are covered by rows of pectinate hairs which have been shown to respond to chemical stimuli (Shelton & Laverack,1970).

(b) Photoreception

Although the lobster lives in a severely light restricted environment, they are highly visual organisms (Ache,1976). Their somewhat surprising acuity can be attributed to the possession of paired compound eyes, which are functionally and morphologically related to those of insects and other arthropods.

The compound eye of Homarus consists of 12,000 functional units, or ommatidia (Nunnemacher,1966). Each is an elongated structure bearing 7 pigmented retinular (photoreceptor) cells, sheathed all around by a sleeve of cells containing a screening pigment. Light enters through the transparent cornea which covers each ommatidium, and is directed axially through a crystalline cone to the photoreceptive element or rhabdom, centered in the retinular cluster. Under conditions of high light intensity, the screening pigment migrates to surround the individual ommatidia (Ache,1976). This response moderates the sensitivity but does not alter acuity, and allows the lobster to accomodate with great flexibility to a

predominantly nocturnal life style (Arechiga & Atkinson,1975).

The optic nerve is an intricate network of functionally diverse neurons (Wiersma & Yanagisawa,1971). The lobster eye is particularly well adapted for the detection of motion, although the ability for form recognition at the level of the optic lobe may be limited (Ache,1976).

There is some evidence to suggest the existence of a low level of caudal photosensitivity, modulated by photosensitive regions in the last abdominal ganglion. It is probably of little behavioural significance (Kennedy,1963).

(c) Mechanoreception

External response to mechanical stimulation is based on innervated hairs or bristles. Mechanically induced deformation of hair or membranous socket disrupts the electrical potential of the neuron and produces excitation (Ache,1976). Mechanoreceptors are classified according to the nature of the stimulation rather than by their morphological situation.

Direct contact or 'tactile' receptors are located over a wide area of the cuticle. Characteristic of this type of receptor are the highly branched 'hair fan' organs of the chelae and anterior carapace (Laverack,1963b). These organs are directionally sensitive and respond phasically along two neurons to transient displacements (Laverack,1962b). Related 'hair peg' organs respond similarly to maintained displacement (Laverack,1962a).

Mechanoreceptors which are responsible for maintaining

the spatial orientation, or equilibrium of the lobster, line a statocyst chamber in the basal segment of each antennule. Unlike the tactile receptors, each hair is innervated by a single neuron (Ache,1976). The physical displacement of fluid and the calcified statolith are detected by four types of specialized receptor; one detects the absolute position about the transverse axis, another the direction of approach to this position,a third responds to angular displacement about any axis, and the fourth responds only to substrate borne vibrations (Ache,1976; Cohen,1955). Each hair monitors a position specific component of the overall gravitational displacement, and the total input is simultaneously interpreted in the brain (Cohen,1960).

Appendage displacement, or proprioception, is monitored by mechanoreceptors in contact with the elastic chordotonal strands that span each joint. In walking limbs an "accessory flexor muscle", associated with the chordotonal fibers, regulates the reflexive adjustment of the limb (Ache,1976). Chordotonal type fibers are thought to monitor and regulate the forces inducing limb autotomy (Clarec et al,1971).

There is no evidence to suggest the presence of thermoreceptors in the lobster (Ache,1976), and "Receptors sensitive to ... biologically relevant acoustic stimuli remain undescribed." (Ache,1976)

C. Life Cycle and Behaviour

1. Reproduction

Mating occurs in the early post-moult female when the shell is still soft. Sperm, usually from a single male, (Hedgecock et al,1975) can be stored for prolonged periods of time, up to 15 months, until the eggs are laid and fertilized (Cobb,1976). Temporary pair formation is thought to act as a deterrent to multiple mating, but half sib families have been detected in the progeny of a single female (Nelson & Hedgecock,1977). (This event is thought to be of insufficient frequency to significantly bias the results of genetic analyses based on the assumption of full sib relationships. In any case, the error would be on the conservative side, resulting in an underestimate of the variance due to additive genetic effects.)

The eggs (7,000 to 80,000 depending on the size of the female) are extruded from the oviduct, pass ventrally along the abdomen and over the sperm receptacle where they are fertilized, and are then cemented firmly to the swimmerets (Cobb,1976). This position provides for the maximum protection and aeration of the developing eggs. The eggs hatch after 10 to 11 months of gestation; the liberation of the larvae occurs as a result of internal hydraulic pressure caused by a gradual uptake of water during embryonic development. The newly emerged and as yet immobile larvae moult almost immediately, and begin their life as freely

.swimming plankton (Cobb,1976).

2. Larval Stages

The following account of larval development and behaviour is taken entirely from the classic, and as yet unsurpassed account of F.H. Herrick (1911).

(a) First Larva

The lobster emerges from the egg capsule, usually at night, and proceeds directly into its first moult. At this point it is free swimming, and although it rises and sinks with some regularity, it is photopositive and remains relatively near the surface throughout its pelagic existence.

The first larva is roughly 8mm in length, and although the body has already assumed a segmented form consistent with that of the adult, functional appendages are lacking on the abdomen. The head bears a large rostral spine and inordinately large compound eyes; biramous swimming legs accur along the carapace, and a triangular telson completes the abdomen. The general anatomy and nervous system have already acquired the general relations and functions of the adult animal.

The most striking habits of the larvae include their incessant activity and voracious appetites, their highly developed instincts of predation and aggression (which may result in cannabalism when food is short and larval density is high), the temerity so uncharacteristic of later stages,

their acute vision and predatory precision, and their lack of discrimination with respect to the object of prey.

The mutual destructiveness of larval lobsters in crowded conditions is a matter of concern for animal husbandry. The attacker attempts to mount the victim dorsally and to nip into the abdomen at its point of junction with the carapace. This technique is facilitated by the sharp prehensile tips of the future chelipeds. The incapacitated animal is then carried along by the victor and devoured.

"The natural food of the larval lobster consists of minute pelagic organisms, whether animals or plants, which through their own movements or their lightness remain suspended near the surface, such as diatoms and other protophytes, copepods, the larvae of crustaceans, echinoderms, worms, and mollusks, the floating eggs of fishes, and, in fact, any member of the pelagic fauna which comes into their zone and is not too large for them to master." (Herrick,1911) This must account for their incessant activity, which is required to bring them into contact with their suspended prey. Their lack of discrimination however, leads them to feed on even highly inappropriate organic and inorganic material.

(b) Second Larva

The second moult produces some characteristic changes in the larval morphology, notably the slightly increased size (approximately 9mm) and the presence of 4

pairs of swimmerets on the second, third, fourth, and fifth abdominal segments. These new appendages lack swimming hairs and are functionally incomplete until the fourth larval stage.

(c) Third Larva

The third larval stage is distinguished by its relatively larger size, the presence of a completed tail fan (although the telson is still disproportionately larger than the uropods), and the less rudimentary nature of the abdominal swimmerets.

In habits, colour, and overall morphology however, the first three stages show no striking dissimilarity.

(d) Fourth Larva or 'Lobsterling' Stage

From the first stage onward there is a progressive reorientation of the appendages - maxillae, maxillipeds, and pereiopods - in the forward direction. This reorientation is completed with the fourth moult when the appendages acquire their adult condition. Failure of the large chelipeds to rotate at the fourth moult is common, and results in a permanently deformed and functionally incapacitated animal (personal observation).

In form and habit the fourth stage lobster is strikingly different from the preceeding stages, and quite closely resembles the adult animal. The accessory swimming exopodites of the thoracic appendages have all but disappeared; in spite of this the animal has acquired a functional agility and precision which greatly surpasses the

reel and jerk of earlier stages. This is due in large part to the development of the proprioceptive organs at the base of the first antennae.

Fourth stage animals usually retreat to the bottom by the end of this period, hiding or burrowing under mud and stones. This conversion to a benthic habit is however, not infrequently delayed until the fifth stage. The instinct of fear associated with the burrowing tendency first appears at this time.

(e) Fifth Stage

Habits are modified only slightly beyond this stage, where the solitary and shelter seeking behaviour is established and continued throughout life. "As the lobster increases in size it becomes bolder and retires farther from the shore, but it never loses its instinct for digging nor abandons the common habit of concealing itself when the necessity arises." (Herrick,1911)

3. Juvenile and Adult Behaviour

(a) General Behaviour

All clawed lobsters appear to be strongly nocturnal. Homarus americanus ventures from its burrow only when the light intensity has diminished below a certain measurable point (Cobb,1977), and its response to strong light is highly photonegative (McLeese & Wilder,1958).

There is no conclusive evidence to suggest the presence of endogenous rhythmicity in Homarus americanus (Cobb,1977).

Actographs showed a weakly rhythmic activity under conditions of total darkness, which correlated well with the pattern observed under a light - dark cycle. Total light produced arhythmic activity (Cobb,1977).

An increase in activity was noted by McLeese and Wilder (1958) with a temperature increase from 2° to 10°C. Artificial increases beyond this point produced no significant change.

Social conditions are known to affect the level of activity in Homarus americanus. Communally reared animals show lower levels of activity than their individually reared counterparts (Zeitlin-Hale,1975). Dominant animals spend a proportionately greater time in shelters and are less inclined to perform nonagonistic patterns of behaviour (walking, grooming, etc.) than are their subordinates (Cobb & Tamm,1975; Cobb,1977).

Nonlocomotor behavioural rhythms have been only sparsely examined (Cobb,1977). Larval hatching is nocturnal (Ennis,1975), and juvenile H. americanus moult during the day (Tamm & Cobb,1976).

Some behaviours vary according to more long term rhythms. Egg laying occurs in mid to late summer and hatching follows the next spring. Feeding rate is also seasonal, being higher in summer and fall, and declining throughout the winter and spring (Ennis,1973). These rhythms are no doubt regulated by the seasonal variation in water temperature (Cobb,1977). Food intake is known to decline throughout the

intermoult period, and food selection, activity, and aggression are all related to the cycle of ecdysis (Cobb, 1977).

(b) Agonistic Behaviour

(i) Description

"Behaviour can be roughly equated with outwardly visible motor patterns and responses." (Atema,1977) Such motor patterns can be analyzed and categorically described as a particular behavioural ethogram. Such an ethogram has been defined for the agonistic behaviour of H. americanus by J.C.E. Scrivener (1971), incorporating 16 specific behaviour patterns. A brief summary of these patterns will be considered here.

Agonistic behaviour can be defined as "any sort of adaptation which is connected with a contest or conflict between two animals, whether fighting, escaping, or freezing." (Scott,1958) Agonistic behaviour therefore, incorporates all aspects of aggressive behaviour, including flight.

'Meral spread' forms an almost universal component of agonistic behaviour in Homarus americanus, and may lead to either aggression or flight. The animal stands elevated on its walking legs with cephalothorax slightly flexed and the abdomen fully extended. The chelae are raised and rigidly spread with the long axis directed towards the opponent. The antennae are firmly pointed up and away from the midplane of the body. Meral spread is a position of threat,

a behavioural attitude which is considered to be the "direct result of a conflict between attack and escape tendencies, when neither can find separate expression." (Manning,1967) The effectiveness of this gesture may determine whether or not the opposing individuals progress to fighting or whether the subordinate animal retreats without a contest. It is probably an adaptive mechanism to prevent needless and energy expensive battles in the wild.

A lobster may indicate its interest in fighting by 'following' a retreating opponent. The abdomen is carried in a fully extended position with the tail fan splayed open. The chelipeds are elevated and frequently in a meral spread position. The antennae are often held at a perpendicular angle to the main axis of the body. If the opponent is neither retreating nor responding aggressively, the following attitude is termed 'approaching'. A more rapid approach with the chelae in the meral spread position is defined as 'rushing'.

'Pushing' occurs when two animals in direct contact stand high on their walking legs and push against each others chelae. The body is often flexed, with the abdomen extended and tail fan open. This behaviour is often coupled with 'boxing', where one animal withdraws the chelae and punches its opponent. 'Scissoring' occurs when both chelae are spread and brought together sharply, in front of, or directly against the body of the opponent. 'Antennae whipping', a rapid lashing of the second antennae, may

accompany any of the previously discussed behaviours.

When approached from the rear, a highly aggressive animal will respond with 'rapid turning'. The animal rotates through an angle of 180° in less than a second, and arrives in a face to face position with its opponent in an attitude of full meral spread.

Avoidance behaviour includes characteristic retreat positions such as 'backing', which occurs with the abdomen partially curled under and the antennae straight and parallel with the long axis of the body; 'abdomen flexing', which carries the lobster vigourously up and away from the bottom and its opponent; 'jumping'; 'walking away'; 'running away'; and 'side-ways running away'. The names are fairly descriptive and for the most part self-explanatory.

(ii) Environmental Components of Aggression

Cyclic changes in aggressive behaviour over the lobster's moult cycle are well recognized. Animals in the midphases of proecdysis (D), appear to be considerably more aggressive than those in intermoult (C), whereas those in intermoult are more aggressive than animals in early or late proecdysis. Animals in early postmoult, (A) and (B), are generally quite submissive (Cobb,1977;1978). Crowded social conditions cause juvenile lobsters to prolong the intermoult period by as much as 32 percent in earlier stages and 71 percent in later stages (Cobb,1974).

Communal housing conditions have been shown to attenuate aggressive behaviour in juvenile Homarus

americanus when compared with individually reared laboratory animals (Dunham,1972; Hoffman et al,1975). Although the specific conditions which mediate this response are unknown, it has been suggested that experiential factors, presumably through the establishment of social hierarchies and pheromonal signals, may be responsible (Dunham,1972).

Elevated temperatures appear to induce higher levels of agonistic activity (Hoffman et al,1975).

D. Genetics

It is impossible to make a particularly protracted statement regarding the biochemical or molecular genetics of Homarus americanus. The bulk of analysis to date has been directed toward specific ecological and evolutionary considerations, particularly those relating to the genetic potential for long term adaptability to aquaculture (i.e. domestication) (Nelson,1977).

A fairly extensive electrophoretic survey of protein variation, involving 8 populations and 44 loci, was conducted by Tracey et al (1975) for H. americanus. They found that "Homarus americanus appears to be genetically homogeneous over the range that we have sampled at 43 of 44 loci." Only the malic enzyme locus exhibited a sufficient degree of interpopulation variation to suggest that inshore and offshore populations may be reproductively isolated. The average proportion of heterozygous loci per individual was only 3.8 percent. It has been suggested that this acute lack of variability may stem from the relatively " ... greater size, mobility and perhaps degree of homeostatic control, ..." of decapod crustaceans compared with other other invertebrates; they therefore " ... rely less upon structural gene variation and more on other behavioural and physiological regulation (Selander & Kaufman,1973) or upon regulatory gene variation to achieve population consonance with environmental variation."

(Nelson,1977). According to Ayala et al (1975), there is an inverse correlation between certain kinds of environmental instability, particularly with respect to resources, and the degree of genetic variability in marine organisms.

An alternative explanation for the observed level of heterozygosity may be inbreeding depression, a result of population subdivision and reduction of effective population size by overfishing (Tracey et al,1975). Between 70 and 90 percent of all legal size inshore lobsters are removed each year (Cobb,1976).

There is some potential for increasing the genetic variability through hybridization of americanus with the European species, Homarus vulgaris. This is a solution which offers a limited improvement at best; the genus Homarus dates from the Cretaceous period (Glaessner,1969), and its two extant species have diverged relatively little during the intervening geological ages. The average genetic identity between the species is 0.902 (Hedgecock et al,1977).

Quantitative genetic research in lobster populations has not been particularly extensive. Growth rate variations have been assessed in studies by Hedgecock et al (1976), Hedgecock and Nelson (1977), and Fairfull and Haley (1981), and heritability estimates for this trait have been consistently high. The interaction of growth rate with environmental variation was also analyzed (Fairfull and Haley,1981; Hedgecock and Nelson,1977), and genetic variation was found to persist across treatments. Genetic variability

for mortality has been found for individually reared juveniles, however, environmental and experimentally induced trauma were found to form a large component of the overall estimate of heritability (Fairfull and Haley,1981).

Empirical Methods

I. Rearing and Maintenance

Thirty gravid female lobsters were sampled from an offshore population (Brown's Bank) in the spring of each of two successive years. They were housed in a communal holding tank with a constant flow, ambient source of sea water, and fed intermittently with various species of waste fish. At intervals corresponding with the availability of space, each female was introduced into an individual hatching tank infused with freely flowing, nonrecirculating, degassed sea water, maintained at a temperature of 18°C (see fig.#1). Hatching usually followed within 1 or 2 weeks.

First stage larvae were collected in a trap on the front of the tank and removed to well aerated 'Hughes' pots (see figs. #2 and #3), also maintained at 18°C, where they were reared until they reached 4th stage. The larvae were fed freshly hatched brine shrimp nauplii, Artemia salina.

Although aeration kept the waters fairly well agitated, and feeding was regular, a certain amount of cannibalism was inevitable. It could therefore be argued that all families underwent a certain amount of preselection for aggressive behaviour. This may in fact be true, particularly as families were housed separately; selection was between full sibs and would be expected to primarily depress the within family variation. Therefore, the potential for preselection under these rearing conditions must be

Figure #1: Hatching Tank With 'Berried' Female; newly hatched larvae are washed into the 'trap' on the front of the tank, and transferred to 'Hughes' pots (fig. #2 and #3).

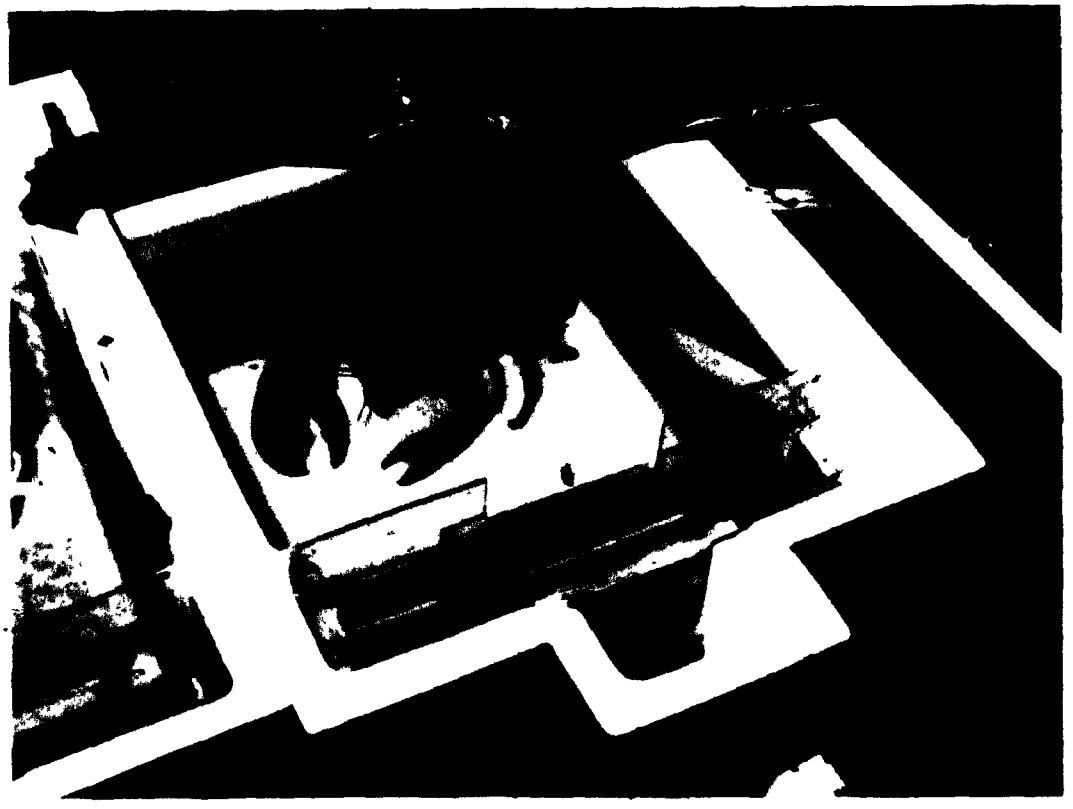


Figure #2: 'Hughes' Pots Containing Newly Hatched Lobster Larvae; larvae are kept in circulation by air stones in the bottom of the pots.

Figure #3: 'Hughes' Pot Containing Newly Hatched Lobster Larvae.

recognized; it is however, unlikely to be of serious consequence in the estimation of heritability (given the assumption of equal family size).

As juveniles reached the 4th stage, 30 (20) from some families, and 60 (40) from others (numbers without and within brackets refer to values for population #1, hatched in the spring of the first year, and population #2, hatched in the spring of the second year, respectively) were randomly selected and removed to the second stage of the experiment. Thirty juveniles per family (20) were introduced into individual containers constructed from 3½ inch diameter sections of PVC tubing; the sides were cut away leaving only 2 symmetrical supporting struts, and the container was covered overall with fine fiberglass screen (see fig #4). Each was identified and immersed to approximately 3/4 of its length in one of 10 troughs supplied with free flowing, nonrecirculating, degassed, 18°C sea water (see fig. #5). Three (two) members of each family were randomly placed within each trough. Containers within troughs were frequently rotated to prevent possible effects due to position. In this manner a total of 14 (18) families was introduced into the experiment.

For 5 (9) families, an equal number of juveniles were released to live freely in the bottom of one of the 10 troughs. These animals represent the 'group reared' component of the experiment. Spatial limitations prevented the group culture of juveniles from all families.

Figure #4: Container for Individually Reared Juveniles of Homarus americanus .

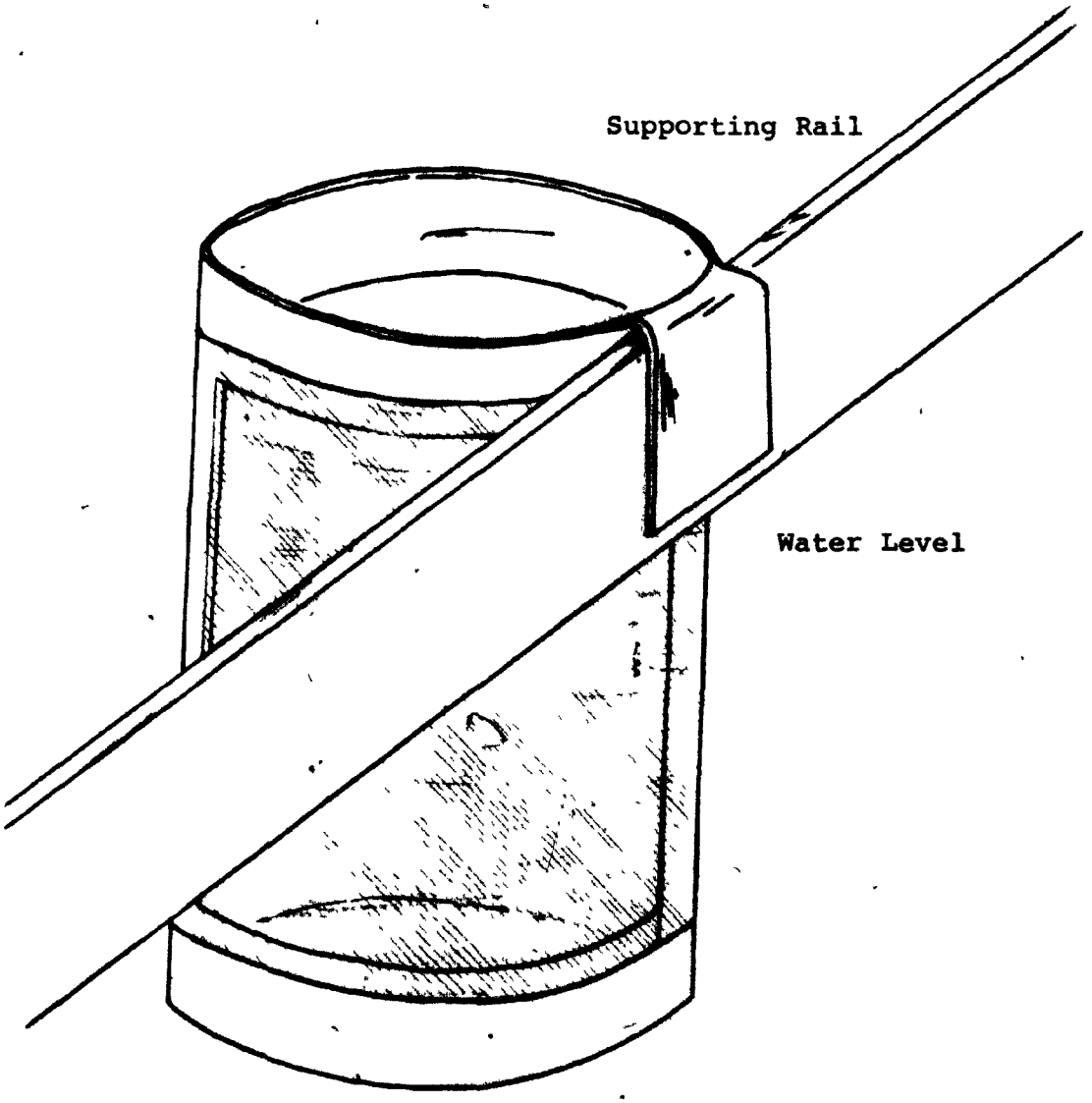


Figure #5: Housing System for Individually Reared Juvenile *Homarus americanus*.

