fashion for every new brain area into which kainic acid is injected that it has indeed spared all fibers of passage. Our results suggest that one type of fiber may be spared while others are not, or that a significant but subtotal percentage of all fibers may be destroyed. Furthermore, this may vary from one brain region to another. Some techniques used to test the integrity of fibers of passage may not be quantitative enough to detect small losses; such techniques include horseradish peroxidase transport through the injected area (*7*) and electrophysiological evocation of a response caused by stimulation of fibers that run through that area (*24*). Conventional histology, either at the light or the electron microscopic level, would not appear to be able to provide an adequately quantitative measure (*2, 3, 25*).

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References and Notes

1. J. W. Olney, V. Rhee, O. L. Ho, *Brain Res.* **77**, 507 (1974).
2. J. T. Coyle and R. Schwarcz, *Nature (London)* **262**, 244 (1976); E. G. McGeer and P. L. McGeer, *ibid.* **263**, 517 (1976).
3. R. Schwarcz and J. T. Coyle, *Life Sci.* **20**, 413 (1977); *Brain Res.* **127**, 235 (1977); R. Schwarcz, J. P. Bennett, J. T. Coyle, *J. Neurochem.* **28**, 867 (1977); J. T. Coyle, R. Schwarcz, J. P. Bennett, P. Compochiaro, *Prog. Neuropsychopharmacol.* **1**, 13 (1977).
4. S. T. Mason, P. R. Sanberg, H. C. Fibiger, *Life Sci.* **22**, 451 (1978); S. T. Mason and H. C. Fibiger, *Brain Res.* **155**, 313 (1978); S. T. Mason, P. R. Sanberg, H. C. Fibiger, *Science* **201**, 352 (1978).
5. R. M. Herndon and J. T. Coyle, *Science* **198**, 71 (1977).
6. E. L. Simson, R. M. Gold, L. J. Standish, P. L. Pellett, *ibid.*, p. 515.
7. I. Divac, H. J. Markowitsch, M. Pritzel, *Brain Res.* **151**, 523 (1978).
8. U. Ungerstedt, *Acta Physiol. Scand. Suppl.* **367**, 1 (1971); O. Lindvall and A. Bjorklund, *ibid.* **412**, 1 (1974).
9. Animals were anesthetized with Nembutal (50 mg/kg, injected intraperitoneally) and positioned in a stereotaxic instrument (David Kopf). Two holes were drilled in the skull. A 34-gauge cannula was lowered to the following coordinates: AP + 2.6 mm from interaural zero, ML ± 1.1 mm from the midline suture at bregma, and DV + 3.7 mm from interaural zero. Kainic acid (0.5 μg) dissolved in 1 μl of 50 m*M* phosphate buffer at *p*H 7.0 was injected at the rate of 1 μl per minute, and the cannula was left in for a further minute to permit diffusion of the drug. Injections were made unilaterally on the left-hand side of the brain. Control animals received injections of 1 μl of phosphate buffer vehicle not containing any kainic acid. 6-Hydroxydopamine hydrobromide (Regis Chemicals) was dissolved in 1 μl of 0.9 percent saline containing ascorbic acid (0.3 mg/ml) as an antioxidant. Concentrations are expressed as the amount injected (6-OHDA as the free base, kainic acid as such) in the total amount of fluid delivered. Kainic acid and 6-OHDA were made up freshly each day, kept on ice, and protected from light between injections.
10. R. A. Ross and D. J. Reis, *Brain Res.* **73**, 161 (1974).
11. D. C. S. Roberts, A. P. Zis, H. C. Fibiger, *ibid.* **95**, 441 (1975).
12. E. G. McGeer and P. L. McGeer, *Can. J. Biochem.* **40**, 1141 (1962).
13. S. H. Snyder and J. J. Coyle, *J. Pharmacol. Exp. Ther.* **165**, 78 (1969).
14. A. C. Cuello, R. Hiley, L. L. Iversen, *J. Neurochem.* **21**, 1337 (1973).
15. O. Lindvall, A. Bjorklund, A. Nobin, U. Stenevi, *J. Comp. Neurol.* **154**, 317 (1974).
16. D. C. S. Roberts, M. T. C. Price, H. C. Fibiger, *J. Comp. Physiol. Psychol.* **90**, 363 (1976); G. M. Anlezark, T. J. Crow, A. P. Greenway, *Science* **181**, 682 (1973); T. J. Crow, A. Longden, A. Smith, S. Wendlandt, *Behav. Biol.* **20**, 184 (1977); G. R. Sessions, G. J. Kant, G. F. Koob, *Physiol. Behav.* **17**, 853 (1976); J. Korf, G. Aghajanian, R. Roth, *Eur. J. Pharmacol.* **21**, 1 (1973); G. Arbuthnott, J. Christie, T. Crow, D. Eccleston, D. Walter, *Experientia* **29**, 52 (1973); T. M. Kobayashi, M. Palkovits, I. J. Kopin, D. M. Jacobowitz, *Brain Res.* **77**, 269 (1974).
17. T. Maeda and N. Shimizu, *Brain Res.* **36**, 19 (1972); L. A. Loizou, *ibid.* **15**, 563 (1969); G. F. Koob, A. F. Kelley, S. T. Mason, *Physiol. Behav.* **20**, 709 (1978).
18. N. T. Buu, E. Puil, N. M. van Gelder, *Gen. Pharmacol.* **7**, 5 (1976).
19. W. Zieglgansberger and E. A. Puil, *Exp. Brain Res.* **17**, 35 (1973); *Arch. Exp. Pathol. Pharmakol.* **277**, Suppl. R89 (1973).
20. V. K. Singh, E. G. McGeer, P. L. McGeer, *Society for Neurosciences Abstract 140* (November 1977), p. 45.
21. N. M. Friedle, P. H. Kelley, K. E. Moore, *Br. J. Pharmacol.* **63**, 151 (1978).
22. L. L. Butcher and R. C. Rogers, *Eur. J. Pharmacol.* **50**, 287 (1978); R. C. Meibach, L. Brown, F. H. Brooks, *Brain Res.* **148**, 219 (1978).
23. S. M. Wuerthele, K. L. Lovell, M. Z. Jones, K. E. Moore, *Brain Res.* **149**, 489 (1978).
24. S. Assaf and J. J. Miller, personal communication.
25. T. Hattori and E. G. McGeer, *Brain Res.* **129**, 174 (1977); J. T. Coyle, M. E. Molliver, M. J. Kuhar, *J. Comp. Neurol.*, in press.
26. Work supported by the Medical Research Council of Canada. S.T.M. is in receipt of an MRC Fellowship. We thank B. Richter for technical assistance and E. G. McGeer and L. L. Iversen for reading the manuscript. We thank J. J. Miller for the radioenzymatic noradrenaline determinations of locus coeruleus.

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Morphological Affinities of *Pan paniscus*

Abstract. *Although the pygmy chimpanzee (*Pan paniscus*) is more similar to man than is the common chimpanzee (*Pan troglodytes*) in some traits, the resemblance is due primarily to the smaller size and concomitant allometric generalization of the former. The two species of chimpanzees are equally good models for the common ancestry of African apes and man.*

The recent discovery of 3- to 4-million-year-old human fossils (*1*) quickens interest in possible living analogs to our common ancestor with apes (*2, 3*). The pygmy chimpanzee (*Pan paniscus*) has been proposed as a good prototype for this common ancestor (*2, 3*). We report our test of the appropriateness of *P. paniscus* as an ancestral prototype by assessing the morphological affinities of *P. paniscus* relative to *P. troglodytes*, *Homo sapiens*, and early hominids, and determining the effects of size and scaling on the morphology of these species.

Morphological similarity was assessed by comparing large sets of measurements (196) that describe body proportions, joint configurations, cranial shape, and dental morphology. Comparisons for each body part were made by calculating the generalized distance among *Homo sapiens*, *Pan paniscus*, *Pan troglodytes*, and the early hominid group of gracile *Australopithecus* in the multidimensional space defined by canonical variates (Table 1). Although the two chimpanzee species are more similar to one another than to either extinct or extant hominids, the affinity between the pygmy chimpanzee and hominids is significantly greater than that between the common chimpanzee and hominids in general body proportions, shoulder, elbow, forearm, premolars, and anterior tooth morphology. Gracile australopithecines are closer to *P. paniscus* than to *P. troglodytes* in proportions, shoulder, and elbow. In all other traits measured, these early hominids significantly converge on *P. paniscus* on the second canonical variate (on which *P. paniscus* is always mutually discriminated from *H. sapiens* and *P. troglodytes*).

Could allometry, the necessary change of proportions with size, explain the differential similarity of *P. paniscus*? While *Pan* species differ less in size than once thought [about 15 percent (*3a*)], the smaller size of *P. paniscus* might still explain some of its more generalized appearance. Static adult allometry often resembles the process of ontogenetic allometry, the earlier (smaller) stages of which usually appear more primitive. Furthermore, the process of neoteny does not necessarily lead to decrease in body size (*4*): differential cessation of growth could still yield a neotenized individual at a larger size. Therefore the differential similarity of pygmy chimpanzees toward humans could be allometric despite the size discrepancy, if it were the result of coincidentally shared juvenile features.