The light regime was mechanically maintained (clock timer) at 12 hours of light alternating with 12 hours of dark.

Each animal was fed a prescribed amount of frozen brine shrimp every second day, and checked regularly for cleanliness and mortality. Moulted cuticles were left in the cages to be consumed by the soft shelled juveniles. Unused brine shrimp was removed prior to additional feeding. In the first set of group rearing experiments, that is for animals in population #1, the juveniles in troughs fed entirely as scavengers. No food or shelter was provided directly. In population #2 the communally reared juveniles were fed an amount of food per lobster equivalent to that of their individually reared sibs. Shelters were also provided (made from 1 inch PVC pipe cut in 2 inch lengths and open at both ends) of an appropriate size and number to accommodate ½ of the animals in each trough.

Each trough was emptied once in every 10 days for cleaning, and the animals were provided with newly sterilized containers. At the same time the communally reared animals were counted and weighed.

Individually reared animals were weighed 7 days (4 days) after entering 4th stage and at approximately 2 week intervals thereafter. At later stages the time of weighing was adapted to coincide with periods of experimental testing. Each animal was gently blotted on a piece of dry paper towel and then weighed using a Mettler balance (precision

0.0001 gram).

Each animal was reared through approximately 14 moult stages; the measurements terminated and the animal was removed from the experiment after reaching 150 days (measured from the date of entering 4th stage).

II. Behavioural Experiments

After approximately 60 days, the animals were introduced into the experimental chambers for the initial period of behavioural testing. (The age of juveniles for the second set of tests, that is, for population #2 in the second year, was far more variable. This was due to an urgency imposed by the increased level of mortality. The death rate for population #2 was unacceptably high in most families at 60 days, and earlier testing was employed to improve the degrees of freedom for within family variability. The probable reason for this elevated mortality will be discussed in a later section.)

The measurements were confined within families as interfamily testing introduces a number of potentially serious mathematical and practical difficulties. The range of family size and age, the inability to isolate the contribution of individuals, and the confounding of statistical variation were some of the difficulties considered. The use of a standard control was impossible due to the habituation which occurs as a result of repeated short term encounters. The basic unit of measurement was

defined as a 5 minute bout rather than the response of a single individual.

The experimental apparatus consisted of a solid plexiglass frame with the bottom perforated at regular intervals to allow the circulation of water. The holes were covered by fine fibreglass screen. Interspersed along the length of the frame were a series of sliding plexiglass partitions covered with black linen tape to prevent communication between chambers. The end partitions, which formed part of the frame, were extended beneath the chamber in order to elevate the entire apparatus and to encourage circulation. For an appreciation of the relative size of lobster and chamber, please refer to figure #6.

Figure #7: Experimental testing chamber for aggression experiments with juvenile Homarus americanus.

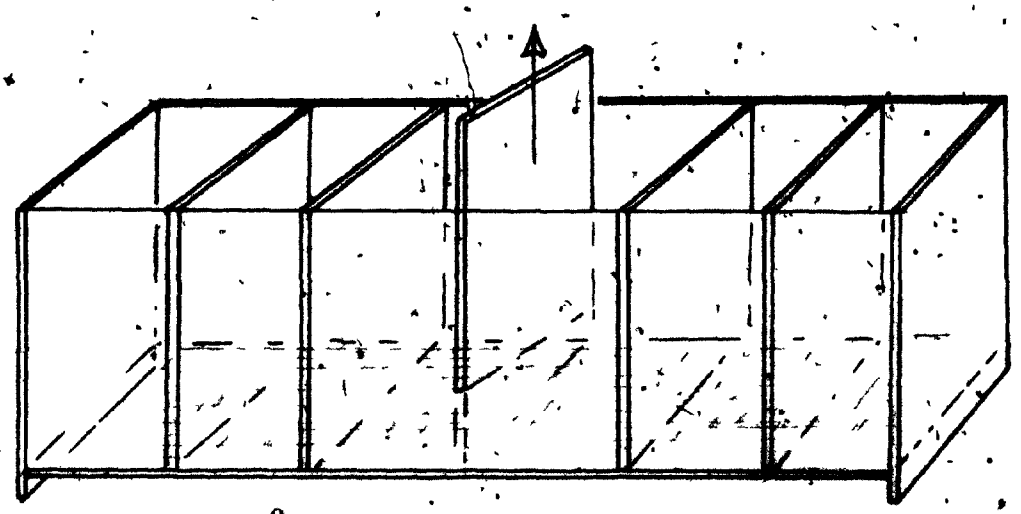


Figure #6: Juvenile Homarus americanus in Behavioural Testing Chamber Before Removal of Interchamber Partition.

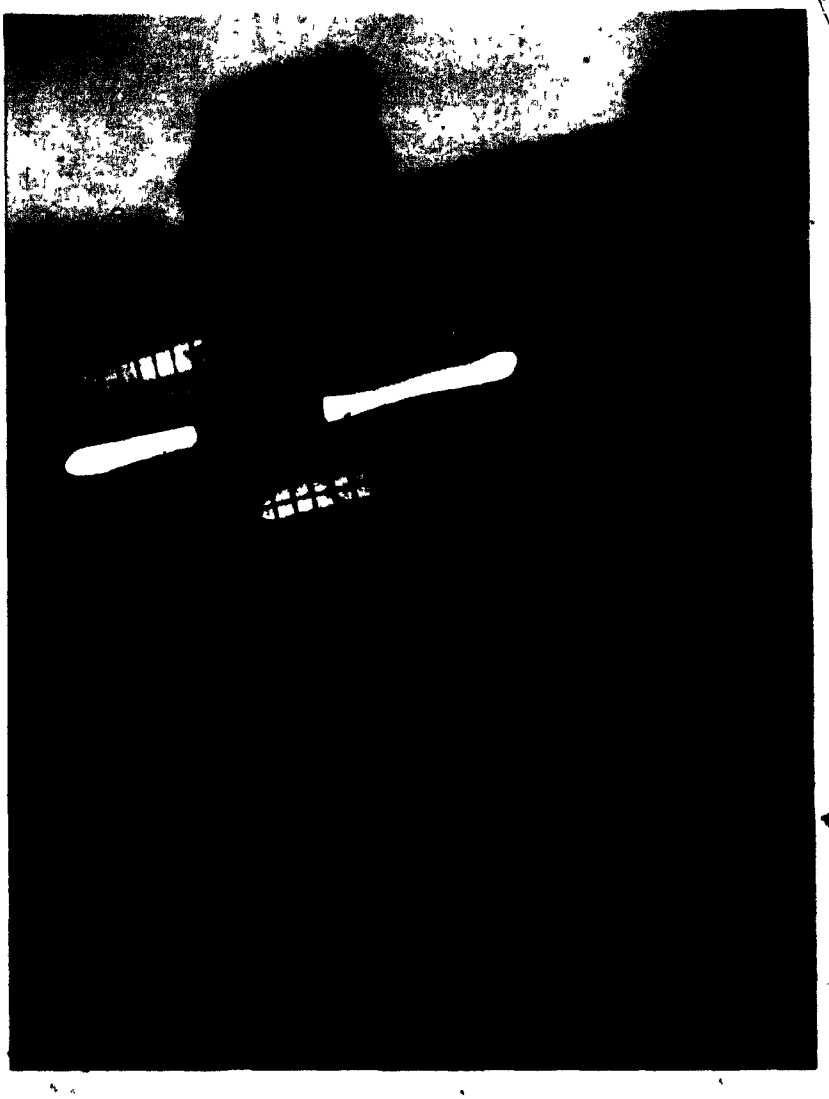


Figure #8: Juvenile Homarus americanus in Behavioural Testing Chamber After Removal of Interchamber Partition, Actively Engaged in 'Aggressive' Encounter.

Three such units holding from 10 to 12 animals each were immersed in a large white tray filled with fresh sea water. The water level was always carefully measured to ensure an equal volume in each chamber at each test period.

One day prior to testing, each animal was weighed and introduced into a chamber. The animals were paired according to weight, but otherwise dispersed randomly throughout the unit. The pairing was an attempt to balance the universal tendency for larger animals to defeat smaller, which appears to be at least partially independent of any innate aggressive tendencies (Scrivener,1971). The animals were allowed a period of overnight accommodation as handling appears to temporarily interfere with the normal expression of agonistic activity (Scrivener,1971). No food was administered to the animals while in the chamber. The water was well aerated but noncirculating, and maintained at an average temperature of 19°C.

The following morning the tests were performed. Any animals moulting overnight in the chambers were eliminated from that days' experiment. Freshly moulted animals are universally recognized as easy 'victims' and subject to severe injury due to the softness of the exoskeleton.

Each pair was allowed a 5 minute interval during which they were able to interact freely. The initiation and conclusion of each aggressive encounter was recorded on tape in order that the experimentor could maintain an uninterrupted observation and refrain from any movements

which would distract the attention of the combatants.

According to Dr. P. Dunham (personal communication), "Aggression should be defined as any physical contact initiated by either lobster which is preceeded by meral spread. Each contact, followed by a break in contact, is counted as one instance." This formula was employed in defining the aggressive encounters, but with one slight modification. The attack must be preceeded by 'meral spread' (see literature review), but must be between two lobsters in face to face contact. Jabs made at the back or flank of an inattentive opponent, or one in active flight, were not counted. In truly aggressive specimens such contact was always followed by rapid turning and a measurable fight. Meral spread implies 'intent' and avoids the false inclusion of chance encounters due to the confinement of two animals in a limited space.

The timing of encounters started following meral spread, when the 2 lobsters made physical contact, and stopped when the large claws of the opponents no longer overlapped in space. An encounter that was still in progress at the end of the 5 minute interval was regarded as having terminated with the experiment.

This pairwise testing proceeded in random sequence until all pairs had been completed. Activity in one test chamber had no observable effect on animals in adjacent chambers. Animals generally remained inactive prior to removal of the partition, and ambulatory animals were

usually engaged in a nonagitated investigation of their chambers.

After testing, the animals were gently returned to their containers, fed, and replaced in the troughs. The test units and the holding tray were washed, refilled, and another family was set up ready for the following day.

Three parameters of aggression were decoded from the information on the recording tape. The first was latency, the time elapsed from the beginning of the 5 minute interval to the initiation of the first encounter. The second parameter was duration, the total time measured in seconds during which the two animals were actively engaged. The final parameter was frequency, the actual number of aggressive encounters occuring in the 5 minute interval.

Results and Statistical Methods

I. Growth Rate

(a) Fourth Stage Weight

The mean fourth stage weight and standard error for each family in population #2 and the first 5 families in population #1 are reported in table #1. Measurements were not available for the remaining families in population #1.

Bartlett's test for homogeneity of variances (Sokal & Rolf,1969) was used to ensure that the data were homoscedastic and thus appropriate for a linear analysis of variance. Differences in mean fourth stage weights were analyzed using a Model II, single classification analysis of variance for unequal sample size. The structure of variation is partitioned according to the following formula,

$$y_{ij} = \mu + \alpha_i + \varepsilon_{ij}$$

where $i = 1,2, \ldots, a$ families, $j = 1,2, \ldots, n$ individuals within families, ε_{ij} represents a normally distributed error term with mean 0 and variance σ^2, and α_i represents a normally distributed variable (the effect of the ith mating) independent of the residual ε_{ij}, with mean 0 and variance σ^2_A (Sokal & Rolf,1969). The variance component σ^2_A is due to the variance of means of full sib families and is therefore equivalent to the covariance of full sibs (Falconer,1960).

For a breakdown of the anova table and relevant formulae for estimation of the variance components and levels of significance, please refer to Appendix #1.

The fourth stage weights were found to vary significantly between families with a 0.001 probability of committing a type I error.

The heritability of fourth stage weight was computed as follows,

$$h^2 = \frac{2\,\hat{\sigma}_A^2}{\hat{\sigma}_A^2 + \hat{\sigma}_W^2}$$

where h^2 stands for heritability (not its square). The value thus obtained was 0.805 .

The numerator includes an estimate of ½ the variance due to dominance, as well as all the variance due to maternal effects (Becker,1975). Therefore, although the computed heritability is thought to provide an indication of selective potential, it is heritability in the broad sense only.

The standard error for the heritability estimate was calculated according to the formula suggested by Walter A. Becker in "Manual of Quantitative Genetics" (1975). For unequal numbers of progeny per mating,

$$S.E.(h^2) = \sqrt{\frac{2(n.-1)(1-t)^2\,[1 + (n_o-1)t]^2}{n_o^2\,(n.-a)(a-1)}}$$

which in this case yields a value of 0.1662 . The variable, t, is the coefficient of intraclass correlation,

$$t = \frac{\sigma_A^2}{\sigma_W^2 + \sigma_A^2} ,$$

where σ_A^2 is the between family component of the total phenotypic variance and σ_W^2 is the within family component.

Table #1: Mean Fourth Stage Weights (in grams) of Juvenile *Homarus americanus* (range 4 to 8 days post 4th stage moult).

Family	N	Mean 4th Stage Weight
D	30	0.0434 ± 0.00068
E	25	0.0388 ± 0.00069
F	30	0.0351 ± 0.00055
G	30	0.0371 ± 0.00051
H	30	0.0457 ± 0.00080
AA	20	0.0420 ± 0.00109
CC	20	0.0411 ± 0.00086
DD	19	0.0424 ± 0.00090
EE	20	0.0377 ± 0.00080
FF	20	0.0384 ± 0.00090
GG	20	0.0376 ± 0.00093
HH	20	0.0294 ± 0.00060
II	20	0.0367 ± 0.00098
JJ	20	0.0463 ± 0.00085
KK	18	0.0308 ± 0.00070
LL	20	0.0388 ± 0.00117
MM	20	0.0460 ± 0.00094
NN	20	0.0349 ± 0.00070
OO	18	0.0429 ± 0.00077
QQ	18	0.0443 ± 0.00076
RR	18	0.0458 ± 0.00115

(b) Growth Rate

The individual growth rates were calculated as a regression of weight on time for each animal, and statistically weighted according to the number of actual measurements employed in each calculation. The weighted mean growth rates for population #1 and population #2 are reported in tables #2 and #3 respectively.

The test for homoscedasticity was applied to the untransformed data, and the family variances were found to differ significantly. The logarithmic transformation (ln) was applied to correct, and the data were divided into two separate populations for the purpose of analysis. The division was one of mathematical necessity; no appropriate transformation could be found which would render the variances sufficiently uniform across the two populations. It was also felt to be a satisfactory biological delineation as the two populations differed subjectively in the quality of brine shrimp with which they were fed. (Family M was omitted from the calculations for the first population in order that the variances could be made to conform. Families O,P, and R were similarly omitted; the growth rates for these families were felt to be biased by an almost universal tendency among individuals toward a progressive weight loss during the latter half of the experiment. This weight loss was felt to be atypical, possibly the result of some undetected pathogen affecting only these families.)

A weighted least squares analysis of variance was

performed on the transformed data for each population. The model employed was the same as that described for fourth stage weights (see Appendix #1 for anova tables and relevant formulae). Differences in weighted transformed family means for growth rate were found to be significant for both populations at an alpha equal to 0.001 . Heritability values were calculated at 0.707 ± 0.234 for population #1 and 0.540 ± 0.171 for population #2.

Overall growth rates were calculated for those families with both group and individually reared juveniles, as a regression of ln mean weight (for all individuals in family) at each time, on time, for each type of rearing. (The ln transformation was chosen to improve goodness of fit of regression line for group reared individuals.) These values are reported in tables #4 and #5 for population #1 and #2 respectively.

Group variances for each rearing method were found to be highly heteroscedastic in both populations. Modified t-tests were therefore employed to determine whether significant differences existed between the means of group versus individually reared animals (see Appendix #2 for detail of computation). The mean growth rates for individually reared juveniles in population #1 were found to differ at 0.001 level of significance from group reared juveniles, and those in population #2 were found to differ at 0.05 level of significance. These differences are illustrated in Figures #9 and #10 for representative

families in each population; these graphs clearly show that group reared animals grow much more rapidly than their individually reared sibs.

Table #2: Mean Growth Rate for Juvenile *Homarus americanus* in Population #1 (based on individual growth rates calculated as regression of weight on time and weighted by number of measurements per individual) in grams x 10^3 per day.

Family	Weighted Mean Growth Rate
D	1.972 ± 1.053
E	2.268 ± 1.059
F	1.784 ± 1.076
G	2.239 ± 1.065
H	1.699 ± 1.062
I	2.234 ± 1.079
J	2.050 ± 1.068
K	1.866 ± 1.065
L	1.611 ± 1.081
M	1.868 ± 1.122
N	0.932 ± 1.075

Table #3: Mean Growth Rate for Juvenile Homarus americanus in Population #2 (based on individual growth rates calculated as regression of weight on time and weighted by number of measurements per individual) in grams x 10^3 per day.

Family	Weighted Mean Growth Rate
AA	4.958 ± 1.134
CC	4.646 ± 1.151
DD	5.387 ± 1.089
EE	4.112 ± 1.105
FF	1.876 ± 1.126
GG	2.565 ± 1.057
HH	5.414 ± 1.124
II	4.141 ± 1.112
JJ	3.971 ± 1.130
KK	2.482 ± 1.149
LL	3.518 ± 1.136
MM	4.637 ± 1.106
NN	3.497 ± 1.123
OO	2.849 ± 1.159
PP	3.025 ± 1.176
QQ	3.550 ± 1.139
RR	3.648 ± 1.113

Table #4: Comparison of Growth Rates for Population #1 Group Reared Juvenile Homarus americanus With Those Reared Individually (growth rate calculated as regression of ln mean weight at each time, on time, for each rearing method; R = Pearson product moment correlation coefficient, used to measure goodness of fit of regression line).

Family	10^2 x Growth Rate (Individual)	R	10^2 x Growth Rate (Group)	R
H	1.374	0.921	3.011	0.997
J	1.331	0.937	3.358	0.986
K	1.140	0.937	3.304	0.984
M	1.400	0.976	2.441	0.992
N	1.046	0.933	2.593	0.883

$\bar{x}_1 = 1.2668$ $\bar{x}_2 = 2.9741$

$s_1^2 = 0.0249$ $s_2^2 = 0.1367$

Growth rate for both methods of rearing in (ln grams) x 10^2 per day.

Table #5: Comparison of Growth Rates for Population #2 Group Reared Juvenile Homarus americanus With Those Reared Individually (growth rate calculated as regression of ln mean weight at each time, on time, for each rearing method; R = Pearson product moment correlation coefficient).

Family	10^2 x Growth Rate (Individual)	R	10^2 x Growth Rate (Group)	R
AA	2.121	0.992	3.040	0.952
CC	2.202	0.992	3.687	0.970
DD	2.118	0.989	3.319	0.980
JJ	1.923	0.990	3.587	0.985
KK	2.064	0.990	3.427	0.987
LL	2.037	0.991	5.019	0.987
MM	1.946	0.988	3.848	0.963
NN	1.978	0.993	3.522	0.976

$\bar{x}_1 = 2.0487$ $\bar{x}_2 = 3.5721$

$s_1^2 = 0.0082$ $s_2^2 = 0.2124$

Growth rate for both methods of rearing in (ln grams) x 10^2 per day.

Figure #9: Growth Curves (eye fitted) for Family J, Population #1 Showing Relationship Between Group Reared and Individually Reared Juvenile Homarus americanus; growth calculated as regression of ln mean weight at each time, on time, for both methods of rearing.

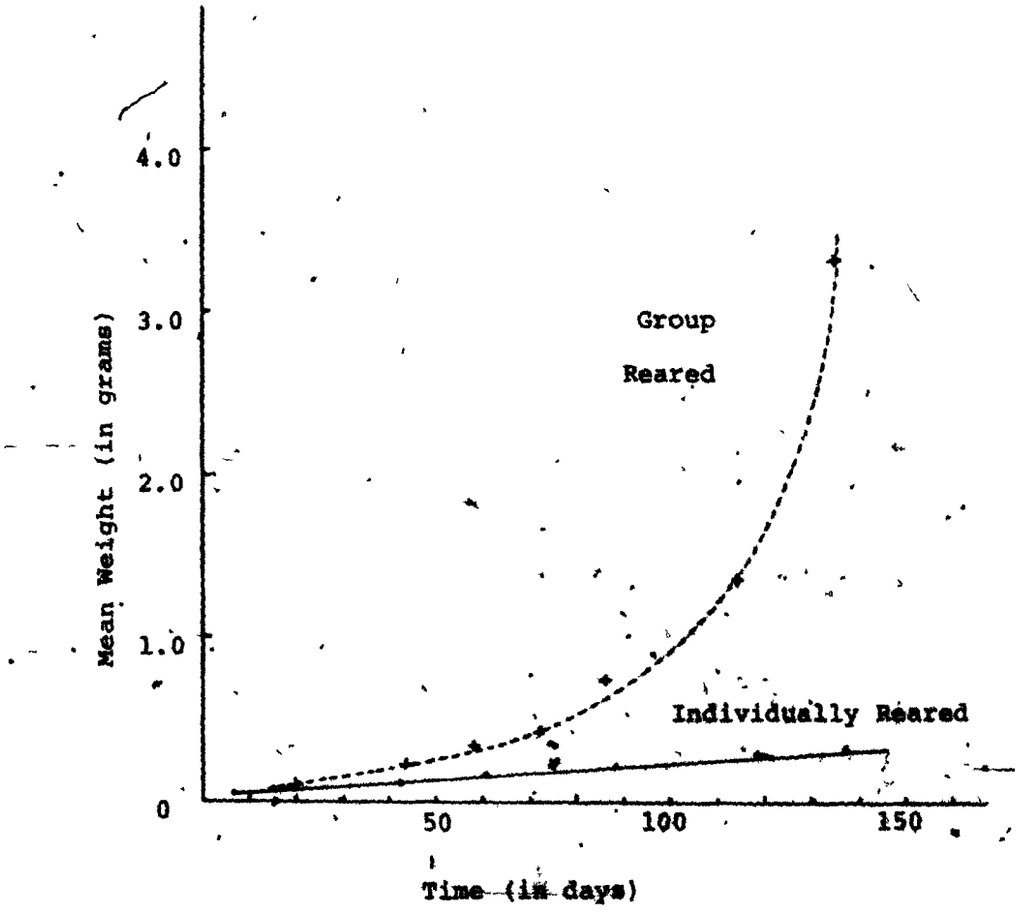
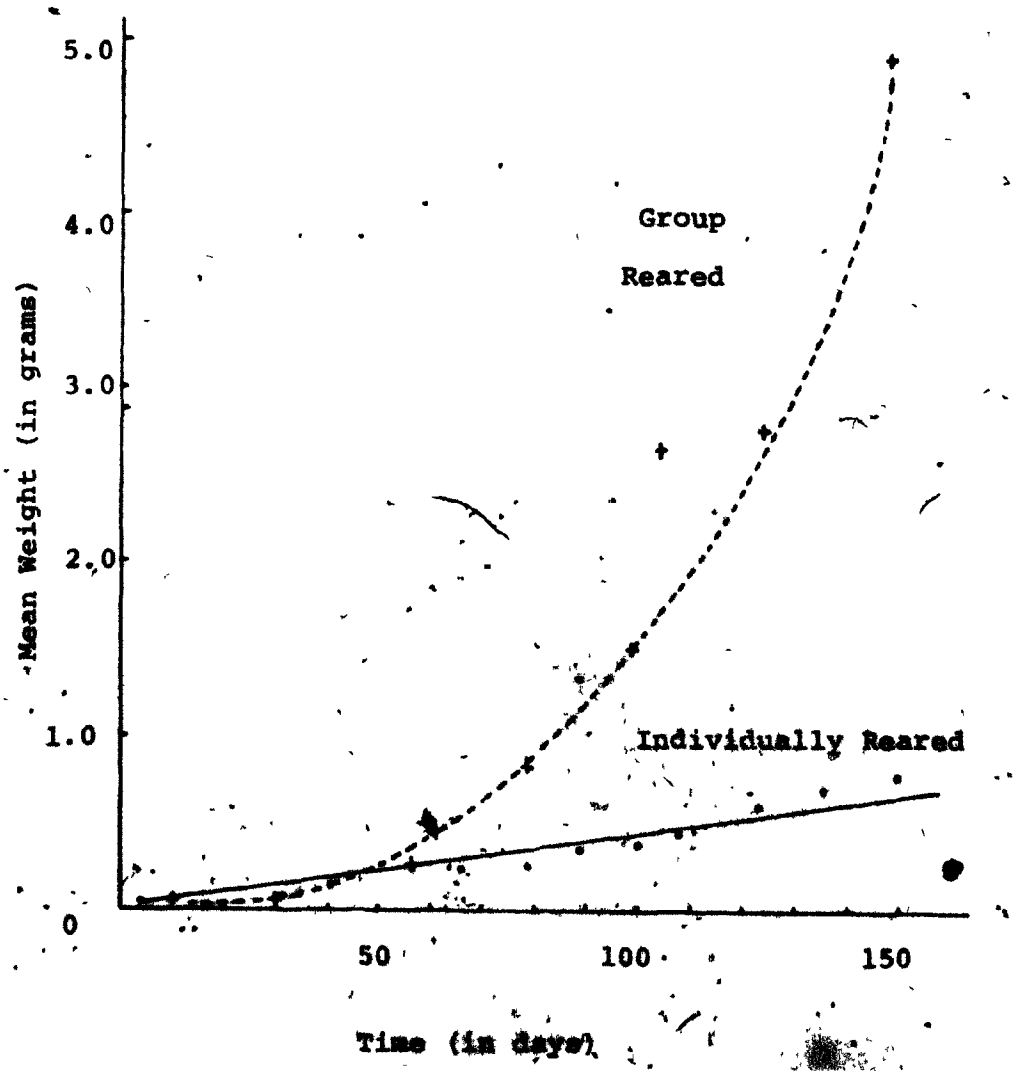


Figure #10: Growth Curves (eye fitted) for Family JJ, Population #2 Showing Relationship Between Group Reared and Individually Reared Juvenile Homarus americanus; growth calculated as regression of ln mean weight at each time, on time, for both methods of rearing.



(c) 150 Day Weights

The mean 150 day weights (calculated from the point of entering fourth stage) where available, are reported with their standard errors in tables #6 and #7 for population #1 and #2 respectively.

The family variances for 150 day weight proved to be sufficiently homogeneous within populations, and no transformations have been applied to the data. In a single classification model II anova (see Appendix #1), the means of population #1 were found to differ significantly at the 0.05 level, and those of population #2 at the 0.001 level of significance.

The heritability of 150 day weight was evaluated for both groups. The heritability for population #1 was estimated at the fairly low value of 0.045 ± 0.114 . This may not be a particularly reliable estimate as only 4 of the initial 14 families of population #1 had members surviving to the age of 150 days. (A system malfunction resulted in fatally high temperatures and the premature death of all of the juveniles in year #1 of the experiment.) The heritability for population #2 was considerably higher at 0.516 ± 0.225 ; all but 1 family in population #2 were included in this estimate.

There was no significant correlation between fourth stage and 150 day weight.

Growth rates were recalculated using only those individuals surviving to 150 days post 4th stage moult (see tables 8 and 9).

Family variances were found to be homogeneous within populations, and no transformations or weighting factors were applied to the data. Differences among family means in population #1 were not significant; those of population #2 were found to differ at a 0.01 level of significance.

Heritabilities were found to correspond closely with estimates for 150 day weight. The calculated heritability for population #1 was 0.138 ± 0.173, and for population #2 was 0.513 ± 0.225 .

Table #6: Mean 150 Day Weight (post 4th stage moult) for Surviving Juvenile Homarus americanus in Population #1.

Family	N	Mean 150 Day Weight (grams)
D	24	0.3400 ± 0.0158
E	22	0.3527 ± 0.0158
F	23	0.3174 ± 0.0213
G	14	0.3998 ± 0.0206

Table #7: Mean 150 Day Weight (post 4th stage moult) for Surviving Juvenile Homarus americanus in Population #2.

Family	N	Mean 150 Day Weight (grams)
AA	8	1.0192 ± 0.0833
CC	6	1.0812 ± 0.0892
DD	15	0.9484 ± 0.0652
EE	6	0.8042 ± 0.0385
FF	3	0.4726 ± 0.0915
HH	9	0.9379 ± 0.0740
II	3	0.7757 ± 0.0722
JJ	7	0.7853 ± 0.0561
KK	2	0.6646 ± 0.0554
LL	4	0.8249 ± 0.0701
MM	8	0.9431 ± 0.0671
NN	10	0.7645 ± 0.0949
OO	2	0.5965 ± 0.1725
PP	2	0.7990 ± 0.0650
QQ	7	0.6316 ± 0.0596
RR	6	0.6643 ± 0.0921

Table #8: Mean Growth Rate for Juvenile Homarus americanus Surviving to 150 Days, Population #1 (growth rate calculated as regression of weight on time for each individual).

Family	N	Mean Growth Rate x 10^3 g/day
D	24	2.066 ± 0.1212
E	22	2.359 ± 0.1365
F	23	1.962 ± 0.1468
G	14	2.435 ± 0.1439

Table #9: Mean Growth Rate for Juvenile Homarus americanus Surviving to 150 Days, Population #2 (growth rate calculated as regression of weight on time for each individual).

Family	N	Mean Growth Rate x 10^3 g/day
AA	8	6.711 ± 0.5139
CC	6	6.862 ± 0,7147
DD	15	5.983 ± 0.3951
EE	6	5:310 ± 0.2113
FF	3	3.267 ± 0.7234
HH	9	6.910 ± 0.5187
II	3	5.158 ± 0.3489
JJ	7	5.217 ± 0.4094
KK	2	4.585 ± 0.3647
LL	4	5.863 ± 1.0205
MM	8	5.803 ± 0.5850
NN	10	4.425 ± 0.5402
OO	2	4.215 ± 1.3849
PP	2	5.080 ± 0.3900
QQ	7	4.249 ± 0.3640
RR	6	4.443 ± 0.5796

II. Mortality

Although mortality was originally measured in absolute units, that is, actual number of days surviving, it was translated for the purpose of analysis to 'percent mortality'. This was done primarily to avoid problems caused by the artificially imposed limits to survival of 125 or 150 days. If mean survivorship is plotted against the standard deviation, (see figure #11) the data are seen to form a parabolic curve; the standard deviation is compressed in families with both low and high survivorship, and no appropriate transformation can be employed to relieve the heteroscedasticity.

Although the underlying genetic factors which regulate the expression of mortality are assumed to follow a continuous type of variation, the phenotype is of an all or none nature. Such truncated data can not be appropriately analyzed using standard analysis of variance techniques.

However, a method has been developed for calculating heritability using quantitative data expressed as a proportion. The method employs an analysis of variance modified for binomial data, and indirectly provides values for the familiar $\hat{\sigma}^2_W$, expected within family variance, and $\hat{\sigma}^2_A$, the expected variance due to family effects. Heritability is expressed in terms of the genotypic variance (for viability in this case) to the mean (viability). For a more complete discussion of the analysis and

associated formulae, the reader is referred to Appendix III, or to Robertson and Lerner (1949).

The percent mortality for each family, calculated at 25 day intervals, has been listed in tables #10 and #11. Mortality values have also been recalculated (table #12) using the combined data for all individuals in each population (paying no attention to family groups), and the differences are illustrated in figure #12.

The binomial data for each population was tested for heteroscedasticity using Bartlett's test for homogeneity of variance. The variances for population #1 were sufficiently uniform and required no transformation; the variances for population #2 were not equal, but the departure from homogeneity was slight, the variances were small, and the raw data were used without transformation.

There was no significant difference between the percent mortalities calculated at 125 days for those families in population #1. The heritability was estimated at 0.040 ± 0.043 .

There was a significant difference at a 0.01 level between the 150 day mortality values for population #2. The heritability was estimated at 0.267 ± 0.111 .

The percent mortality at 125 days was calculated for each family having both group and individually reared juveniles. These values are reported in tables #15 and #16 for populations #1 and #2 respectively.

Variances were compared and found to differ

nificantly between the two rearing methods. Group means for each method were therefore compared using a modified t-test for equality of means of 2 samples (see Appendix II) and found to differ significantly at 0.05 level in population #1 and at 0.01 level in population #2.

These differences are most clearly illustrated in figures #13 and #14, where the percent mortality for each population, calculated at 25 day intervals, is plotted against time. The mortality estimates are reported in tables #13 and #14 for individually and group reared juveniles respectively.

Figure #11: Plot of Mean Family Survivorship (number of days living beyond 4th stage moult) Versus Standard Deviation of Family Survivorship.

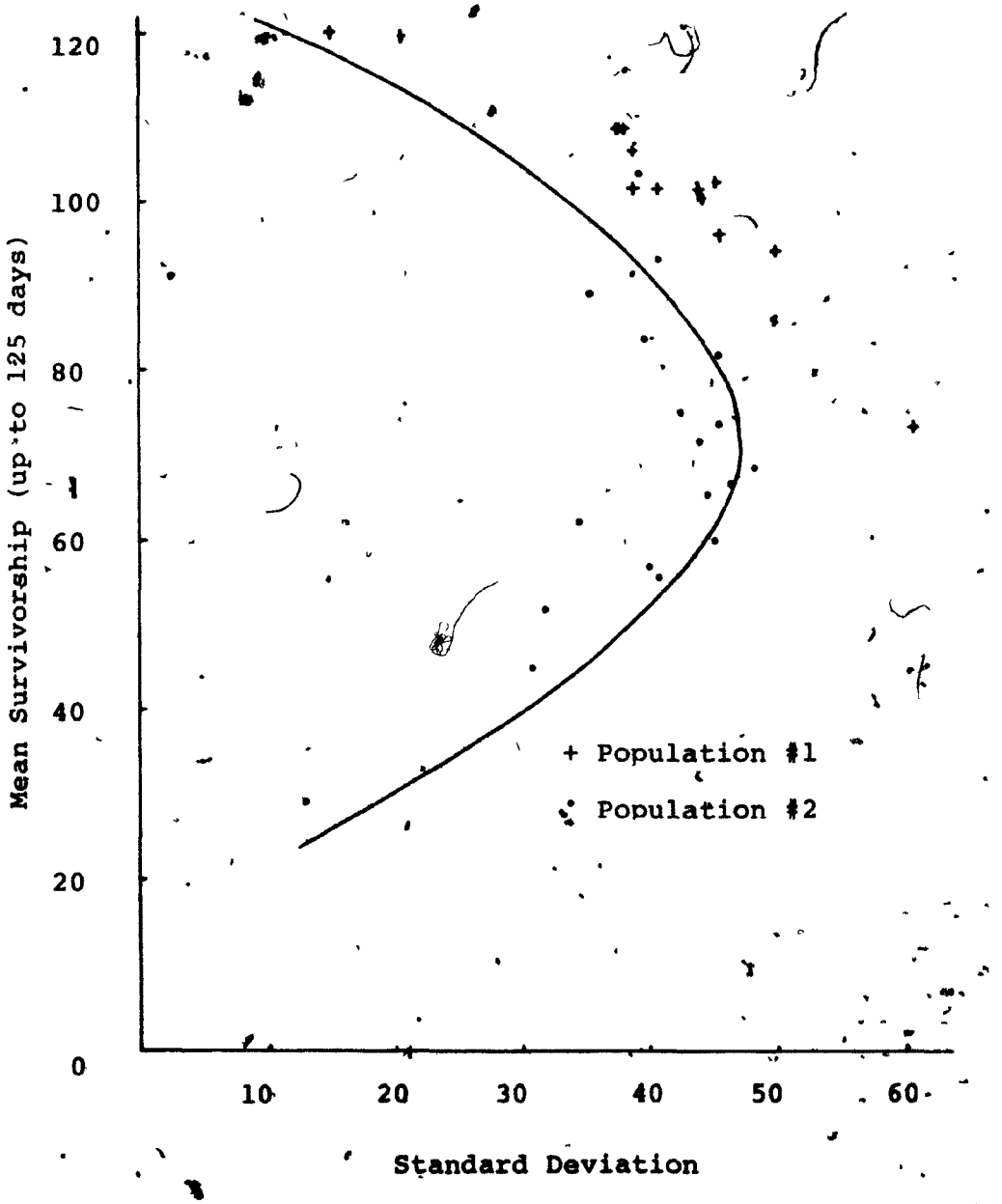


Table #10: Percent Mortality at 25 Day Intervals Calculated for Individually Reared Juvenile Homarus americanus in Population #1.

Family	25D	50D	75D	100D	125D	150D
D	00.00	00.00	6.67	6.67	16.67	20.00
E	42.86	42.86	42.86	42.86	42.86	47.62
F	9.38	9.38	9.38	12.50	18.75	21.88
G	17.14	22.86	28.57	28.57	34.29	54.29
H	3.33	3.33	3.33	6.67	10.00	20.00
I	18.18	21.21	21.21	30.30	33.33	100*
J	15.15	15.15	21.21	24.24	24.24	100*
K	3.23	6.45	6.45	12.90	12.90	100*
L	15.15	18.18	24.24	24.24	30.30	100*
M	13.33	16.67	20.00	23.33	40.00	100*
N	12.90	12.90	16.13	19.35	29.03	100*
O	12.90	12.90	12.90	12.90	25.81	100*
P	16.13	19.35	19.35	19.35	19.35	100*
Q	3.23	3.23	9.68	29.03	100*	100*

* These figures are not representative but are the result of death from unnatural causes; a system malfunction resulted in fatally high temperatures and a premature termination of the experiment for Population #1.

Table #11: Percent Mortality at 25 Day Intervals Calculated for Individually Reared Juvenile Homarus americanus in Population #2.

Family	25D	50D	75D	100D	125D	150D
AA	10.00	35.00	60.00	60.00	60.00	60.00
BB	35.00	95.00	100	100	100	100
CC	10.00	35.00	60.00	65.00	70.00	75.00
DD	5.00	20.00	25.00	25.00	25.00	25.00
EE	9.52	61.90	71.43	71.43	71.43	71.43
FF	0.00	40.00	60.00	85.00	85.00	85.00
GG	5.00	75.00	90.00	90.00	95.00	95.00
HH	0.00	20.00	45.00	55.00	55.00	55.00
II	10.00	60.00	65.00	65.00	80.00	80.00
JJ	15.00	40.00	55.00	60.00	65.00	65.00
KK	20.00	55.00	70.00	80.00	90.00	90.00
LL	10.00	20.00	55.00	60.00	80.00	80.00
MM	10.00	40.00	40.00	50.00	55.00	60.00
NN	5.00	30.00	35.00	40.00	45.00	50.00
OO	20.00	65.00	65.00	75.00	90.00	90.00
PP	5.00	55.00	90.00	90.00	90.00	90.00
QQ	15.00	55.00	60.00	65.00	65.00	65.00
RR	25.00	45.00	55.00	60.00	70.00	70.00

Table #12: Percent Mortality at 25 Day Intervals for Individually Reared Juvenile Homarus americanus Calculated for Each Population.

Day	Population #1	Population #2
25	13.90 ± 0.00076	11.30 ± 0.00087
50	15.90 ± 0.00081	45.30 ± 0.00137
75	18.30 ± 0.00085	59.70 ± 0.00135
100	22.00 ± 0.00091	64.90 ± 0.00132
125	26.70 ± 0.00105	69.10 ± 0.00128
150	34.30 ± 0.00281	72.40 ± 0.00123

Figure #12: Percent Mortality at 25 Day Intervals for Individually Reared Juveniles in Population #1 and Population #2, Calculated as a Total for All Families in Each Population.

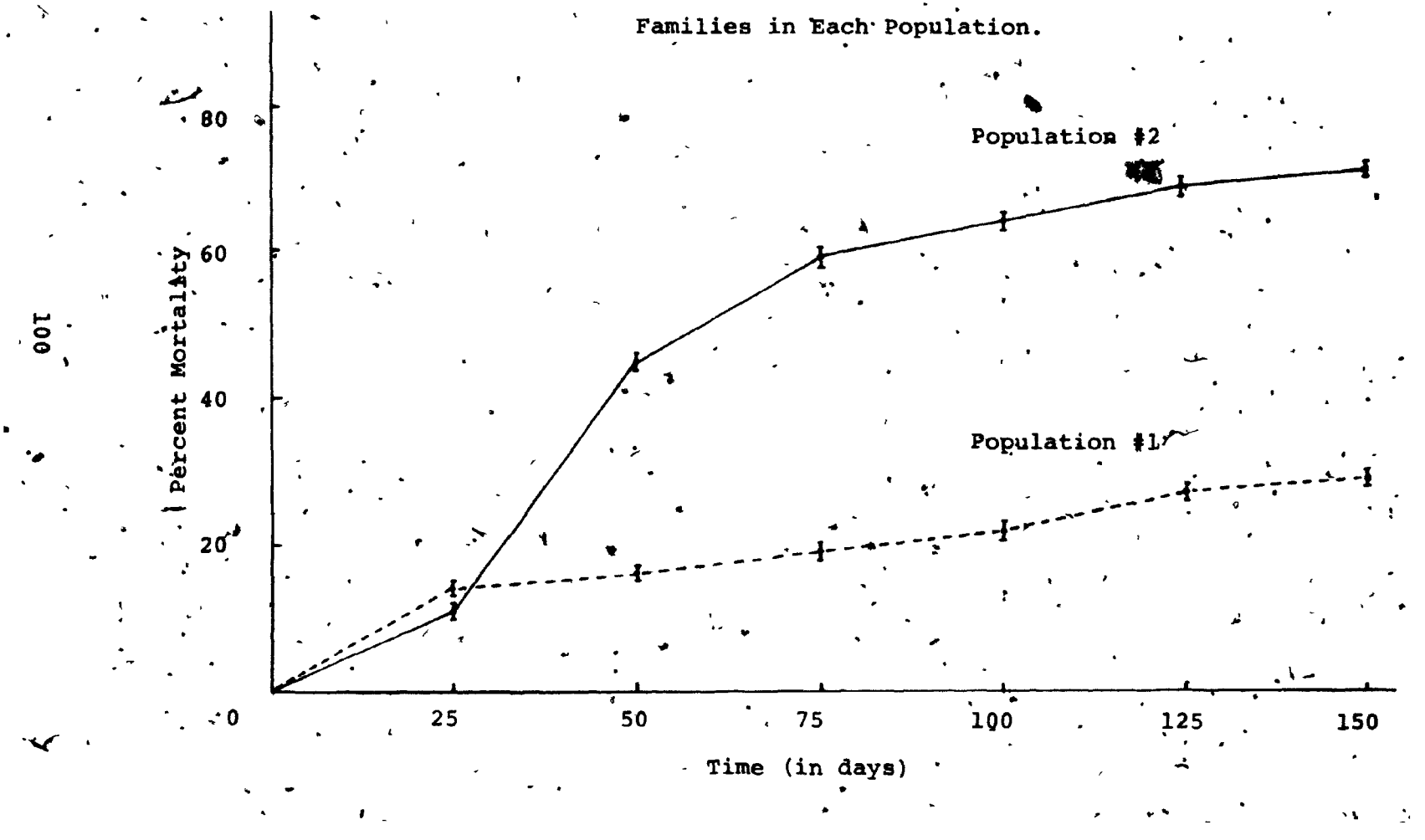


Table #13: Percent Mortality for Individually Reared Juvenile Homarus americanus Calculated for Each Population at 25 Day Intervals.

Day	Population #1	Population #2
25	10.20 ± 0.00193	12.70 ± 0.00184
50	12.10 ± 0.00208	39.80 ± 0.00270
75	14.60 ± 0.00225	54.10 ± 0.00275
100	18.50 ± 0.00247	59.10 ± 0.00272
125	23.60 ± 0.00270	63.00 ± 0.00267
150		66.30 ± 0.00261

Includes families: Population #1, H,J,K,M,&N; Population #2, AA,BB,CC,DD,JJ,KK,LL,MM,&NN.

Table #14: Percent Mortality for Group Reared Juvenile Homarus americanus Calculated for Each Population at 25 Day Intervals.

Day	Population #1	Population #2
25	28.00 ± 0.00299	18.30 ± 0.00215
50	53.30 ± 0.00333	51.10 ± 0.00278
75	76.00 ± 0.00285	80.00 ± 0.00222
100	86.70 ± 0.00226	94.40 ± 0.00128
125	94.70 ± 0.00149	96.10 ± 0.00108
150		96.10 ± 0.00108

Includes families: Population #1, H,J,K,M & N; Population #2, AA,BB,CC,DD,JJ,KK,LL,MM, & NN.

Figure #13: Mortality Curves For Population #1 (includes families J,H,K,M, and N) Illustrating Difference Between Group and Individually Reared Juvenile Homarus americanus.

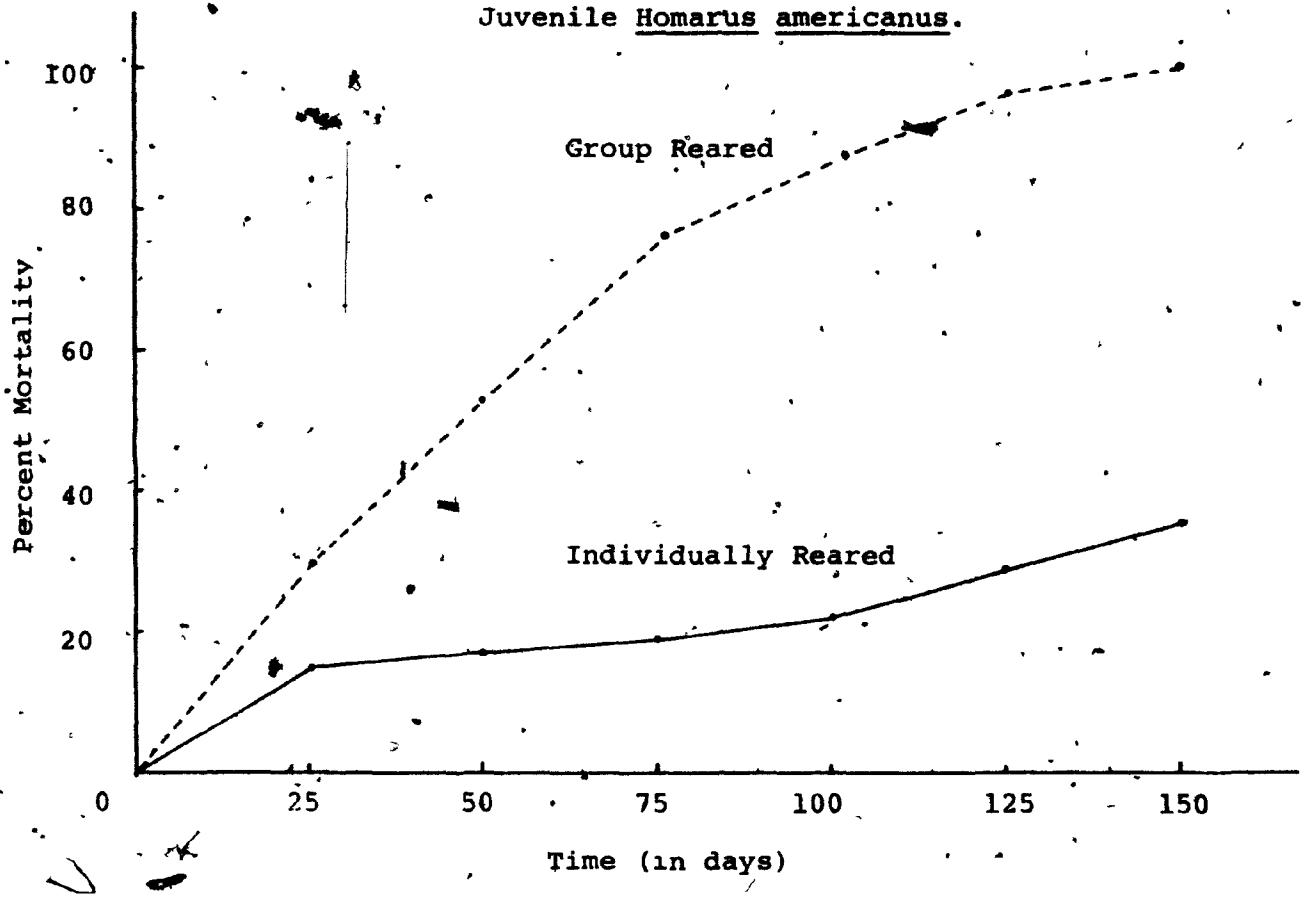


Figure #14: Mortality Curves for Population #2 (includes families AA,BB,CC, DD,JJ,KK,LL,MM,&NN) Illustrating Difference Between Group and Individually Reared Juvenile Homarus americanus.

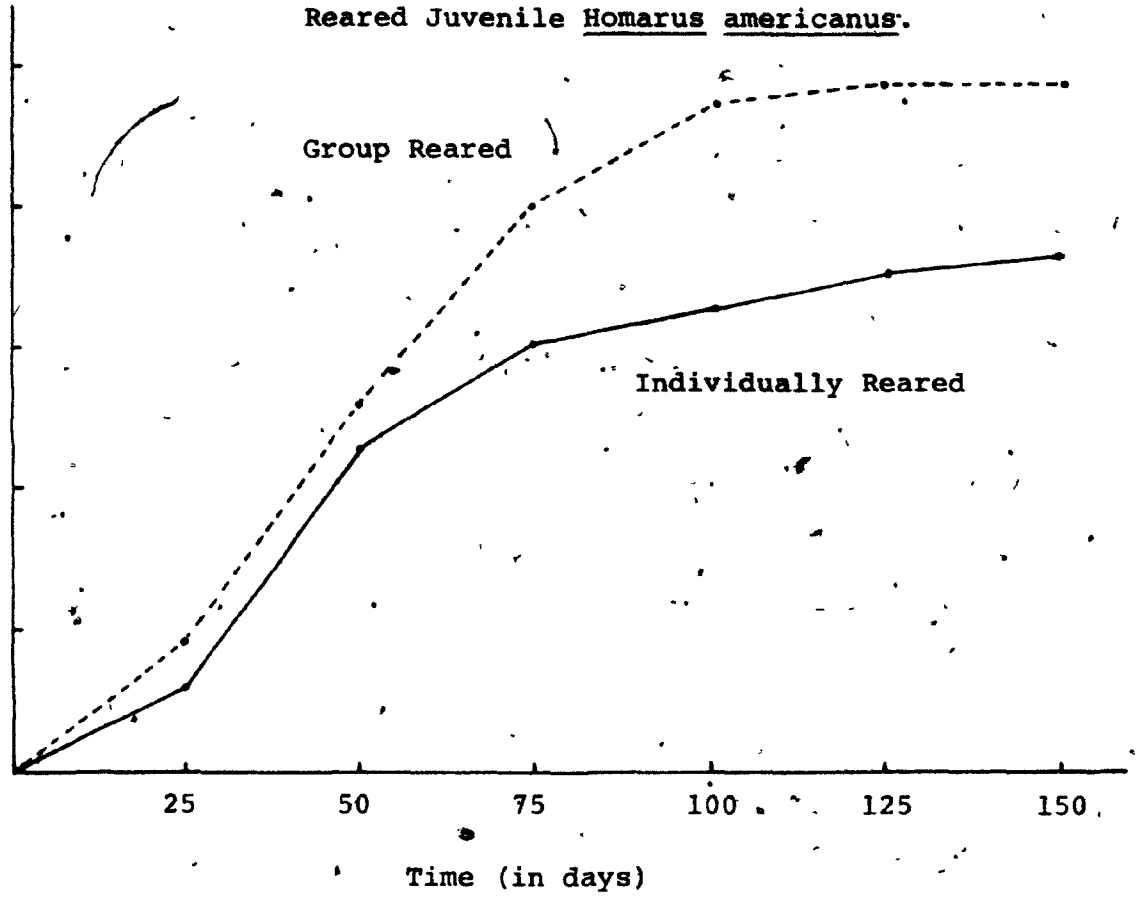


Table #15: Percent Mortality at 125 Days for Individual Versus Group Reared Juvenile Homarus americanus in Population #1.

Family	% Mortality (Ind)	% Mortality (Group)
H	10.00	93.00
J	24.24	93.00
K	12.90	93.00
M	40.00	93.00
N	29.03	100.00

$\bar{x}_1 = 23.23$ $\bar{x}_2 = 94.40$

$s_1^2 = 149.41$ $s_2^2 = 9.80$

Table #16: Percent Mortality at 125 Days for Individual Versus Group Reared Juvenile Homarus americanus in Population #2.

Family	% Mortality (Ind)	% Mortality (Group)
AA	60.00	95.00
CC	70.00	100.00
DD	25.00	95.00
JJ	65.00	95.00
KK	90.00	95.00
LL	80.00	100.00
MM	55.00	95.00
NN	45.00	95.00
BB	100.00	95.00

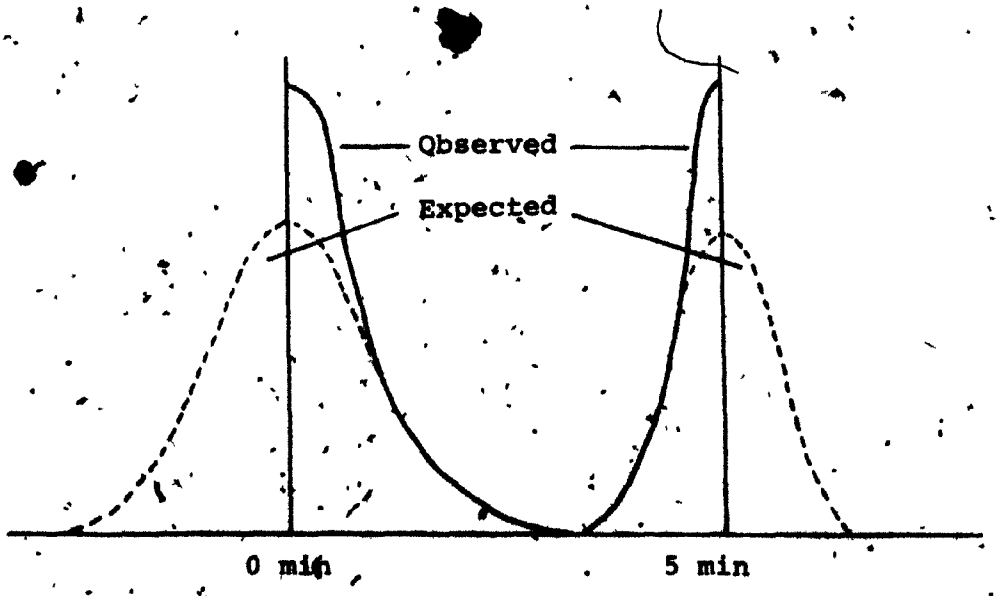
$\bar{x}_1 = 65.56$ $\bar{x}_2 = 96.11$

$s_1^2 = 527.78$ $s_2^2 = 4.86$

III. Parameters of Aggression

Frequency histograms illustrating the distribution for each of the parameters of aggression are plotted in figures #16 through #18. All three can be visualized as 'truncated normal' distributions; the left tail of each normal curve can be represented by an imaginary line extending beyond and to the left of the 0 value on the x axis (see figure #15). The right tail may also extend in a similar fashion beyond the artificial limit imposed by the 5 minute experimental testing interval (especially apparent in the case of latency).

Figure #15: Hypothetical Frequency Distribution for 'Truncated' Data Showing the Probable Observed Frequency and the 'Assumed' Underlying Distribution.



It should be noted that although the assumption of a 'truncated normal' distribution can not be strictly proven, it is theoretically 'expected' with behavioural characters, which, although represented genotypically on a continuous underlying scale, are phenotypically discontinuous. Although the system is 'polygenic', there is a threshold level of contributing alleles which must be present before the character can be expressed.

The observed phenotypic variation may therefore not be strictly representative of the available genetic variation, and the calculated estimates of σ_A^2 and heritability should be regarded as conservative estimates only.

Because the aggression experiments were not strictly random, an analysis of covariance was performed to assess the possibility of added effects due to weight, and or individual animals.

In terms of the analysis of variance models employed elsewhere throughout this paper, the covariance model for a one way classification can be described as follows,

$$y_{ij} = \mu + \alpha_i + bz_{ij} + \varepsilon_{ij}$$

where y_{ij} is the measured response, μ is the family mean, α_i is the effect due to animal i (represented by a dummy variable that takes the value of 0 or 1 depending on presence or absence), b is the regression style coefficient

of the vector z of observed values of the covariate weight, and ϵ_{ij} is a randomly distributed error term with mean 0 and variance σ^2 (Searle,1971).

In practise, the calculations were performed using the general linear model

$$y = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \ldots + \beta_n x_n + \epsilon$$

which relates the response, y, to a quantitative variable (the covariate x_1, weight) and a set of qualitative variables (the animal effects x_2 to x_n), (Ott & Lyman,1977). The two models are mathematically equivalent; the general linear model was chosen for ease of computation.

Although all 3 parameters showed some degree of dependence on weight or animals within some families, it was concluded that overall, neither animals nor weight contributed significantly to the response, and that an analysis of variance could be appropriately applied to the data without first removing these effects. A complete report on the analysis of covariance for each family is provided in Appendix IV, including a justification for the conclusion stated above. It should be noted that choosing to ignore these effects may make it more difficult to pick up differences between families, and therefore estimates should be regarded as conservative.

The mean experimental test weight for each family is reported in tables #17 and #18 for population #1 and #2 respectively, and family means for all 3 aggression

parameters are reported in tables #19 and #20.

All 3 parameters were tested for homoscedasticity. Latency proved to be sufficiently homogeneous, but variances for duration and frequency were heteroscedastic; the ln[y + 1] transformation was applied to correct (where y represents the response parameter, duration or frequency).

The data for both populations was pooled in a two level hierarchical anova for unequal sample size. The statistical model is defined as follows,

$$y_{ijk} = \mu + \alpha_i + \beta_{ij} + \varepsilon_{ijk}$$

where y_{ijk} is the response of the kth individual of the jth family of the ith group (population), μ is the common mean, α_i is the effect due to the ith group, β_{ij} is the effect due to the jth family in the ith group, and ε_{ijk} is the random error component attributed to individuals (Becker,1975). All of the effects are random, independent, and normal. The expected variance components are $\hat{\sigma}^2_B$, the variance due to groups; $\hat{\sigma}^2_A$, the added component due to families, and $\hat{\sigma}^2_W$, the within family variance. A full description of the anova tables and relevant formulae for estimation of the variance components are included in Appendix V.

Latency showed no significant difference between populations, and subdivision of the data into two populations was therefore deemed unnecessary. The between family variance was significantly different at an alpha equal to

0.01, and the heritability for latency was estimated at 0.126 ± 0.027 .

The between population variance for frequency was also nonsignificant. The between family variance was significant at 0.01, and the heritability was estimated at 0.109 ± 0.050.

The between population variance for duration was nonsignificant, and the data were not subdivided. Family differences were significant at an alpha of 0.01 and the heritability was 0.086 ± 0.045 .

Figure #16: Frequency Histogram for Latency (elapsed time before initiation of encounter).

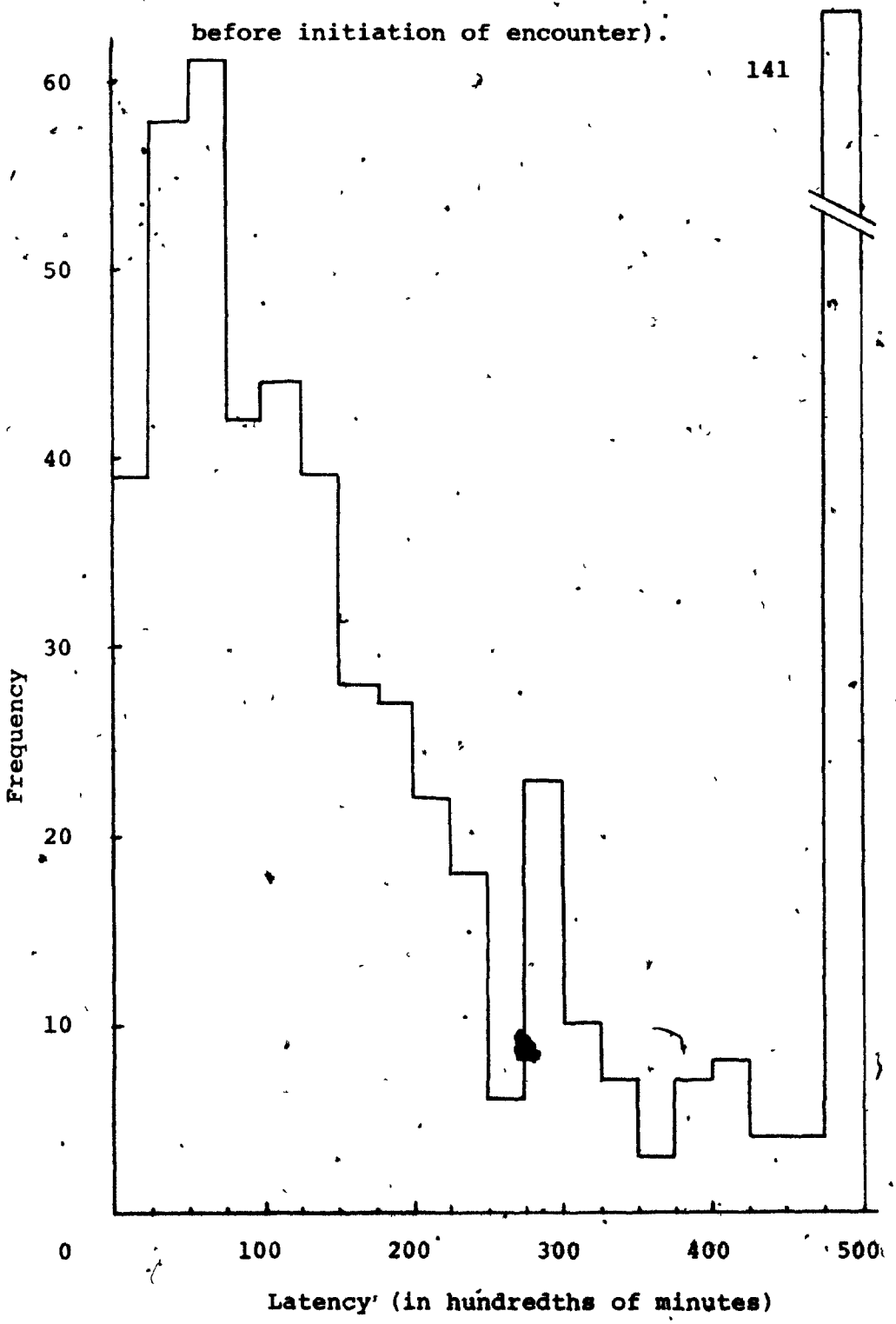


Figure #17: Frequency Histogram for Duration (total response time).

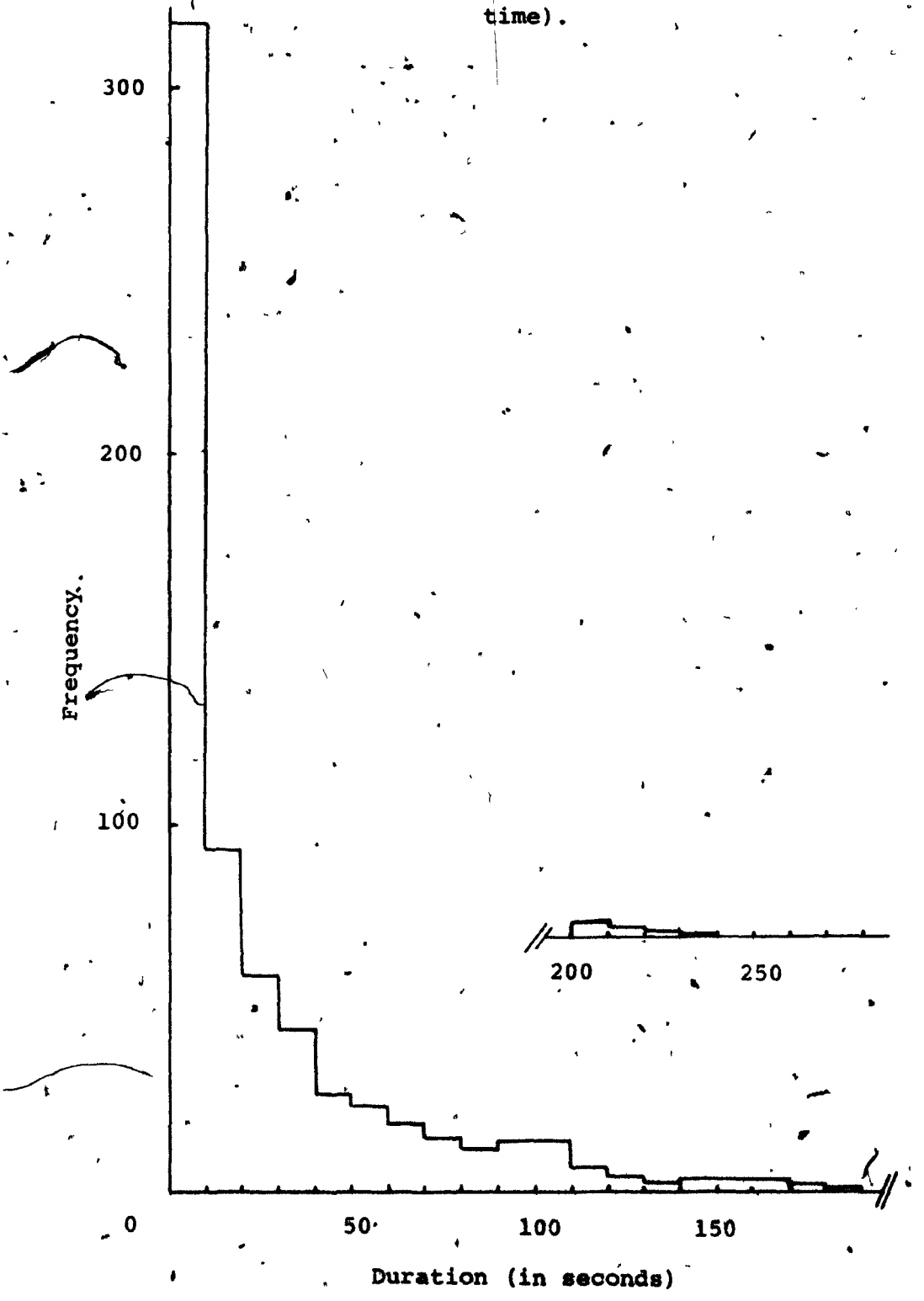


Figure #18: Frequency Histogram for Frequency (number of encounters).

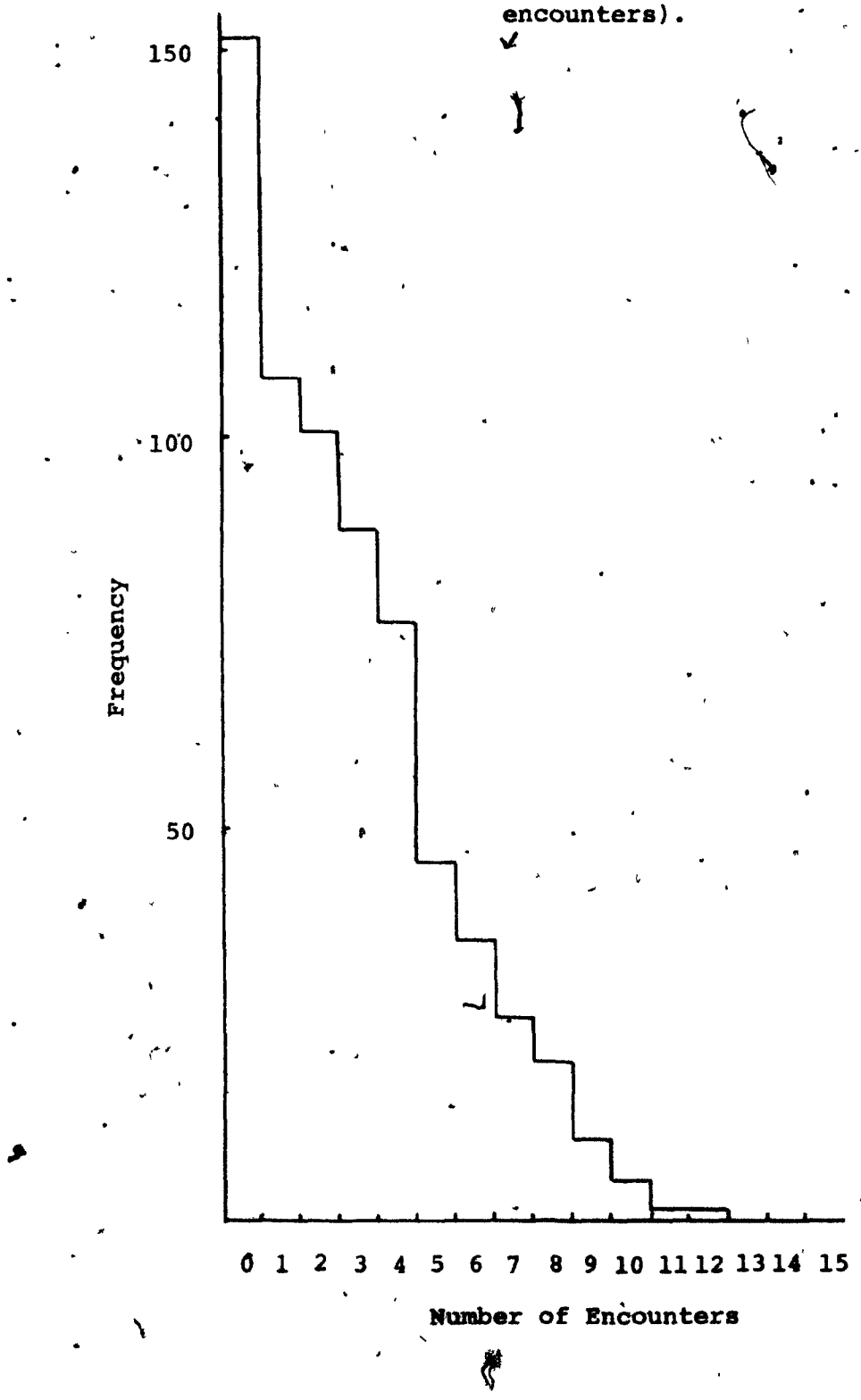


Table #17: Mean Experimental Test Weight for Each Family of Juvenile Homarus americanus in Population #1 (calculated over all bouts).

Family	Mean Weight (grams)
D	0.271 ± 0.013
E	0.235 ± 0.015
F	0.208 ± 0.014
G	0.289 ± 0.018
H	0.221 ± 0.012
I	0.280 ± 0.018
J	0.217 ± 0.011
K	0.239 ± 0.013
L	0.223 ± 0.018
M	0.269 ± 0.026
N	0.122 ± 0.015
O	0.139 ± 0.009
P	0.158 ± 0.009
Q	0.0167 ± 0.014

Table #18: Mean Experimental Test Weight for Each Family of Juvenile Homarus americanus in Population #2 (calculated over all bouts).

Family	Mean Weight (grams)
AA	0.394 ± 0.047
CC	0.313 ± 0.037
DD	0.465 ± 0.039
EE	0.355 ± 0.035
FF	0.124 ± 0.014
GG	0.126 ± 0.008
HH	0.328 ± 0.040
II	0.299 ± 0.037
JJ	0.254 ± 0.021
KK	0.143 ± 0.016
LL	0.213 ± 0.017
MM	0.215 ± 0.021
NN	0.162 ± 0.017
OO	0.203 ± 0.014
PP	0.114 ± 0.005
QQ	0.168 ± 0.013
RR	0.181 ± 0.012

Table #19: Family Means (calculated over all bouts) of Aggression Parameters for Juvenile Homarus americanus in Population #1.

Family	Latency (min)	Duration (sec)	Frequency
D	1.640 ± 0.401	7.576 ± 0.274	1.951 ± 0.113
E	1.802 ± 0.362	10.359 ± 0.351	1.878 ± 0.121
F	1.898 ± 0.460	12.915 ± 0.200	3.477 ± 0.097
G	2.748 ± 0.478	11.478 ± 0.334	2.449 ± 0.138
H	2.754 ± 0.292	7.776 ± 0.278	1.445 ± 0.113
I	1.331 ± 0.262	13.325 ± 0.285	2.666 ± 0.115
J	2.544 ± 0.330	6.737 ± 0.278	1.801 ± 0.121
K	2.940 ± 0.301	2.684 ± 0.239	1.217 ± 0.124
L	1.566 ± 0.358	9.848 ± 0.322	2.155 ± 0.146
M	2.386 ± 0.421	11.756 ± 0.501	2.589 ± 0.224
N	2.867 ± 0.637	2.831 ± 0.667	0.976 ± 0.235
O	2.017 ± 0.506	4.795 ± 0.499	1.858 ± 0.229
P	2.387 ± 0.609	3.246 ± 0.575	1.361 ± 0.269
Q	3.667 ± 0.502	3.216 ± 0.632	0.950 ± 0.247

Table #20: Family Means (calculated over all bouts) of Aggression Parameters for Juvenile Homarus americanus in Population #2.

Family	Latency (min)	Duration (sec)	Frequency
AA	1.992 ± 0.263	14.456 ± 0.398	2.180 ± 0.094
CC	1.483 ± 0.348	17.375 ± 0.418	3.297 ± 0.184
DD	2.700 ± 0.300	11.354 ± 0.343	1.614 ± 0.125
EE	1.727 ± 0.429	31.362 ± 0.530	3.221 ± 0.195
FF	2.993 ± 0.546	2.476 ± 0.539	0.990 ± 0.213
GG	2.157 ± 1.093	17.138 ± 1.729	3.864 ± 0.702
HH	1.493 ± 0.318	9.805 ± 0.318	2.408 ± 0.149
II	1.807 ± 0.306	27.991 ± 0.353	2.924 ± 0.147
JJ	2.346 ± 0.417	11.962 ± 0.636	1.664 ± 0.185
KK	1.347 ± 0.358	17.065 ± 0.597	3.371 ± 0.247
LL	3.153 ± 0.378	6.599 ± 0.534	1.226 ± 0.155
MM	1.960 ± 0.412	8.263 ± 0.409	1.790 ± 0.156
NN	2.202 ± 0.363	7.559 ± 0.471	2.206 ± 0.178
OO	2.948 ± 0.563	6.456 ± 0.844	1.509 ± 0.318
PP	2.055 ± 1.046	21.760 ± 1.843	2.673 ± 0.791
QQ	2.715 ± 0.493	4.906 ± 0.516	1.474 ± 0.213
RR	2.373 ± 0.358	10.531 ± 0.486	1.945 ± 0.179

IV. Correlations

Correlations were performed to determine the level of relationship between the various parameters of aggression and quantitative traits such as growth rate and mortality. Significant associations between these variables could be of considerable importance in the establishment of selective breeding programs.

Each of the 3 parameters of aggression was correlated in turn with the mean weighted and transformed growth rate, the 125 day mortality value (used instead of the 150 day mortality values in order that families from population #1 could be included in the calculations), and each of the remaining 2 parameters. The Pearson product moment correlation coefficients and their levels of significance are reported in table #21. Confidence limits were established by first converting the sample r to a z value using the formula,

$$z = 1/2 \ln \left[\frac{1 + r}{1 - r} \right]$$

setting confidence to z, and retransforming the limits back to the appropriate scale. Standard errors are not reported for samples of less than 500; the distribution of sample values for r (when N is less than 500) is highly asymmetrical when ρ, the parametric correlation coefficient, does not equal 0 (Sokal & Rolf,1969).

The high level of correlation between the various parameters of aggression is expected, and requires no further explanation.

The positive correlation between mortality and growth rate is statistical confirmation of a qualitative observation. The increase in death rate among the faster growing families may be associated with moulting difficulties; almost all of the deaths appeared to result from an inability of the juveniles to escape from the shed exoskeleton. The precise nature of this 'disease' is unknown and may or may not occur predominantly among laboratory reared populations.

There is no significant correlation between fourth stage weight and mortality, or fourth stage weight and 150 day weight.

There is a notable lack of association between the parameters of aggression and growth rate or mortality. A weak correlation exists between duration and growth rate, but this relationship lacks conviction in the light of subsequent comparisons (see below).

Because the experiments for aggressive behaviour could only be performed on those individuals in any one family who were alive at the time of testing, it seemed likely that those animals surviving to 150 days were responsible for the bulk of the experimental data. Correlations were consequently performed between mean growth rate for survivors and the 3 parameters of

aggression. The results are listed in table #22. It will be seen that none of the parameters appear to be significantly related to the growth rate based only on 150 day survivors.

Table #21: Correlations Between Aggression Parameters (frequency and duration transformed ln[y + 1), latency untransformed), Mean Transformed and Weighted Growth Rate (ln), and Percent Mortality at 125 Days (post 4th stage moult).

	Weighted Mean Growth Rate	Mean Latency	Mean Frequency ln[y + 1]	Mean Duration ln[y + 1]
Mean Latency	r = -.220 .95 conf. lim. -.537 to .167 not signif.			
Mean Frequency	r = .273 .95 conf. lim. -.115 to .574 not signif.	r = -.754 S.E. = .477 N = 593 signif. .00001		
Mean Duration	r = .484 .95 conf. lim. .124 to .713 signif. at .05	r = -.694 S.E. = 1.209 N = 593 signif. .00001	r = .824 S.E. = .941 N = 685 signif. .00001	
Percent Mortality	r = .4416 .95 conf. lim. .073 to .687 signif. at .05	r = -.054 .95 conf. lim. -.414 to .317 not signif.	r = .257 .95 conf. lim. -.130 to .564 not signif.	r = .347 .95 conf. lim. -.036 to .733 not signif.

Table #22: Correlations Between Aggression Parameters and Mean Weighted and Transformed (ln) Growth Rate for Those Juvenile Homarus americanus Surviving to 150 Days.

	Mean Growth Rate
Mean Latency	r = -.3009 .95% confidence limits -.743 to .156 not significant
Mean ln[y + 1] Transformed Frequency	r = .058 .95% confidence limits -.396 to .504 not significant
Mean ln[y + 1] Transformed Duration	r = .291 .95% confidence limits -.165 to .735 not significant

Table #23: Correlations Between Fourth Stage Weight and 150 Day Weight (post 4th stage moult); and Between Fourth Stage Weight and Survivorship (actual number of days surviving), For Juvenile Homarus americanus (calculations based on total of both populations).

	Fourth Stage Weight
150 Day Weight	r = .0285
	.95% confidence limits
	-.174 to .228
	not significant
Survivorship	r = -.037
	.95% confidence limits
	-.125 to .052
	not significant

Table #24: Summary of Results

Object of Analysis	Method Employed	Data Used	Results
1. Fourth Stage Weight			
(a) Genetic component to fourth stage weight.	Model II single classification anova.	Individual 4th stage weights.	Significant family difference at 0.001
(b) Heritability of 4th stage weights.	$2 (V_A / V_P)$, where V_A is additive genetic variance and V_P is total phenotypic var.	Individual 4th stage weights; estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$.	$h^2 = 0.805 \pm 0.166$
2. Growth Rate			
(a) Genetic component to growth rate.	Model II single classification anova.	Weighted family mean growth rate, calc. as regression of weight on time.	Population #1 sig. family diff. at 0.001; Population #2 at 0.001
(b) Heritability of growth rate	$2 (V_A / V_P)$	Estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$ for growth rate.	Population #1, $h^2 = 0.707 \pm 0.234$; Population #2, $h^2 = 0.540 \pm 0.171$

Table #24: continued

Object of Analysis	Method Employed	Data Used	Results
(c) Difference between individually and group reared juveniles.	T-test for equality of means of two samples.	Family growth rate calculated as regression of ln mean weight at each time, on time.	Population #1 signif. at 0.001; Population #2 signif. at 0.05
(d) Genetic component to growth rate for 150 day survivors.	Model II single classification anova.	Family mean of growth rate for 150 day survivors, calculated as reg. of weight on time.	Population #1 not significant Population #2 signif. at 0.01
(e) Heritability of growth rate for 150 day survivors.	$2 (V_A / V_P)$	Estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$ for growth rate of 150 day surviv.	Population #1 $h^2 = .138 \pm .173$ Population #2 $h^2 = .513 \pm .225$
3. 150 Day Weights			
(a) Genetic component to 150 day weight.	Model II single classification anova for unequal sample size.	150 day weight (post 4th stage moult).	Population #1 signif. at 0.05; Population #2 signif. at 0.001

Table #24: continued

Object of Analysis	Method Employed	Data Used	Results
(b) Heritability of 150 day weight.	2 (V_A / V_P)	Estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$ for 150 day weight.	Population #1 h^2 = .045 ± .114 Population #2 h^2 = .516 ± .225
4. Mortality			
(a) Genetic component to mortality.	Analysis of var. modified for binomial data.	Population #1; % mort. at 125 days; Population #2; arcsine trans. of % Mort. at 150 d.	Population #1 no signif. diff. Population #2 signif. at 0.01
(b) Heritability of mortality.	Ratio of genotypic var. to mean.	Population #1; % mort. at 125 days; Population #2; arcsine trans. of % mort. at 150 d.	Population #1 h^2 = .040 ± .043 Population #2 h^2 = .225 ± .101
(c) Differences in mortality between individually and group reared juveniles.	T-test for equality of means of two samples.	Population #1; % mort. at 125 days for family; Pop. #2, % mort. for fam at 150 days.	Population #1 signif. at 0.05 Population #2 signif. at 0.01

Table #24: continued

Object of Analysis	Method Employed	Data Used	Results
5. Parameters of Aggression			
(a) Genetic component to latency.	Two level nested anova for unequal sample size.	Mean family latency based on individ. bouts.	Significant at 0.01
(b) Heritability of latency.	2 (V_A / V_P)	Estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$ for latency.	h^2 = .126 ± .027
(c) Genetic component to frequency.	Two level nested anova for unequal sample size.	Mean family freq. based on individ. bouts.	Significant at 0.01
(d) Heritability of frequency.	2 (V_A / V_P)	Estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$ for frequency.	h^2 = .109 ± .050
(e) Genetic component to duration.	Two level nested anova for unequal sample size.	Mean family dur. based on ind. bouts.	Significant at 0.01
(f) Heritability of duration.	2 (V_A / V_P)	Estimated $\hat{\sigma}^2_A$ and $\hat{\sigma}^2_W$ for duration.	h^2 = .086 ± .045
6. Correlations summarized in tables 21,22, and 23.			

Discussion

I. Growth Rate

(a) Fourth Stage Weight

There appears to be a conspicuous lack of relationship between the weight of a fourth stage lobster and any of the other parameters measured. In view of the fact that fourth stage weight varies so significantly among families, this may at first seem surprising. However, a moment of reflection will show why this is so.

The heritability of fourth stage weight is very high ($h^2 = 0.805$). The standard error for both mean fourth stage weight and heritability is in turn, relatively low, suggesting that there is a high level of consistency within families for this trait, and that the realized heritability should not deviate too markedly from the calculated estimate. What this should mean biologically is that maternal investment in egg production is fairly symmetrical, and assuming that a certain upward limit exists with respect to the number of eggs produced, females with more available energy produce larger eggs, and probably larger offspring. Fourth stage weight may well be a 'maternal effect' in the true mammalian sense of the word, however, the lack of correlation between this trait and other parameters measured still justifies the assumption that maternal effects are for the large part unimportant in the estimation of genetic

variation in invertebrate species.

(b) Growth Rate and 150 Day Weight

That 150 day weight and growth rate show similar patterns and values for the partitioning of variance and heritability is almost inevitable. The only time that this would not be so, given the high level of observed mortality, is if the growth rate differed significantly across the temporal range of the observation period. The goodness of fit for the calculated regression as an estimate of average daily gain ($\bar{R} > 0.90$ for all families) showed that growth rate was reasonably constant, at least within the first 150 days of development. The 150 day weight is simply an accumulated growth rate, and should therefore vary accordingly.

The pattern of growth varied from population number one to population number two. As the two groups were taken from the same offshore population (albeit in two consecutive years), and environmental conditions within the laboratory were as constant as possible, the population differences are thought to relate primarily to the difference in quality of the brine shrimp fed in the two years; the second batch was of much higher grade (qualitative assessment). For this reason, the analyses were performed separately for each population.

The estimated variation between families for growth rate is highly significant for both populations, and the

mean upper limit for the heritability, $h^2 = 0.625$, is very high. This estimate is inflated somewhat beyond earlier estimates for growth in juvenile Homarus (Fairfull & Haley, 1981; Hedgecoek et al,1976), but generally concurs with trends established in previous studies. Fairfull and Haley found that "heritabilities of growth traits were moderate to high with a few exceptions", and that there was a general family consistency across environments. This suggests the presence of a large component due to genetic variability and supports the conclusion reached in Fairfull's study that the potential for artificial selection is good. The predicted response for growth rate in population two, given the assumption that S_A^2 (between families) is predominantly additive, is

$$R = i\sigma_A h = (1.75)(\sqrt{0.082})(\sqrt{0.54})$$
$$= 0.396$$

which translates to a genetic improvement of 1.445×10^{-3} grams per day; i is the intensity of selection and depends only on the proportion of the individuals to be included in the selected group (Falconer,1960), in this case, 10 percent of a large (N = 500) population (Table for i from Becker, 1975). This is a reasonably good return for one generation of selection.

There is some doubt however, as to how much of the available genetic variability can be attributed to strictly additive effects. A high heritability estimate (in the

broad sense) may be indicative of traits that are positively correlated with fitness (Broashurst,1979). Such traits are maintained in nature by either 'directional' or 'stabilizing' selection. If directional selection is the driving force, the proportion of dominance and epistatic to additive variation is likely to be large, and the high estimate of heritability does not provide a completely reliable measure of potential for response to artificial selection. (This could be empirically tested by reproducing the experiments using half sib rather than full sib families, thereby eliminating the component due to dominance completely and reducing the epistatic variance by half. Practical difficulties have thus far made the acquisition of half sib families impossible.)

In choosing new species for 'domestication', consideration must be given to the "...long-term genetic adaptability ...", or evolutionary potential of the population (Nelson,1977). According to Nelson (1977), "... one expects heritability |in the narrow sense| to be higher in members of evolutionary flourishing and recently expanding groups in which there is great variation in the trait in question." There is little evidence to suggest that Homarus is an evolutionary 'flourishing' genus. It dates from the Cretaceous period and contains only two extant species, the European vulgaris and the American americanus (Glaessner,1969). In addition, biochemical estimates of overall allelic variance in offshore and inshore populations

of Homarus americanus have revealed consistently low levels of heterozygosity, the proportion falling in the range of 0.05 to 0.06 for all loci studied (Tracey et al,1975). These findings apply to both within and between population comparisons. Although these estimates cannot be directly related to any particular quantitative traits it seems probable that "...decapod crustaceans ... with greater size, mobility and perhaps degree of homeostatic control than other invertebrates ...", and living in fairly stable environments "...rely less upon structural gene variation and more on either behavioural and physiological regulation (Selander and Kaufman,1973) or upon regulatory gene variation to achieve population consonance ..." (Nelson,1977).

Whether the observed levels of homozygosity have been achieved through long term directional selection, or possibly through inbreeding due to overfishing |between 70 and 90 percent of the legal sized inshore lobsters are taken each year (Cobb,1976)| is unclear, but the homogeneity across all populations thus far examined suggests the former. In either case, a high proportion of the estimated heritability is likely to be attributable to nonadditive genetic factors, including the effects due to common maternal environment, which are relatively inflated in homozygous populations.

If the proportion of additive variance is reduced even by half, giving heritability estimates as low as 0.3, the response may still be worthy of consideration. However, Hedgecock et al (1976) found that an heritability of 0.3

translated into an expected maximum gain of one month in the estimated time to marketable size in one generation of selection at an intensity of 25 percent. This may be acceptable over the long term, particularly if fairly economical practices of selection such as mass selection could be employed, but for more immediate results it may be more profitable to aim at maximizing productivity through environmental manipulation.

Some consideration has also been given to the possibility of producing hybrids between the American and European species of Homarus. Preliminary results indicate taht hybrids produce values for growth rate which are intermediate between the two parents, suggesting that the interspecific variation may well be additive (Carlberg at al, 1978). This outbreeding may provide an immediate approach to improving the heterozygosity, but in the absence of heterosis can not be expected to produce long term genetic gains through selection , particularly in view of the estimated interspecific genetic identity coefficient of 0.902 (Hedgecock et al,1977).

The difference in growth rate between those animals reared collectively and those confined to individual containers is quite dramatic. These differences reflect within family variability and serve to emphasize the importance of environment in determining the rate of growth. Unfortunately, the large gains made in growth among communally reared individuals are presently outweighed by

the high level of mortality due to intraspecific aggression and cannibalism.

It appears that high growth rate, at least under laboratory conditions, is positively correlated with high levels of mortality. There are several alternative ways of explaining this association.

The two traits may be genetically linked and stabilized at suboptimal levels by opposing forces of natural selection. According to Lerner (1954), "Attempts to shift populations too rapidly and too far from adapted mean values for specific traits, either by artificial selection or by changes in the breeding system, are counteracted by natural selection which is directed towards the maintenance of a phenotypic balance between fitness-determining characters. This behaviour is a product of the previous evolutionary history of the population. The resistance of natural selection can be overcome when new balanced combinations based on the utilization of free genetic variability arise." Therefore, although genetic variability may be preserved by linkage, it is inaccessible for either trait unless the linkage groups can be disrupted.

It is difficult to imagine a genetic system whereby two such vital developmentally and biochemically mediated processes could have been stabilised at low levels through natural selection after so long an evolutionary history. Even the most tightly linked genes are subject to periodic recombination, and nature is not remiss in seizing upon such

valuable genetic opportunities. If the two traits are in fact linked in apposition, the genetic balance must be associated with a phenotypic, or physiological balance, the disruption of which results in a reduced coefficient of selection. That is, a departure from the homeostatic mean is accompanied by an overall reduction in fitness. Evidence exists for the operation of this mechanism in the correlation of growth rate and mortality in Homarus. Nearly every death (no statistics available) which occured over the course of the experiment in both populations could be attributed directly to an inability of the moulting animal to cast its old skeleton. Similar results were reported in earlier studies (Fairfull and Haley,1981) although alternate forms of death were thought to result from experimentally induced trauma. There is a fairly high biological probabilty for the existence of a physiological association between high growth rate and moulting difficulties.

A second possibility is that the optima for the two traits may be established by a coincident correlation with an environmental trait such as nutrition. If a particular nutrient is missing from a diet based solely on brine shrimp, it seems likely that this deficiency would be manifest more quickly in faster growing animals, particularly if it interfered with moulting.

The third possibility is that the optima may be pleiotropically regulated through association with a third trait. There is however, no immediate evidence as to what

this trait might be, and more obvious explanations are available.

II. Mortality

There are three principal known causes of death in laboratory reared populations of Homarus americanus. The nutritional requirements of the animal are poorly understood and there is always the problem of inadequate or incomplete sources of protein, minerals, and vitamins. This factor is particularly important during the intermoult period, when the major accumulation of organic reserves occurs, and at moulting proper, when epicuticle formation and mineral deposition are in progress.

The second major contribution to the demise of the laboratory population is the growth of a parasitic fungus which attacks the soft tissues of the lobster's body and actually reduces it to a chitinous shell packed full of mycelium (Herrick,1911). Little expertise is needed in the diagnosis of this ailment, as the mycelial filaments are readily observed on the lobster and cages.

The third main cause of death is cannibalism, a factor induced in laboratory populations by enforced cohabitation. The lobster has evolved an evolutionarily sophisticated behavioural mechanism to deal with social encounters under natural conditions, but the unnatural and often crowded artificial rearing enclosures force a breakdown of this system.

It is apparent from this experiment that mortality differed markedly between population #1 and population #2 (see fig. #12). This large interpopulation variation is unlikely to be the result of genetic differences, as the two groups originated from the same wild, and presumably panmictic, population. The quality of food was thought to account for the difference in growth rate between population number one and number two. (Admittedly, this was a qualitative assessment based on experience and a perceived improvement in the appetite and enthusiasm demonstrated by juveniles for the second batch of Artemia.) Given the positive correlation between mortality and growth rate, already discussed, it seems reasonable to extend a common explanation for the observed interpopulation variance.

There was no qualitative or statistical variation in survivorship among families in population number one. However, the differences in percent mortality among families in population number two were quite significant, and the heritability was estimable, if not particularly high. These results agree well with those reported by Fairfull and Haley (1981) for postmoult mortality under similar environmental conditions (i.e. temperature and diet). A genetic component may be attributed to this differential mortality observed among families within the same population. If a biological flexibility exists with respect to an individual's ability to utilize available nutrients, some families may be more well equipped to deal with an all

brine shrimp diet than others. Such low levels of genetic variability might however, be difficult to distinguish from phenotypic plasticity.

The apparent family differences may be nothing more than an artifact due to the association between mortality and growth rate. If real additive differences exist they will remain inaccessible to artificial selection unless the linkage groups can be disrupted.

The differential mortality between group and individually reared animals of the same family is explained in a more unequivocal manner. The deaths in the troughs were directly related to aggressive and cannibalistic interactions between cohabiting family members. Even in the second part of the experiment, where animals were provided with shelters, the numbers were rapidly reduced, a size differential was quickly established, and the ultimate product was one very large, very antagonistic animal. As the individually housed animals were not provided with an opportunity for social interaction (except in the controlled conditions of the experiments on aggression), this form of mortality was impossible.

The third form of mortality, fungal infection, did not appear to be a significant factor in the death rate during this experiment. Constant cleaning and removal of food prior to the establishment of fungal mats seemed to adequately discourage mycelial growth.

III. Aggression

Aggression, or intraspecific conflict, occurs in all animals which have the apparatus to inflict injury (Southwick,1970). It would seem that natural selection has placed a high value on agonistic behaviour, and it is obvious that it must have evolved independently many times. It would therefore be unreasonable to assume that the genetic mechanisms regulating such behaviour are identical, or even similar, in all organisms. There are also, as mentioned earlier, fundamental physiological (both genetically and environmentally mediated) differences in aggression, depending upon the environmental and motivational stimuli.

Aggression is an important factor in the social organization of many species. Natural selection operates to define an environmentally appropriate set of adaptive reactions for each social unit, in the same way that it draws upon individual cell reactions in determining individual organization (Collias,1970). Although the underlying genetic architecture may elude definition, it seems probable that for a particular type of aggressive behaviour, for example maternal defense or social dominance, the nature of the selective pressures would be similar. It may therefore be possible to make some broad generalizations with respect to the nature of the variance components which might predominate for a given behavioural phenotype, and an

educated guess as to how the trait would respond to selection given that some genetic contribution to the variance can be detected.

Highly significant family differences for all three parameters of aggression were found in this experiment. There was also a high level of correlation among the three parameters. This might suggest linkage, or pleiotropic gene action if the parameters were thought to measure different traits, however, there is reason to believe that all three are merely different aspects of the same behaviour (see literature review). The high level of statistical association is therefore to be expected. The estimated Heritabilities are not large [the realized heritability in selection experiments for aggressive behaviour in other species is usually in the range of 0.3 or better (Ebert and Hyde,1976; Komai et al,1959)], but provide an indication that genetic variability for aggressive behaviour does exist.

Some confusion may arise at this point regarding the difference between strictly 'aggressive', and 'agonistic' behaviour. Agonistic behaviour is a whole ethogram, or behavioural phenotype, which is probably highly associated with fitness, and strongly canalized. It is a method of communicating, and deviations from a readily recognized (by conspecifics) pattern are maladaptive to say the least. Aggressive behaviour normally occurs only when this sophisticated system fails to resolve the situation without conflict. It indeed forms only one component of the

behavioural ethogram recognized as agonistic behaviour, and is delimited from other components by its implied intention of inflicting injury upon the opponent. Under natural circumstances the animals probably only rarely come to blows; aggression for its own sake is counterproductive.

A certain degree of behavioural flexibility is however, desirable; the ability to make differential response based on the nature of the stimulus is of selective value. Therefore, although high levels of aggression, or very low levels of aggression would be maladaptive, moderate levels of aggression are probably important. Moderate genetic variability should be maintained through stabilizing selection, and the principal component of this genetic variability is probably additive rather than dominant or epistatit.

There is also some reason to believe that the between family variance for aggression may have been compressed due to the presence of phenotypically discontinuous thresholds. This threshold effect, and conservative assumptions employed in the statistical analyses, suggest that the estimated heritabilities may actually represent a lower limit.

In spite of this genetic potential, practical difficulties may seriously limit the efficacy of a behaviourally oriented selection program. The results of the communal rearing experiment have demonstrated that even among families expressing the lowest values for intraspecific

aggression, mortalities resulting from cannibalism approach 30 to 40 percent. The 'genotypically useful' range of variation does not at this point correspond to a 'phenotypically useful' range of behaviour. Given the long generation time |5 years in the wild to a present minimum of 2 years in the laboratory under optimum conditions (Hughes et al,1972)| and the necessity of employing complicated and expensive methods of selection based on family relationships, the estimated heritabilities are apt to produce a fairly slow response. It would seem that aiming to achieve economically useful levels of behavioural modification through artificial selection is a long term project at best.

There is no significant correlation of aggression with growth rate or survivorship. While a positive association between low aggression and economically valuable qualities would normally be desirable, it may be advantageous in this case that they are not. High levels of genetic correlation suggest linkage, or pleiotropic gene action; if high survivorship is associated with a low rate of growth, a positive correlation of low aggression with either trait would reduce the potential for a selective response. A correlation with low survivorship or low growth rate would of course be equally unproductive. It is just as well then, from the point of view of the breeder, that aggression seems to occur independently of these other characters.

Little has been said here of the possible effect of

genotype-environment interactions. Values for possible interactions could not be calculated, as only one environment was used for any given method of rearing. Fairfull (1980) found that "...genetic relationships for traits between environments were positive and high...". However, the results reported here and the forgoing discussion are understood to apply explicitly to the conditions described for this experiment.

IV. Summary and Conclusions

Growth rate, measured under the conditions described in this thesis, appears to contain genetically useful amounts of intraspecific variability. However, the immediate potential for genetic improvement using methods of artificial selection is questionable. This may be attributed in part to the unknown nature of the genetic variability (dominant and epistatic versus additive), and to the apparently positive correlation of growth rate and mortality. If these traits are genetically or physiologically linked in apposition, the variability maintained in the past by natural selection is inaccessible to the breeder. This condition will prevail until linkage groups can be effectively disrupted.

Artificial selection is therefore not recommended for either of the above traits at the present time. Environmentally induced phenotypic variation is considerable, and breeders would be well advised to concentrate on the maximization of growth rate and minimization of mortality through environmental manipulation. When the optimal response has been achieved through environmental modification, a limited mass selection program might be profitably introduced to maximize fitness within the most economically auspicious range of environments. A premature reduction of the available genetic variability may result in the selection of animals which, although expressing superior characteristics in less favourable conditions, may

be less than maximally adapted for optimal environments.

It is impossible at this point to make very broad generalizations concerning the probable efficacy of a program designed to reduce intraspecific aggression in communally reared stocks of Homarus americanus. It is likely that some genetic variance of an additive nature does exist for this trait, and that over the long term response to selection might be good, if very slow, The practicality of such a program is likely to be rejected however on economical grounds, due to the protracted generation time and rearing complications associated with the maintenance of full or half sib families.

Literature Cited

Abplanalp,H., and L.F. Marrou. Genotype-environment interactions in field tests of poultry. Poultry Science, 40,1368 (1961).

Ache,B.W. The sensory physiology of spiny and clawed lobsters. In: Workshop on Lobster and Rock Lobster Ecology and Physiology,Division of Fisheries and Oceanography Circular #7, Commonwealth Scientific and Industrial Research Organization, Australia (1977).

Aiken,D.E. Molting and growth in decapod crustaceans with particular reference to the molt cycle. In: Workshop on Lobster and Rock Lobster Ecology and Physiology, Division of Fisheries and Oceanography Circular #7, Commonwealth Scientific and Industrial Research Organization, Australia (1977).

Arechiga,H.,and R. Atkinson. The eye and some effects of light on locomotor activity in Nephrops norvegicus. Marine Biology, (Woods Hole Mass.) 32, 63-76 (1975).

Atema,J. Neurophysiology. In: Workshop on Lobster and Rock Lobster Ecology and Physiology, Division of Fisheries and Oceanography Circular #7, Commonwealth Scientific and Industrial Research Organization, Australia (1977).

Ayala,F.J., J.W. Valentine, T.E. DeLaca, and G.S. Zumwalt. Genetic variability of the Antarctic brachiopod Liothyrella notorcadensis and its bearing on mass extinction hypotheses. Journal of Paleontology, 49, 1-9 (1975).

Barnes,R.D. Invertebrate Zoology. 3rd Ed. W.B. Saunders & Co.,(1974).

Becker,W.A. Manual of Quantitative Genetics. 3rd Ed. Washington State University Press, Washington State University, Pullman, Washington (1975).

Beerman,W. Cytological aspects of information transfer in cellular differentiation. In: E.Bell (ed.) Molecular and Cellular Aspects of Development,New York, Harper,

204-212 (1965).

Biswas,D.K., and J.V. Craig. Genotype environment interactions in chickens selected for high and low social dominance. Poultry Science, 49, 681-692 (1970).

Britten,R.J., and E.H. Davidson. Gene regulation for higher cells: a theory. Science, 165, 349-357 (1969).

Broadhurst,P.L. The experimental approach to behavioural evolution. In: Theoretical Advances in Behaviour Genetics, Royce,J.R., and L.P. Mos (ed.), 43-100, Sijthoff and Noordhoff, Alphen aan den Rijn, the Netherlands (1979).

Bruell,J.H. Behavioural heterosis. In: Hirsch J. (ed.) Behaviour-Genetic Analysis, Mcgraw-Hill Book Co., New York, (1967).

Buchsbaum,R. Animals Without Backbones. Penguin Books Ltd., Harmondsworth, Middlesex, England (1938).

Busser,J., A. Zweep, and G.A. van Oortmerssen. Variability in the aggressive behaviour of Mus musculus domesticus, its possible role in population structure. In: J.H.F. van Abeelen (ed.) The Genetics of Behaviour, North-Holland Publishing Company, Amsterdam, Oxford, American Elsevier Pub. Co. Inc., New York, (1974).

Carlberg et al. Unpublished data.(1978).

Caspari,E.W. Gene action as applied to behaviour. In: J. Hirsch (ed.), Behaviour-Genetic Analysis, McGraw-Hill Book Co., New York, (1967).

Catlett,R.H. An evaluation of methods for measuring fighting behaviour with special reference to Mus musculus. Animal Behaviour, 9, 8-10, (1961).

Ciaranello,R.D., A.Lipsky, and J.Axelrod. Association between fighting behaviour and catecholamine biosynthetic activity in two inbred mouse sublines. Proc. Natl. Acad. Sci., U.S.A., 71, 3006-3008, (1974).

Clarac,F., W. Wales, and M. Laverack. Stress detection at the autotomy plane in the decapod crustacea. II.

The function of receptors associated with the cuticle of the basi-ischiopodite. Z. Vgl. Physiol., 73, 383-407, (1971).

Cobb,J.S. Dominance status and moult order in lobsters (Homarus americanus). Marine Behav. Physiol., 3, 119-124, (1975).

Cobb,J.S. The American Lobster: The Biology of Homarus americanus. U. of Rhode Island, Marine Technical Report #49, (1976).

Cobb,J.S. Review of the habitat behaviour of the clawed lobsters (Homarus and Nephrops). Workshop on Lobster and Rock Lobster Ecology and Physiology, Division of Fisheries and Oceanography Circular #7, Commonwealth Scientific and Industrial Research Organization; Australia, (1977).

Cobb,J.S. Social conditions increase intermoult period in juvenile lobsters Homarus americanus. Journal Fish. Research Board, 31, 12, (1974).

Cohen,M.J. The function of receptors in the statocyst of the lobster Homarus americanus. J. Physiol. London, 130, 9-34, (1955).

Cohen,M. The response patterns of single receptors in the crustacean statocyst. Proc. Roy. Soc. B., 152, 30-49, (1960).

Colins,R.A. Aggression in mice selectively bred for brain weight. Behav. Genet., #1, 169 (1970).

Collias,N.E. Aggressive behaviour and evolution. In: C.S. Southwick (ed.) Animal Aggression: Selected Readings, van Nostrand Reinhold Company, New York, (1970).

Craig,J.V., and R.A. Baruth. Inbreeding and social dominance ability in chickens. Animal Behaviour,13, 109,(1965).

Craig,J.V., M.L. Jan, C.R. Polley, A.L. Bhagwat, and A.D. Dayton. Changes in relative aggressiveness and social dominance associated with selection for early egg production in chickens. Poultry Science,54, 1647-1658, (1975).

Craig,J.V.,L.L. Ortman, and A.M. Guhl. Genetic selection for social dominance ability in chickens. Animal Behaviour, 13, 114-131, (1965).

Dawson,W.M. Inheritance of wildness and tameness in mice. Genetics, 17,296, (1932).

DeFries,J.C. Quantitative genetics and behaviour: overview and perspective. In: Behaviour-Genetic Analysis, J. Hirsch (ed.), McGraw-Hill Book Co., New York, (1967).

DeFries,J.C.,A.R. Kuse, and S.G. Vandenberg. Genetic correlations, environmental correlations, and behaviour. In: J.R. Royce, and L.P. Mos (ed.) Theoretical Advances in Behaviour Genetics, sijthoff and Noordhoff, Alphen aan den Rijn, the Netherlands, (1979).

DeFries,J.C., and G.E. McLearn. Social dominance and Darwinian fitness in the laboratory mouse. American Naturalist, 104, 408-411, (1970).

Dunham,P. Some effects of group housing on the aggressive behaviour of the lobster Homarus americanus.Journal Fish. Research Bd. Canada, 29, 598-601, (1972).

Ebert,P.D., and J.S. Hyde. Selection for agonistic behaviour in wild female Mus musculus.Behav. Genet., 6,291-304, (1976).

Eleftheriou,B.E., D.W. Bailey, and V.H. Denenberg. Genetic analysis of fighting behaviour in mice. Physiol. Behav., 13, 773-777, (1974).

Eleftheriou,B.E., and R.L. Church. Brain levels of serotonin and norepinephrine in mice after exposure to aggression and defeat. Physiol. Behav., 3, 977, (1968).

Ennis,G.P. Food, feeding and condition of lobsters, Homarus americanus, throughout the seasonal cycle in Bonavista Bay, Newfoundland. J.Fish. Research Bd. Canada,30, 1905-1909, (1973a).

Ennis,G.P. Behavioural responses to changes in hydrostatic pressure and light during larval development of the lobster Homarus gammarus. J. Fish. Research Bd. Can., 30, 1349-1360, (1973b).

Ennis, G.P. Observations on hatching and larval release in the lobster Homarus americanus. J. Fish Res. Bd. Can., 32, 2210-2213, (1975).

Ewbank,R., and M.J. Bryant. Aggressive behaviour amongst groups of domesticated pigs kept at various stocking rates. Animal Behaviour, 20, 21 (1972).

Fairfull,W., and L.E. Haley. The early growth of artificially reared American lobsters I. Genetic parameters within environments. Theor. Appl. Genet., 60, 269-273, (1981).

Falconer,D.S. Introduction to Quantitative Genetics. Reprinted 1976, Longman Group Ltd. Ronald Press Co. New York.

Fredericson,E. Aggressiveness in female mice. J. Comp. Psychol., 45, 254-257, (1952).

Fuller,J.L., and W.R. Thompson. Foundations of Behaviour Genetics. C.V. Mosby Co., St. Louis, (1978).

Gandleman, Mice: postpartum aggression elicited by the presence of an intruder. Hormone Behaviour, 3, 23-28, (1972).

Ginsberg,B.E. Genetic parameters in behavioural research. In: J. Hirsch (ed.) Behaviour-Genetic Analysis, McGraw-Hill Book Co., New York, (1967).

Ginsberg,B.E. Intra-uterine effects on behavioural capacities in mice. Paper presented at the Symposium of the Effects of Intrauterine Experience. XIX International Congress of Psychology, London, (1969).

Glaessner,M.F. Decapods. In: R.C. Moore (ed.) Treatise on Invertebrate Paleontology, R400 - R533, (1969).

Green,E.L., and M.C. Green. Transplantation of ova in mice. Journal of Heredity, 50, 109-114, (1959).

Guhl,A.M., J.V. Craig, and C.D. Mueller. Selective breeding for aggressiveness in chickens. Poultry Science, 39, 970, (1960).

Hazlett,B. Antennule chemosensitivity in marine decapod crustacea. J. Animal Morphol. Physiol., 18, 1-10, (1971).

Hedgecock,D. Biochemical genetic markers for broodstock

identification in aquaculture. Proceedings of the Eighth Annual Meeting World Mariculture Society, (1977).

Hedgecock,D., and K. Nelson. Components of growth rate variation among laboratory cultured lobsters (Homarus). Proc. World Mariculture Soc., 9, (1977).

Hedgecock,D., K. Nelson, R.A. Shleser, and M.L. Tracey. Biochemical genetics of lobsters (Homarus) II. Inheritance of allozymes in Homarus americanus. Journal of Heredity, 66, 114-118, (1975).

Hedgecock,D., K. Nelson, J.Simons, and R. Shleser. Genic similarity of American and European species of the lobster Homarus. Biological Bulletin, Woods Hole, 152, 41-50, (1977).

Hedgecock,D., K. Nelson, and R. Shleser. Growth differences among families of the lobster, Homarus americanus. Proc. Seventh World Mariculture Soc. Meeting, 347-361, (1976).

Henderson,N.D. Adaptive significance of animal behaviour: the role of gene-environment interaction. In: J.R. Royce and L.P. Mos (ed.) Theoretical Advances in Behaviour Genetics, Sijthoff and Noordhoff, Alphen aan den Rijn, the Netherlands, (1979).

Herrick,F.H. Natural history of the American lobster. Bulletin of the U.S. Bureau of Fisheries, Vol. XXIX, (1911).

Hirsch,J. Behaviour genetic analysis at the chromosome level of organization. In: J. Hirsch (ed.) Behaviour-Genetic Analysis, McGraw-Hill Book Co., (1967).

Hoffman,R.S., P.J. Dunham, and P.V. Kelly. Effects of water temperature and housing conditions upon the aggressive behaviour of the lobster Homarus americanus. J. Fish. Research Bd. Can., 32, 713-718 (1975).

Hook, E.B. Behavioural implications of the human XYY genotype. Science,179, 139-150, (1973).

Horn,J.M. Aggression as a component of relative fitness in four inbred strains of mice. Behav. Genet., 4, 373-

381, (1974).

Hughes,J.T., J.J. Sullivan, and G. Tchobanoglous. Enhancement of lobster growth. Science, 177, 1110-1111, (1974).

Jacob,F., and J. Monod. Genetic regulatory mechanisms in the synthesis of proteins. Journal of Molecular Biology,3, 318-356, (1961).

Jarvik,L.F., V. Klodin, and S.S. Matsuyama. Human aggression and the extra Y chromosome. Am. Psychol., 28, 674-682, (1973).

Johnson,G.B. Enzyme polymorphism and biosystematics: the hypothesis of selective neutrality. In: R.E. Johnston, P.W. Frank, and C.E. Michener (ed.) Annual Review of Ecology and Systematics, 4, 93-116, (1973).

Karli,P., F. Eclancher, M. Vergnes, J.P. Chaurand, and P. Schmitt. Emotional responsiveness and interspecific aggressiveness in the rat: interactions between genetic and experiential determinants. In: J.H.F. van Abeelan (ed.) The Genetics of Behaviour, North Holland Publishing Co. Amsterdam, Oxford, American Elseviier Publishing Co. Inc., New York, (1974).

Kennedy,D. Physiology of photoreceptor neurons in the abdominal nerve cord of the crayfish. Journal of Gen. Physiol., 46, 551-572, (1963).

Kessler,S., G.R. Elliott, E.K. Orenberg,and J.D. Barchas. A Genetic Analysis of Aggressive Behaviour in Two Strains of Mice. Behaviour Genetics,7, 313-321 (1977).

Lagerspetz,K.M.J. Studies on the Aggressive Behaviour of Mice. Ann. Acad. Sci. Ser. B., 131,3,1 (1964).

Lagerspetz,K.M.J., and K.Y.H. Lagerspetz. Changes in the Aggressiveness of Mice Resulting from Selective Breeding, Learning, and Social Isolation. Scan. J. Psychol., 12, 241 (1971).

Lagerspetz,K.M.J., and K.Y.H. Lagerspetz. Genetic Determination of Aggressive Behaviour. In: J.H.F. van Abeelen (ed.) The Genetics of Behaviour, North Holland Publishing Co., Amsterdam, Oxford, (1974).

Lagerspetz,K.Y.H., R. Tirri, and K.M.J. Lagerspetz. Neurochemical and Endocrinological Studies of Mice Selectively Bred for Aggressiveness, Scand. J. of Psychol. 9, 157 (1968).

Laverack,M.S. Responses of Cuticular Sense Organs of the Lobster, Homarus vulgarus, I. Comp. Bioch. Physiol., 5, 319-325 (1962a).

Laverack,M.S. Responses of Cuticular Sense Organs of the Lobster, Homarus vulgarus II. Comp. Bioch. Physiol., 6, 137-145 (1962b).

Laverack,M.S. Response of Cuticular Sense Organs of the Lobster, Homarus vulgarus III. Comp. Bioch. Physiol., 10, 261-272 (1963b).

Laverack,M.S. Aspects of Chemoreception in Crustacea. Comp. Bioch. Physiol., 8, 141-151 (1963a).

Laverack,M., and D. Ardill. The Innervation of the Aesthetasc Hairs of Panulirus argus. Q.J. Microsc. Sci., 106, 45-60 (1965).

Lee, Yen-Pai, and J.V. Craig. Evaluation of Egg-Laying Strains of Chickens in Different Housing Environments: Role of Genotype by Environment Interactions. Poultry Science, Vol.60, 1769-1781 (1981).

Lee, Yen-Pai, and J.V. Craig. Agonistic and Nonagonistic Behaviours of Pullets of Dissimilar Strains of White Leghorns When Kept Separately and Intermingled. Poultry Science, Vol.60, 1759-1768 (1981).

Lerner,I.M. Genetic Homeostasis. New York, John Wiley and Sons (1954).

Levine,L., G.E. Barsal, and C.A. Diakow. Interaction of Aggressive and Sexual Behaviour in Mice. Animal Behaviour, 13, 272-280 (1965).

Levine,L., C.A. Diakow, and G.E. Barsel. Interstrain Fighting in Mice. Animal Behaviour,13, 52 (1965).

Lindzey,G., M. Manosevitz, and H. Winston. Social Dominance in the Mouse. Psychon. Sci., 5, 451-452 (1966).

Lorenz,K. On Aggression. London and Metheden & Co. (1963).

Lowry,D.C., and H. Abplanalp. Genetic Adaptation of White Leghorn Hens to Life in Single Cages. Brit. Poultry Sci., 11, 117-131 (1970).

Lowry,D.C., and H. Abplanalp. Social Dominance Difference, Given Limited Access to Common Food, Between Hens Selcted and Unselected for Increased Egg Production. Brit. Poultry Sci., 13, 365-376 (1972).

Lush,J.L. Animal Breeding Plans.1st ed. Ames Iowa, Collegiate Press, (1937).

Manning, A. An Introduction to Animal Behaviour. Addison-Wesley Pub. Co., Reading Mass. (1967).

Marks,H.L., C.H. Moore, N.R. Gyles, H.R. Wilson, W.F. Krueger, L.D. Tindell, W.A. Johnson, L.J. Dreesen, W.L. Blow, and P.B. Siegel. Genotype Environment Interactions in Egg Production Stocks of Chickens. 3 Main Effects and Interactions of Parent-Flock Location, Parent Stock and Growing Location. Poultry Science, 48, 1543-1552 (1969).

Mather,K. Polygenic Inheritance and Natural Selction. Biol. Reviews, 18, 32-64 (1943).

Mather,K. Genetical Control of Stability in Development.

Heredity, 7, 297-336, (1953).

Mather,K. Evolution in polygenic systems. Academia Nazionale dei Lincei, 47, 131-152, (1960).

Mather,K. Variability and selection. Proceedings of the Royal Society, Series B, 164, 328-340, (1966).

Mather,K. Genetical Structure of Populations. Chapman and Hall, (1973).

Maynard,D. Integration in crustacea ganglion. In: G.Hughes (ed.) Nervous and Hormonal Mechanisms of Integration, 111-149, Academic: New York. Sym. Soc. Exp. Biol. 20, (1966).

McBride,G. Behavioural measurement of social stress. In: E.S.E. Hafez (ed.) Adaptation of Domestic Animals, Lea and Febiger, Philadelphia, (1968).

McLeese,D.W., and D.G. Wilder. The Activity and Catchability of the lobster (Homarus americanus) in relation to temperature. J. Fish. Res. Bd. Can., 15, 1345-1354, (1958).

Messeri,R., B.E. Eleftheriou, and A. Oliverio. Dominance behaviour: a phylogenetic analysis in the mouse. Physiol. Behav., 14, 53-58, (1975).

Moyer,K.E. Kinds of aggression and their physiological basis. Comm. Beh. Biol., A, 2, 65-87, (1968).

Mukherjee,T.K., P. Horst, D.K. Flock, and J. Peterson. Sire x location interactions from progeny tests in different countries. Brit Poultry Science, 21, 123-129, (1980).

Nelson,K. Genetic considerations in selecting crustacean species for aquaculture. Proc. Eighth Annual Meeting World Mariculture Society, (1977).

Nunnemacher,R. The fine structure of optic tracts of decapoda. In: C. Bernhard (ed.) The Functional Organization of the Compound Eye, 363-375, Pergamon, Oxford, (1966).

Ohno,S. So much "junk" DNA in our genome. In: H.H. Smith (ed.) Evolution of Genetic Systems, Vol. 22, New York, Gordon and Breach, (1972).

Omenn,G.S., and A.G. Motulsky. Biochemical genetics and the evolution of human behaviour. Office of Education, Rye, New York, October 3-8, (1971).

Ortman,L.L., and J.V. Craig. Social dominance in chickens modified by genetic selection - physiological mechanisms. Animal Behaviour, 16, 33-37. (1968).

Ott,L. An Introduction to Statistical Methods and Data Analysis. Wadsworth Publishing Co. Inc., Belmont,Cal., Duxbury Press, (1977).

Parsons,P.A. The Genetic Analysis of Behaviour. Methuen and Co. Ltd. London, (1967).

Roberts,R.C. Implications of behaviour genetics for genetics. In: J.Hirsch (ed.) Behaviour-Genetic Analysis, McGraw-Hill Book Co., New York, (1967).

Robertson,A., and I.M. Lerner. The heritability of all-or-none traits: viability of poultry. Genetics, 34, 395-411, (1949).

Sastry,A.N., and L. Zeitlin-Hale. Factors affecting survival of communally reared larval and juvenile lobsters, Homarus americanus. Recent Advances in Aquaculture, Technical Session, Abstracts, U. of Rhode Island, Kingston, (1975).

Scott,J.P. Aggression. University of Chicago Press, Chicago, (1958).

Scott,J.P., and E. Freericson. The causes of fighting in mice and rats. Physiol. Zool., 24, 273-309, (1951).

Scrivener,J.C.E. Agonistic behaviour of the american lobster Homarus americanus. Fish Res. Bd. of Can. Tech Rep., 235, 114 pages, (1971).

Searle,S.R. Linear Models. John Wiley and Sons, New York, Chichester, Brisbane, Toronto, (1971).

Selander,R.K., and S.Y. Yang. Biochemical genetics and behaviour in wild house mouse populations. In: G. Lindzey and D.D. Thiessen (ed.) Contributions to Behaviour Genetic Analysis: The Mouse as a Prototype, New York, Appleton - Century - Crofts, (1970).

Selander,R.K., and D.W. Kauffman. Genetic variability and strategies of adaptation in animals. Proc. of the National Academy of Sciences, 70, 1875-1877, (1973).

Selmanoff,M.K., S.C. Maxson, and B.E. Ginsberg. Chromosomal determinants of intermale aggressive behaviour in inbred mice. Behav. Genet., 6, 53-69, (1976).

Shelton,R.G.J., and M.S. Laverack. Receptor hair structure and function in the lobster. J. Exp. Mar. Biol., 4, 201-210, (1970).

Shleser,R., and G. Tchobanoglous. The American lobster as a model for the continuous production of quality seafood through aquaculture. MTS Journal, 8, 4-8, (1974).

Siegel, P.B. Evidence of a genetic basis for aggressiveness and sex drive in the white plymouth rock cock. Poultry Science, 38, 115-118, (1959).

Sokal,R.R., and F.J. Rohlf. Biometry. W.H. Freeman & Co., (1969).

Southwick,C.H. Effects of maternal environment on aggressive behaviour of inbred mice. Commun. Behav. Biol., 1, 129-132,(1968).

Southwick,C.H. Conflict and violence in animal societies. In: C.H. Southwick (ed.) Animal Aggression, van Nostrand Reinhold Co., New York, (1970).

Southwick,C.H., and L.H. Clark. Interstrain differences in aggressive behaviour and exploratory activity of inbred mice. Commun. Behav. Biol., 1, 49-59, (1968).

St. John,R.D., and P.A. Corning. Maternal aggression in mice. Behav. Biol., 9, 635-639, (1973).

Tamm,G.R., and J.S. Cobb. Diel ecdysis rhythms in juvenile lobsters Homarus americanus. J. Fish. Res. Bd. Can., 33, 819-821, (1976).

Tamm,G.R., and J.S. Cobb. Behaviour and the crustacean molt cycle: changes in aggression of Homarus americanus. Science, 200, (1978).

Tazaki,K. Sensory units responsive to osmotic stimuli in

the antennae of the spiny lobster Panulirus japonicus. Comp. Biochem. Phsiol., 51A, 647-653, (1975).

Tellegen,A., and J.M. Hörn. Primary aggressive motivation in three inbred strains of mice. J. Comp. Physiol. Psychol. 78, 297-304, (1972).

Thiessen, D. Biological trends in behaviour genetics. In: J.R. Royce and L.P. Mos (ed.) Theoretical Advances in Behaviour Genetics, Sijthoff and Noordhoff, Alphen aan den Rijn, the Netherlands, (1979).

Tindell,D., and J.V. Craig. Effects of social competition in laying house performance in chickens. Poultry Science, 38, 95-105, (1959).

Tindell,L.D., C.H. Moore, N.R. Gyles, H.R. Wilson, W.A. Johnson, L.J. Dreesen, W.L. Blow, W.F. Krueger, and P.B. Siegel. Genotype-environment interactions in the egg production stocks of chickens I. The importance of stock by location and stock by year interactions. Poultry Science, 46, 1077-1088, (1967).

Tracey,M.L., K. Nelson, D.Hedgecock, R.A. Shleser, and M.L. Pressick. Biochemical genetics of lobsters: genetic variation and the structure of American lobster (Homarus americanus)population. Journal Fish Res. Bd. Canada,32, 2091-2101, (1975).

Utsurikawa,N. Tempermental differences between outbred and inbred strains of the albino rat. J. Animal Behav., 7, 111, (1917).

Vale,J.R., D. Ray, and A. Vale. The interaction of genotype and exogenous neonatal androgen: agonistic behaviour in female mice. Behavioural Biology, 7, 321, (1972).

Vale,J.R., A. Vale, and J.P. Harley. Interaction of genotype and population number with regard to aggressive behaviour. Comm. Behav. Biol., 6, 209, (1971).

van Abeelen, J.H.F. Direct genetic and maternal influences on behaviour and growth in two inbred mouse strains. Behaviour Genetics, 10, #6, (1980).

van Oortmerssen,G.A. Biological significance, genetics, and evolutionary origin of variability within and between inbred strains of mice. A behaviour genetic study. Behaviour, 38, 1, (1970).

van Oortmerssen,G.A., and Th. C.M. Bakker. Artificial selection for short and long attack latencies in wild Mus musculus domesticus. Behav. Genet., 11, #2, 115 - 126, (1981).

Wahlsten,D. A critique of the concepts of heritability and heredity in behavioural genetics. In: J.R. Royce and L.P. Mos (ed.) Theoretical Advances in Behaviour Genetics, Sijthoff and Noordhoff, Alphen aan den Rijn, the Netherlands, (1979).

Walter,R.O., and J.B. Hamilton. Supermales (YY sex chromosome) and androgen treated XY males: competition for m[redacted]ng with female killifish Oryzias latipes. Animal Behav., 18, 128-131, (1970).

Weltman,A.S., A.M. Sackler, R. Schwartz, and H. Owens. Maternal effects on behaviour and white blood cells of isolated female mice. Life Science, 9, 291-300, (1970).

White,M., M. Smith, and D.A. Edwards. Fighting in Female mice as a function of the size of the opponent. Psychon. Sci., 16, 14-15, (1969).

Wiersma,C. and K. Yanagisawa. On types of interneurons responding to visual stimulation present in the optic nerve of the rock lobster, Panulirus interruptus. J. Neurobiol., 2, 291-309, (1971).

Wright,S. On the roles of directed and random changes in gene frequency in the genetics of populations. Evolution, 2, 279-294, (1948).

Yerkes,R.M. The heredity of savageness and wildness in rats. J. Animal Behav., 7, 11, (1913).

Zeitlin-Hale,L. Effects of environmental manipulation on the locomotor activity and agonistic behaviour of juvenile American lobsters, Homarus americanus. MSc. Thesis, University of Rhode Island, (1975).

Appendix I

Single Classification Analysis of Variance

The structure of variation in a single classification, Model II Anova for single pair matings is,

$$y_{ij} = \mu + \alpha_i + \epsilon_{ij}$$

where μ is the common mean, α_i is the effect of the ith mating, and ϵ_{ij} is a random error term due to individual variance within families. These effects are all random, normally distributed, independent, and have expected variance equal to zero (Becker,1975).

The analysis of variance table is set up as follows:

Source of Variation	df	SS	MS	F_s	Expected MS
$\bar{Y} - \bar{\bar{Y}}$ Among Groups	a-1	SS_G	$\frac{SS_G}{a-1}$	$\frac{MS_G}{MS_W}$	$\sigma_W^2 + n_o\sigma_A^2$
$Y - \bar{Y}$ Within Groups	$\sum^a n_i - a$	SS_W	$\frac{SS_W}{\sum^a n_i - a}$		σ_W^2
$Y - \bar{\bar{Y}}$ Total	$\sum^a n_i - 1$	$SS_G + SS_W$			

n_i = the number of individuals in the ith group

n_o = the average sample size, computed as

$$\frac{1}{a-1}\left[\sum^a n_i - \frac{\sum^a n_i^2}{\sum^a n_i}\right]$$

which is always just less than $\bar{n}$.

The variance components are estimated as follows:

$$S_A^2 = \frac{\text{MS groups - MS within}}{n_o}$$

$$S_W^2 = \text{MS within}$$

(Sokal & Rolf,1969)

The level of significance is determined by comparing the computed $F_s = \frac{\text{MS groups}}{\text{MS within}}$ with the tabulated F value for $a-1$ and $\sum n_i - a$ degrees of freedom, and the desired α, where α represents the acceptable probability of committing a type I error (the rejection of a true null hypothesis).

(a) Analysis of Variance and Variance Components for Fourth Stage Weight.

(i) Anova Table

Source of Variation	df	SS	MS	F_s
Among Families	20	0.01143	0.00057	15.572***
Within Families	435	0.01595	0.0000367	
Total	455			

$F_{.001[20,120]} = 2.53$ (conservative critical value employed to avoid interpolation).

(ii) Variance Components

$$S_W^2 = 0.0000367$$

$$S_A = 0.00002468$$

(b) Analysis of Variance and Variance Components for Growth Rate.

(i) Anova Tables

- Population #1

Source of Variation	df	MS	F_s
Among Families	9	I.875	16.024 ***
Within Families	265	0.11708	
Total	274		

$F_{.001[9,120]}$ = 3.10 (conservative critical value employed to avoid interpolation).

Computational Formulae for Weighted Least Squares Analysis:

1. Overall Mean = $\frac{\sum_{ij} w_i x_i}{\sum_{ij} w_i}$

where w_i is the weighting factor for the ith individual in the jth family, and x is the transformed (ln) growth rate.

2. Variance Among Families = $\frac{\sum_{j} w_j (\bar{x}_{j[w]} - \bar{\bar{x}}_{ij})^2}{a - 1}$

where w_j is the weighting factor for the jth family (N), $\bar{x}_{j[w]}$ is the weighted transformed (ln) mean for each family, and $\bar{\bar{x}}_{ij}$ is the overall mean.

3. Variance Within Families = $\frac{\sum_{ij} w_i (x_i - \bar{x}_{j[w]})^2}{\sum_{ij} w_i}$

- Population #2

Source of Variation	df	MS	F_s
Among Families	16	1.42387	6.403***
Within Families	233	0.22236	
Total	249		

$F_{.001[15,120]} = 2.78$ (conservative critical value employed to avoid interpolation).

(ii) Variance Components

- Population #1

$S_W^2 = 0.11708$

$S_A^2 = 0.06398$

- Population #2

$S_W^2 = 0.22236$

$S_A^2 = 0.08218$

(c) Analysis of Variance and Variance Components for 150 Day Weights.

(i) Anova Tables

- Population #1

Source of Variation	df	SS	MS	F_s
Among Families	3	0.0609	0.0203	2.74*
Within Families	79	0.5856	0.0074	
Total	82			

$F._{05[3,79]}$ = 2.72 (by harmonic interpolation).

- Population #2

Source of Variation	df	SS	MS	F_s
Among Families	15	2.1298	0.14199	3.087*
Within Families	82	3.7714	0.04599	

$F._{05[15,60]}$ = 1.84 (conservative critical value employed to avoid interpolation).

(ii) Variance Components

- Population #1

S_W^2 = 0.0074

S_A^2 = 0.000629

- Population #2

S_W^2 = 0.04599

S_A^2 = 0.01600

(d) Analysis of Variance and Variance Components for Growth Rates for Those Individuals Surviving to 150 Days.

(i) Anova Tables

- Population #1

Source of Variation	df	SS	MS	F_s
Among Families	3	3.2459	1.0819	2.593 (ns)
Within Families	83	34.6262	0.41718	
Total	97			

$F_{.05[3,120]} = 2.68$ (conservative critical value employed to avoid interpolation)

- Population #2

Source of Variation	df	SS	MS	F_s
Among Families	15	94.374	6.2916	3.0685**
Within Families	82	168.130	2.0504	
Total	97			

$F_{.01[15,60]} = 2.35$ (conservative critical value employed to avoid interpolation).

(ii) Variance Components

- Population #1

$S_W^2 = 0.41718$

$S_A^2 = 0.03083$

- Population #2

$S_W = 2.0504$

$S_A = 0.7067$

Appendix II

Test of Equality of Means When Variances Are Heterogeneous

Equality, or 'homoscedasticity' of variance in a group of samples is an important prerequisite for testing means in a conventional analysis of variance. If variances are nonhomogeneous, but certain observable relationships exist between the mean and standard deviation, the original data can often be stabilized by systematically converting it to a new scale (transformation), (Ott & Lyman,1977). If the data are inherently heteroscedastic, approximate tests can be conducted using methods which presuppose the inequality of variance.

One such test is the modified t-test for equality of means of two samples.

$$t'_s = \frac{(\bar{Y}_1 - \bar{Y}_2) - (\mu_1 - \mu_2)}{\sqrt{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}}$$

where t'_s is expected to be distributed approximately as t' when the null hypothesis is true (Sokal & Rolf,1969).

The critical value of t'_α is computed as

$$t'_\alpha = \frac{t_{\alpha[\nu_1]}\frac{s_1^2}{n_1} + t_{\alpha[\nu_2]}\frac{s_2^2}{n_2}}{\frac{s_1^2}{n_1} + \frac{s_2^2}{n_2}}$$

which is equivalent to t_α when sample sizes are equal.

Appendix III

The Heritability of All or None Traits

It is the value of the underlying variate, that is, the genotypic value, which actually determines whether or not a given threshold character will be expressed. Lerner and Robertson (1949) have devised a method for calculating heritability in terms of the probability of expression, where the phenotypes assume the values 0 and 1 for 'absence' of, and 'expression' of the character respectively.

Represent the genotypic values for expression of a given character as $p_1, p_2, \ldots, p_n$ with a mean of $\bar{p}$ and a variance σ_p^2 . That is, the probability of expressing the trait is p_1 or p_2 etc. for the genotype under a strictly defined set of environmental circumstances. The corresponding phenotype is then $(p_m + e_m)$ where e_m represents all nongenetic or 'environmental' components of the variance, and the phenotype assumes the value of 0 or 1 by definition, and $p_m + e_m = 1$. The mean genotype for those individuals expressing the trait is then

$$\bar{p} = \frac{\sum_{m=1}^{n} p_m(p_m + e_m)}{\sum_{m=1}^{n} (p_m + e_m)}$$

$$= \frac{\sum_{m=1}^{n} (p_m)^2 + p_m e_m}{\sum_{m=1}^{n} (p_m + e_m)}$$

However, the expected values of e_m, and therefore $p_m e_m$, are by definition zero, as the postulated environmental deviation, or variance, over all genotypes is equal to zero. Therefore,

$$E({}_1\bar{p}) = \frac{E\left(\sum_{m=1}^{n} (p_m)^2\right)}{E\left(\sum_{m=1}^{n} p_m\right)}$$

where E represents the 'expected' value for the terms in parentheses.

The expected response after one generation of mating among individuals expressing the trait is

$$\begin{aligned} \text{Response} &= E\,({}_1\bar{p} - \bar{p}) \\ &= \frac{E\left(\sum_{m=1}^{n} p_m^2\right)}{E\left(\sum_{m=1}^{n} p_m\right)} - \bar{p} \\ &= \frac{n(\bar{p}^2 + \sigma_p^2)}{n\bar{p}} - \bar{p} \\ &= \frac{\sigma_p^2}{\bar{p}} \end{aligned}$$

The selection differential employed in this situation is $1 - \bar{p}$, the difference between the mean phenotype of those expressing the trait, (1), and the mean phenotype of the entire population prior to selection ($\bar{p}$), because by definition the mean phenotype in a population is assumed to be equal to the mean genotypic value (Falconer, 1960).

Therefore,

$$\text{Heritability} = \frac{\text{Genetic Gain}}{\text{Phenotypic Selection Differential}}$$

$$= \frac{\sigma_p^2}{\bar{p}\ (1 - \bar{p})}$$

which provides an estimate of heritability in terms of the genotypic variance of expressivity, and mean expressivity (Robertson & Lerner, 1949).

The genotypic variance can be determined using the method of analysis of variance for binomial data. (A valid objection to the application of anova methods to binomial data is that the variance is not independent of the mean; this can be overcome by use of the arcsine transformation. If the genotypic variance is sufficiently small, or homogeneous, the analysis may be applied directly to the raw data.)

If the data is set up as follows:

Offspring of Family	Total #	Expressing Trait
I	n_1	a_1
II	n_2	a_2
N	n_n	a_n

the appropriate anova computations can be performed in a

manner analogous to that of continuous variation.

The between class sum of squares becomes

$$\sum_{1}^{N} \frac{a_i^2}{n_i} - \frac{(\Sigma a_i)^2}{\Sigma n_i}$$

with N-1 degrees of freedom.

The expected value for the sum of squares becomes

$$(N - 1)\ \bar{p}\ (1 - \bar{p}) + n_0 r \sigma_p^2$$

where r is the coefficient of relationship, and

$$n_0 = \Sigma n_i - \frac{\Sigma n_i^2}{\Sigma n_i} \div (N - 1)$$

the correction term for unequal sample size.

The component for within class variation is $\bar{p}\ (1 - \bar{p})$, by definition of the variance of a binomial population. The genetic variance is therefore

$$\sigma_p^2 = \frac{\left(\sum \frac{a_i^2}{n_i} - \frac{(\Sigma a_i)^2}{\Sigma n_i} \right)}{r n_0} - (N - 1)\ \bar{p}\ (1 - \bar{p})$$

and heritability becomes

$$\frac{\sigma_p^2}{\bar{p}(1 - \bar{p})} = \frac{\left[\frac{\sum \frac{a_i^2}{n_i} - \frac{(\Sigma a_i)^2}{\Sigma n_i}}{\bar{p}\ (1 - \bar{p})} - (N - 1) \right]}{r n_0}$$

(a) Analysis of Variance and Heritability Calculations for Mortality - Population #1.

Source of Variation	df	MS	F
Among Families	12	0.3168	1.634 (n.s.)
Within Families	411	0.1939	
Total	423		

$F_{.05[12,\infty]} = 1.75$ (conservative critical value employed to avoid interpolation).

$$h^2 = \left[\frac{\sum \frac{a_i^2}{n_i} - \frac{(\Sigma a_i)^2}{\Sigma n_i}}{\hat{p}\,(1 - \bar{p})} - (N - 1) \right] \Big/ rn_0$$

$$= \frac{\frac{(3.802)}{0.1939} - 12}{189.547}$$

$$= 0.040$$

Standard Error $h^2 = \sigma_t / r$, where $t = 1/2\ h^2$, r is the coefficient of relationship of full sibs, and

$$\sigma_t = [1 + (n_0 - 1)t]\,(1 - t)\sqrt{\frac{2}{n(n-1)(N-2)}}$$

$$= 0.02125$$

The standard error of heritability = 0.0425 .

(b) Analysis of Variance and Heritability Calculations for Mortality - Population #2.

Source of Variation	df	MS	F
Among Families	17	0.7440	3.724*
Within Families	344	0.1998	
Total	361		

$F_{.05[15,120]} = 1.75$ (conservative critical value employed to avoid interpolation).

$$h^2 = \frac{\left[\dfrac{\sum \frac{a_i^2}{n_i} - \frac{(\Sigma a_i)^2}{\Sigma n_i}}{\bar{p}\,(1-\bar{p})} - (N-1)\right]}{rn_0}$$

$$= \frac{\dfrac{11.9043}{0.1998} - 16}{162.942}$$

$$= 0.267$$

Standard Error $h^2 = \sigma_t / r$

$\sigma_t = 0.0557 \quad r = 0.5$

S.E. $h^2 = 0.114$

Appendix IV

Least Squares Analysis of Covariance

The independent and adjusted effects (weight adjusted for animals and animals adjusted for weight) of weight and animals were calculated using the general linear model for analysis of covariance

$$y_i = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \ldots + \beta_n x_n + \varepsilon$$

where x_1 represents the covariate weight, x_2 to x_n represent the effects of animals 2 to n, and ε represents a randomly distributed error term with mean 0 and variance σ^2.

Calculations were performed on each family for which the number of bouts exceeded the number of independent variables, and for each of the 3 parameters of aggression. Residuals were plotted for each calculation and found to be completely random.

Although the covariance tables are included at the end of this appendix for the sake of completeness, a summary of the results is tabled in table A1.

The effect of animals was significant in one family only for both variables, duration and frequency. Although this is certainly well within the bounds of reasonable expectation for Type I error, it is interesting to note that all significant tests were the result of only 3 test periods, 2 of which occured within a two day interval. The response

of any given animal for repeated measurements within a short span of time would be more similar than for an equal number of tests performed over longer intervals and incorporating more environmental and physiological variables.

The high proportion of families showing a significant effect due to animals on latency is thought to be an artifact due to small sample size. In families HH, QQ, and KK, particularly the latter 2, the majority of tests were performed on the same few animals. If 1 or 2 animals out of a test population of from 2 to 8 members respond similarly on several occassions, the level of association is inordinately elevated. In view of the small sample size and the fact that the level of significance was not particularly high, (0.05), the decision was made to reject the hypothesis that animals have a significant effect on latency.

It was therefore concluded that the effect due to animals on the parameters of aggression was negligable, and that a regular analysis of variance could be appropriately applied to the data.

Weight is seen to affect 3 families out of 21 for duration, 2 families out of 21 for frequency, and 1 out of 17 for latency. In each of these families, the increased order of aggressive activity coincides with the temporal sequence of growth. All families experienced some test periods where overall aggression was heightened or reduced with respect to others. This variation was expected as general activity is known to fluctuate with the

physiological state of the animal. As these within family family groups were equal in age and moulted at regular and coincident intervals, their behaviour varied more or less uniformly between test periods. In a series of 5 tests extended over an interval of several months, some families show more aggressive activity in the later experiments, some in the middle, and some at the beginning. This order of activity is purely random overall. All 3 aggression parameters have been shown to be totally independent of order. The correlation between latency and order is -0.035, between frequency and order is -0.072, and between duration and order is 0.126 . These coefficients are not significant at 0.05 level of acceptance.

It is evident that in situations where the increasing order of activity is in the forward direction, the level of association between growth (weight), and aggressive behaviour will be artificially increased. It was concluded therefore, that weight is not a significant factor in the expression of aggressive behaviour (in situations where animals are of similar weight).

Table A1: Summary of Results of Covariance Analysis Between Weight, Animal Effect, and Each of Three Parameters of Aggression for All Families in Population #1 and #2.

Parameters (y)	Duration	Frequency	Latency
Animal Effect	1 family sig. (I) out of 21 families.	1 family sig. (E) out of 21 families.	4 families sig. (I;EE;KK;QQ) out of 17 fam.
Weight	3 families sig. (AA;KK; MM) out of 21 families.	2 families sig. (EE;MM) out of 21 families.	1 family sig. (HH) out of 17 families.

Covariance Tables for Appendix IV

Family D, Population #1:

Duration

Source of Variation	df	SS	MS	F
Weight & Animals	26	78.08248	3.00317	1.7127 (ns)
Animals adj for Weight	25	73.12962	2.92518	1.6682 (ns)
Weight adj for Animals	1	5.67570	5.67570	3.2369 (ns)
Residual	16	28.05542	1.75346	
Total	42	106.13790		

Frequency

Source of Variation	df	SS	MS	F
Weight & Animals	26	14.34414	0.55170	1.379 (ns)
Animals adj Weight	25	14.24854	0.56994	1.425 (ns)
Weight adj Animals	1	1.11813	1.11813	2.795 (ns)
Residual	16	6.40058	0.40004	
Total	42	20.74472		

Family E, Population #1:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	21	69.80722	3.32415	1.247	(ns)
Animals adj Weight	20	60.06660	3.00333	1.127	(ns)
Weight adj Animals	1	2.60146	2.60146	0.976	(ns)
Residual	12	31.98640	2.66550		
Total	33	101.79362			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	21	12.63254	0.60155	3.500	**
Animals adj Weight	20	11.17294	0.55865	3.251	*
Weight adj Animals	1	0.02216	0.02216	0.129	(ns)
Residual	12	2.06229	0.17186		
Total	33	14.69483			

Family G, Population #1:

Duration

Source of Variation	df	SS	MS	F
Weight & Animals	24	64.05839	2.66910	0.766 (ns)
Animals adj Weight	23	59.16028	2.57219	0.739 (ns)
Weight adj Animals	1	0.43623	0.43623	0.125 (ns)
Residual	10	34.82442	3.48244	
Total	34	98.88281		

Frequency

Source of Variation	df	SS	MS	F
Weight & Animals	24	12.71248	0.52969	0.751 (ns)
Animals adj Weight	23	12.52035	0.54436	0.772 (ns)
Weight adj Animals	1	0.11041	0.11041	0.157 (ns)
Residual	10	7.05116	0.70512	
Total	34	19.76364		

Family H, Population #1:

Duration.

Source of Variation	df	SS	MS	F	
Weight & Animals	28	49.86447	1.78087	0.476	(ns)
Animals adj Weight	27	48.81648	1.80802	0.483	(ns)
Weight adj Animals	1	1.54807	1.54807	0.414	(ns)
Residual	8	29.92256	3.74032		
Total	36	79.78703			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	28	11.29835	0.40351	0.828	(ns)
Animals adj Weight	27	10.86305	0.40234	0.826	(ns)
Weight adj Animals	1	1.42493	1.42493	2.924	(ns)
Residual	8	3.89859	0.48732		
Total	36	15.19694			

Family I, Population #1:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	24	52.55063	2.18961	4.856	*
Animals adj Weight	23	48.07486	2.09021	4.636	*
Weight adj Animals	1	2.77863	2.77863	6.163	(ns)
Residual	5	2.25441	0.45088		
Total	29	54.80504			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	24	9.72188	0.40508	3.166	(ns)
Animals adj Weight	23	7.77578	0.33808	2.642	(ns)
Weight adj Animals	1	0.09236	0.09236	0.722	(ns)
Residual	5	0.63977	0.12795		
Total	29	10.36165			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	24	58.52646	2.43860	11.742	**
Animals adj Weight	23	52.74076	2.29308	11.041	*
Weight adj Animals	1	0.07498	0.07498	0.361	(ns)
Residual	5	1.03841	0.20768		
Total	29	53.85415			

Family J, Population #1:

Duration

Source of Variation	df	SS	MS	F
Weight & Animals	26	71.08917	2.73420	0.828 (ns)
Animals adj Weight	25	67.36487	2.69459	0.816 (ns)
Weight adj Animals	1	3.72430	3.72430	1.128 (ns)
Residual	23	75.96053	3.30263	
Total	49	147.04970		

Frequency

Source of Variation	df	SS	MS	F
Weight & Animals	26	15.08890	0.58034	0.806 (ns)
Animals adj Weight	25	13.95023	0.55801	0.775 (ns)
Weight adj Animals	1	0.00074	0.00074	0.001 (ns)
Residual	23	16.55769	0.71990	
Total	49	31.64659		

Latency

Source of Variation	df	SS	MS	F
Weight & Animals	26	78.84755	3.03260	0.456 (ns)
Animals adj Weight	25	78.61550	3.14462	0.473 (ns)
Weight adj Animals	1	2.04438	2.04438	0.307 (ns)
Residual	10	66.49689		
Total	36	145.34440		

Family K, Population #1:

Duration

Source of Variation	df	SS	MS	F
Weight & Animals	26	31.08698	1.19565	0.399 (ns)
Animals adj Weight	25	30.25218	1.21009	0.404 (ns)
Weight adj Animals	1	4.01147	4.01147	1.339 (ns)
Residual	10	29.96166	2.99617	
Total	36	61.04864		

Frequency

Source of Variation	df	SS	MS	F
Weight & Animals	26	9.16442	0.35248	0.395 (ns)
Animals adj Weight	25	8.96586	0.35863	0.401 (ns)
Weight adj Animals	1	1.07079	1.07079	1.199 (ns)
Residual	10	8.93295	0.89329	
Total	36	18.09737		

Latency

Source of Variation	df	SS	MS	F
Weight & Animals	26	72.46311	2.78704	0.579 (ns)
Animals adj Weight	25	64.29137	2.57165	0.534 (ns)
Weight adj Animals	1	22.84303	22.84303	4.746 (ns)
Residual	10	48.13349	4.81335	
Total	36	120.59660		

Family AA, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Animals & Weight	11	24.05616	2.18692	0.945	(ns)
Animals adj Weight	10	12.30090	1.23009	0.531	(ns)
Weight adj Animals	1	13.20322	13.20322	5.703	*
Residual	8	18.51959	2.31495		
Total	19	42.57575			

Frequency

Source of Variation	df	SS	MS	F	
Animals & Weight	11	0.95634	0.08694	0.326	(ns)
Animals adj Weight	10	0.88217	0.08822	0.331	(ns)
Weight adj Animals	1	0.33909	0.33909	1.272	(ns)
Residual	8	2.13333	0.26667		
Total	19	3.08967			

Latency

Source of Variation	df	SS	MS	F	
Animals & Weight	11	15.84828	1.44075	1.115	(ns)
Animals adj Weight	10	15.81373	1.58137	1.223	(ns)
Weight adj Animals	1	3.73959	3.73959	2.893	(ns)
Residual	8	10.34084	1.29260		
Total	19	26.18912			

Family CC, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Animals & Weight	13	20.67490	1.59038	0.366	(ns)
Animals adj Weight	12	10.01665	0.834721	0.192	(ns)
Weight Adj Animals	1	1.88223	1.88223	0.433	(ns)
Residual	7	30.41796	4.34542		
Total	20	51.09286			

Frequency

Source of Variation	df	SS	MS	F	
Animals & Weight	13	7.03970	0.54152	0.772	(ns)
Animals adj Weight	12	4.76283	0.39690	0.566	(ns)
Weight adj Animals	1	0.08905	0.08905	0.127	(ns)
Residual	7	4.90750	0.70107		
Total	20	11.94720			

Family DD, Population #2:

Duration

Source of Variation	df	SS	MS	F
Animals & Weight	16	31.60503	1.97531	0.492 (ns)
Animals adj Weight	15	30.67022	2.04468	0.510 (ns)
Weight adj Animals	1	0.42923	0.42923	0.107 (ns)
Residual	18	72.23508	4.01306	
Total	34	103.84011		

Frequency

Source of Variation	df	SS	MS	F
Animals & Weight	16	3.78842	0.23678	0.334 (ns)
Animals adj Weight	15	3.38745	0.22583	0.319 (ns)
Weight adj Animals	1	0.10111	0.10111	0.143 (ns)
Residual	18	12.74456	0.70803	
Total	34	16.53298		

Latency

Source of Variation	df	SS	MS	F
Animals & Weight	16	34.94674	2.18417	0.545 (ns)
Animals adj Weight	15	29.91765	1.99451	0.498 (ns)
Weight adj Animals	1	2.54227	2.54227	0.634 (ns)
Residual	18	72.12226	4.00679	
Total	34	107.069		

Family EE, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	6	17.25464	2.87577	1.287	(ns)
Animals adj Weight	5	16.60084	3.32017	1.486	(ns)
Weight adj Animals	1	0.56789	0.56789	0.254	(ns)
Residual	7	15.64082	2.23440		
Total	13	32.89546			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	6	4.71254	0.78542	5.144	*
Animals adj Weight	5	4.71111	0.94222	6.171	*
Weight adj Animals	1	0.00297	0.00297	0.019	(ns)
Residual	7	1.06884	0.15269		
Total	13	5.78138			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	6	19.95252	3.32542	1.725	(ns)
Animals adj Weight	5	17.99648	3.59930	1.867	(ns)
Weight adj Animals	1	3.68044	3.68044	1.909	(ns)
Residual	7	13.49516			
Total	13	33.44768			

Family HH, Population #2:

Duration

Source of Variation	df	SS	MS	F
Animals & Weight	18	36.43590	2.02422	0.723 (ns)
Animals adj Weight	17	35.10423	2.06495	0.738 (ns)
Weight adj Animals	1	0.91559	0.91559	0.327 (ns)
Residual	7	19.59906		
Total	25	56.03496		

Frequency

Source of Variation	df	SS	MS	F
Animals & Weight	18	10.02026	0.55668	0.853 (ns)
Animals adj Weight	17	10.01750	0.58926	0.903 (ns)
Weight adj Animals	1	1.27952	1.27952	1.961 (ns)
Residual	7	4.56746	0.65249	
Total	25	14.58772		

Latency

Source of Variation	df	SS	MS	F
Animals & Weight	16	55.44863	3.46554	3.048 *
Animals adj Weight	15	51.24490	3.54966	3.122 *
Weight adj Animals	1	7.06975	7.06975	6.218 *
Residual	9	10.23348	1.13705	
Total	25	65.68211		

Family II, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	8	15.25424	1.90682	2.917	(ns)
Animals adj Weight	7	15.20750	2.17250	3.324	(ns)
Weight adj Animals	1	0.13989	0.13989	0.214	(ns)
Residual	6	3.92191	0.65365		
Total	14	19.17645			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	8	2.91306	0.36413	2.127	(ns)
Animals adj Weight	7	2.91111	0.41587	2.430	(ns)
Weight adj Animals	1	0.30338	0.30338	1.772	(ns)
Residual	6	1.02702	0.17117		
Total	14	3.94008			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	8	13.02352	1.62794	1.457	(ns)
Animals adj Weight	7	12.95877	1.85125	1.657	(ns)
Weight adj Animals	1	0.17549	0.17549	0.157	(ns)
Residual	6	6.70361	1.11727		
Total	14	19.72713			

Family JU, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	10	50.87326	5.08733	1.109	(ns)
Animals adj Weight	9	49.41426	5.49047	1.196	(ns)
Weight adj Animals	1	19.76769	19.76769	4.309	(ns)
Residual	9	41.28795	4.58755		
Total	19	92.16121			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	10	6.11794	0.61179	1.125	(ns)
Animals adj Weight	9	5.60675	0.62297	1.145	(ns)
Weight adj Animals	1	0.97133	0.97133	1.786	(ns)
Residual	9	4.89501	0.54389		
Total	19	11.01295			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	10	35.13165	3.51316	1.020	(ns)
Animals adj Weight	9	34.60792	3.84532	1.116	(ns)
Weight adj Animals	1	0.00914	0.00914	0.003	(ns)
Residual	9	31.00643	3.44516		
Total	19	66.13808			

Family KK, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	10	32.85014	3.28501	4.992	(ns)
Animals adj Weight	9	26.70698	2.96744	4.510	(ns)
Weight adj Animals	1	15.29383	15.29383	23.243	*
Residual	2	1.31599	0.65799		
Total	12	34.16613			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	10	7.54981	0.75498	17.635	(ns)
Animals adj Weight	9	7.48674	0.83186	19.431	*
Weight adj Animals	1	0.63505	0.63505	14.834	(ns)
Residual	2	0.08562	0.04281		
Total	12	8.20741			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	10	19.88287	1.98829	26.439	*
Animals adj Weight	9	19.82094	2.20233	29.286	*
Weight adj Animals	1	0.60424	0.60424	8.035	(ns)
Residual	2	0.15041	0.07520		
Total	12	20.57559			

Family LL, Population #2:

Duration

Source of Variation	df	SS	MS	F
Weight & Animals	13	53.55964	4.11997	1.534 (ns)
Animals adj Weight	12	46.84502	3.90375	1.453 (ns)
Weight adj Animals	1	5.72354	5.72354	2.131 (ns)
Residual	6	16.11576	2.68596	
Total	19	69.6754		

Frequency

Source of Variation	df	SS	MS	F
Weight & Animals	13	6.54207	0.50324	2.341 (ns)
Animals adj Weight	12	5.51718	0.45977	2.139 (ns)
Weight adj Animals	1	0.50162	0.50162	2.334 (ns)
Residual	6	1,28965	0.21494	
Total	19	7.83172		

Latency

Source of Variation	df	SS	MS	F
Weight & Animals	13	41.18443	3.16803	1.446 (ns)
Animals adj Weight	12	29,48659	2.45722	1.122 (ns)
Weight adj Animals	1	8.33571	8.33571	3.805 (ns)
Residual	6	13.14439	2.19073	
Total	19	54.32882		

Family MM, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	12	42.63713	3.55309	2.739	(ns)
Animals adj Weight	11	25.66918	2.33356	1.799	(ns)
Weight adj Animals	1	13.21746	13.21746	10.191	*
Residual	9	11.67305	1.29701		
Total	21	54.31018			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	12	6.14361	0.51197	1.269	(ns)
Animals adj Weight	11	4.46134	0.40558	1.005	(ns)
Weight adj Animals	1	2.41110	2.41110	5.974	*
Residual	9	3.63222	0.40358		
Total	21	9.77583			

Family NN, Population #2:

Duration

Source of Variation	df	SS	MS	F
Weight & Animals	14	51.83506	3.70250	1.523 (ns)
Animals adj Weight	13	48.48315	3.72947	I.534 (ns)
Weight adj Animals	1	0.08435	0.08435	0.035 (ns)
Residual	7	17.01641	2.43092	
Total	21	68.85147		

Frequency

Source of Variation	df	SS	MS	F
Weight & Animals	14	9.69313	0.69237	1.741 (ns)
Animals adj Weight	13	9.33042	0.71773	1.805 (ns)
Weight adj Animals	1	0.00097	0.00097	0.002 (ns)
Residual	7	2.78361	0.39766	
Total	21	12.47674		

Latency

Source of Variation	df	SS	MS	F
Weight & Animals	14	46.47270	3.31948	1.619 (ns)
Animals adj Weight	13	46.47268	3.57482	1.743 (ns)
Weight adj Animals	1	2.59304	2.59304	1.264 (ns)
Residual	7	14.35462	2.05066	
Total	21	60.82732		

Family O6, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	6	38.48585	6.41431	1.932	(ns)
Animals adj Weight	5	36.30505	7.26101	2.187	(ns)
Weight adj Animals	1	0.00016	0.00016	0.000	(ns)
Residual	6	19.92183	3.32031		
Total	12	58.40768			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	6	7.14090	1.19015	1.501	(ns)
Animals adj Weight	5	6.30812	1.26162	1.591	(ns)
Weight adj Animals	1	0.00134	0.00134	0.002	(ns)
Residual	6	4.75674	0.79279		
Total	12	11.89764			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	6	28.08267	4.68044	1.318	(ns)
Animals adj Weight	5	22.22142	4.44429	1.251	(ns)
Weight adj Animals	1	1.04612	1.04612	0.294	(ns)
Residual	6	21.31350	3.55225		
Total	12	49.39617			

Family QQ, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	8	30.52170	3.81521	1.846	(ns)
Animals adj Weight	7	23.13663	3.30523	1.599	(ns)
Weight adj Animals	1	0.17394	0.17394	0.084	(ns)
Residual	8	16.53741	2.06718		
Total	16	47.05911			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	8	5.98677	0.74835	1.431	(ns)
Animals adj Weight	7	3.45631	0.49376	0.944	(ns)
Weight adj Animals	1	0.05609	0.05609	0.107	(ns)
Residual	8	4.18348	0.52293		
Total	16	10.17025			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	8	54.53852	6.81731	4.684	*
Animals adj Weight	7	40.01158	5.71594	3.927	*
Weight adj Animals	1	3.34284	3.34284	2.297	(ns)
Residual	8	11.64410	1.45551		
Total	16	66.18262			

Family RR, Population #2:

Duration

Source of Variation	df	SS	MS	F	
Weight & Animals	11	28.43146	2.58468	0.661	(ns)
Animals adj Weight	10	20.19345	2.01935	0.517	(ns)
Weight adj Animals	1	6.23012	6.23012	1.594	(ns)
Residual	8	31.27388	3.90923		
Total	19	59.70534			

Frequency

Source of Variation	df	SS	MS	F	
Weight & Animals	11	6.41400	0.58309	1.176	(ns)
Animals adj Weight	10	6.05416	0.06054	1.221	(ns)
Weight adj Animals	1	0.28663	0.28663	0.578	(ns)
Residual	8	3.96560	0.49570		
Total	19	10.37960			

Latency

Source of Variation	df	SS	MS	F	
Weight & Animals	11	33.15198	3.01382	1.544	(ns)
Animals adj Weight	10	32.98159	3.29816	1.689	(ns)
Weight adj Animals	1	1.21492	1.21492	0.622	(ns)
Residual	8	15.61564	1.95195		
Total	19	48.76762			

Appendix V

Two Level Nested Analysis of Variance

The structure of variation in a Model II, two level hierarchical analysis of variance is

$$y_{ijk} = \mu + \alpha_i + \beta_{ij} + \varepsilon_{ijk}$$

where y_{ijk} is the response due to the kth individual of the jth family of the ith group, μ is the common mean, α_i is the effect due to the ith group, β_{ij} is the effect due to the jth family in the ith group, and ε_{ijk} is the random error component due to individuals (Becker, 1975).

The analysis of variance table for an hierarchical anova with unequal sample size is set up as follows:

Source of Variation	df	SS	MS	F_s	Expected MS
$\bar{Y} - \bar{\bar{Y}}$ Among Groups (Populations)	a-1	SS_G	$SS_G/a-1$	*	$\sigma^2+n_0\sigma^2_{SG}+(nb)_0\sigma^2_A$
$\bar{Y}_B - \bar{Y}_A$ Among Subgroups (Families)	b-a	SS_{SG}	$SS_{SG}/b-a$		$\sigma^2+n_0\sigma^2_{SG}$
$Y - \bar{Y}_B$ Within Subgroups (Error)	(Σn_i-1) $-(b-1)$	SS_W	SS_W/df		σ^2
$Y - \bar{\bar{Y}}$ Total	$(\Sigma n_i - 1)$	ΣSS			

The coefficients of variance components are computed as follows:

$$n_0' = \frac{\sum\left(\frac{\sum n_{ij}^2}{\sum n_{ij}}\right) - \frac{\sum\sum n_{ij}^2}{\sum\sum n_{ij}}}{\text{df Groups}}$$

$$(nb)_0' = \frac{\sum\sum n_{ij} - \frac{\sum(\sum n_{ij})^2}{\sum\sum n_{ij}}}{\text{df Groups}}$$

$$n_0 = \frac{\sum\sum n_{ij} - \sum\left(\frac{\sum n_{ij}^2}{\sum n_{ij}}\right)}{\text{df Subgroups}}$$

The variance components are estimated in the following manner.

$$S^2 = \text{MS Within}$$

$$S_{SG}^2 = \frac{\text{MS Group} - \text{MS Within}}{n_0}$$

$$S_A^2 = \frac{\text{Ms Groups} - \text{MS Within} - n_0'(S_{SG}^2)}{(nb)_0}$$

An approximate test of significance for groups can be performed using a newly synthesized denominator mean square, where MS' Subgroups = $S^2 + n_0' S_{SG}^2$

$$* \quad F_s = \frac{\text{MS Groups}}{\text{MS}' \text{ Subgroups}}$$

The degrees of freedom for the reconstituted MS′ is

$$df' = \frac{(MS')^2}{\sum_i (w_i MS_i)^2/df_i}$$

where w_i are the coefficients for the mean squares,

$$\left(1 - \frac{n_0'}{n_0}\right) \text{MS Within, and } \left(\frac{n_0'}{n_0}\right) \text{MS Subgroups}$$

df_i are the original degrees of freedom of mean squares MS_i and $\sum_i$ implies summation over all mean squares MS_i.

The F test for subgroups is carried out in the standard fashion as,

$$** \quad F = \frac{\text{MS Subgroups}}{\text{MS Within}}$$

(a) Analysis of Variance and Variance Components for 'Duration'.

(i) Anova Table

Source of Variation	df	SS	MS	F_s
Between Populations	1	15.611	15.611	2.745 (ns)
Among Families	29	149.550	5.157	1.971 **
Within Families	653	1708.509	2.616	
Total	683			

$F_{.01[24,120]}$ = 1.95 (conservative critical value employed to avoid interpolation).

(ii) Variance Components

$n_0' = 26.258$ $S_W^2 = 2.616$

$n_0 = 21.719$ $S_A^2 = 0.02984$

$(nb)_0 = 336.594$ $S_{SG}^2 = 0.11699$

(b) Analysis of Variance and Variance Components for 'Latency'.

(i) Anova Table

Source of Variation	df	SS	MS	F_s
Between Populations	1	1.439	1.439	<1 (ns)
Among Families	29	199.76	6.888	2.262 **
Within Families	562	1711.476	3.045	
Total	592			

$F_{.01[24,120]} = 1.95$ (conservative critical value employed to avoid interpolation).

(ii) Variance Components

$n_0' = 23.165$ $\quad S_W^2 = 3.045$

$n_0 = 18.852$ $\quad S_{SG}^2 = 0.2039$

$(nb)_0 = 10.223$ $\quad S_A^2 = -0.6191$

(c) Analysis of Variance and Variance Components for 'Frequency'.

(i) Anova Table

Source of Variation	df	SS	MS	F_s
Between Populations	1	0.174	0.174	<1 (ns)
Among Families	29	33.111	1.142	2.252 **
Within Families	653	331.128	0.507	
Total	683			

$F_{.01[24,120]} = 1.95$ (conservative estimate employed to avoid interpolation).

(ii) Variance Components

$n_0' = 26.258$ $\quad S_W^2 = 0.507$

$n_0 = 21.719$ $\quad S_{SG}^2 = 0.0292$

$(nb)_0 = 336.594$ $\quad S_A^2 = -0.0033$