To test the effects of allometry we calculated the correlations between interspecific and intraspecific allometry coefficients for *P. paniscus* and *P. troglodytes* (Table 2). This approximates the angle between the vectors describing the allometric growth pattern within *P. troglodytes* and the vector intersecting the *P. troglodytes* and *P. paniscus* centroids (*5*). The highest correlation in Table 2 is for skeletal proportions resulting from the fact that pygmy chimpanzees differ from common chimpanzees in having shorter arms, longer legs, and

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Table 1. Distances between *Homo sapiens*, *Pan troglodytes*, *P. paniscus*, and *Australopithecus* for various morphometric analyses (*10*).

Study	Number of measurements	*Homo*-*P. trog.*	*Homo*-*P. panisc.*	*P. trog.*-*P. panisc.*	*P. trog.*-*Australop.*	*P. panisc.*-*Australop.*
Proportions (*11–13*)	13	19.92	18.14 (*14*)	3.06		
Shoulder (*15*)	9	3.37	2.78 (*14*)	1.26	1.63	1.26 (*14*)
Elbow (*6*)	16	5.29	4.72 (*14*)	3.57	4.73	3.48 (*14*)
Wrist (*16*)	20	9.57	9.89	4.28		
Capitate (*17*)	12	10.69	10.91	2.78	8.32	7.77 (*18*)
Forearm (*13*)	14	8.29	7.81 (*14*)	4.12		
Os coxae (*19*)	17	17.77	18.61	4.69	14.65	14.31 (*18*)
Hip (*20*)	10	4.86	5.12	1.87	3.69	3.15 (*18*)
Knee (*20*)	7	6.84	7.02	1.46	4.27	3.52 (*18*)
Cranium (*21, 22*)	9	1.74	2.59	1.48		
Lower dentition (*22, 23*)	16	1.05	1.05 (*18*)	1.04		
Upper dentition (*22, 23*)	16	1.12	0.94 (*18*)	1.01		
Molars (*24*)	24	23.31	22.97 (*18*)	21.46		
Premolars (*25*)	7	24.47	14.80 (*14*)	10.33		
Anterior teeth (*26*)	6	6.24	4.32 (*14*)	3.53		

*Excludes specimen classified in the robust species of *Australopithecus*.

smaller joint surfaces; this precisely characterizes the growth difference from small to large specimens of *P. troglodytes*. The correspondence of size to shape changes within *P. troglodytes* with interspecific differences in shape show that *P. paniscus* is merely a scaled, reduced version of *P. troglodytes* in these measurements (or, the equally valid thesis, that *P. troglodytes* is a scaled-up *P. paniscus*). The coefficients vary considerably for the other morphometric studies (Table 2). In most of those body parts in which *P. paniscus* converges significantly on *H. sapiens* (shoulder, forearm, premolars, anterior teeth) the coefficients are high, which indicates that the differential resemblances between *P. paniscus* and *H. sapiens* can be explained by allometric effects. The only postcranial convergence not explained by this analysis of allometric effects is the distal humerus, which in a previous study (*6*) by a different method showed no differential similarity for *P. paniscus*.

Neoteny has reappeared as an important, although controversial, theory for the origin of human peculiarities (*4, 7*). This theory is difficult to test (*8*). Possible support comes from the correspondence between these findings: *P. paniscus* is distinguished from *P. troglodytes* by the similarity of many of its features to the human condition; many of these features can be directly ascribed to juvenile retention associated with smaller size or arrested growth. In contrast, we reason that the australopithecine predecessors to humans were less juvenilized than humans, by this theory. They therefore should show less differential similarity to *P. paniscus*; instead they show considerably more. Perhaps this indicates the greater likelihood of similarities between *Australopithecus* and *P. paniscus* being due to shared primitive characters (symplesiomorphy) independent of size factors.

Our results show that a considerable proportion of the difference between *Pan* species can be attributed to allometry. The remaining differences are probably not all distributed as the primitive state in *P. paniscus*, either: for instance, in the molar dentition, the system in which *Pan* species differ most, *P. paniscus* is most probably derived. The enlarged crown components, increased talonid height, and square shape of pygmy chimpanzee molars do not resemble earlier phylogenetic stages or earlier growth stages as they are known from the human ontogenetic sequence (*9*). Thus, by subtracting the allometric effect and critically examining remaining differences, we can see that the common ancestor of pygmy and common chimps could have resembled both about equally. Modeling of a "missing link" based on *P. paniscus* is probably premature. Nevertheless, the similarity of pygmy chimpanzees and australopithecines does indicate that samples of the former may be crucially important in comparative studies of the earlier hominid fossil record.

Table 2. Correlations between interspecific and intraspecific allometry coefficients for *P. paniscus* and *P. troglodytes*.

Study	*r*
Proportions	.94
Shoulder	.67
Elbow	−.06
Wrist	−.22
Capitate	.01
Forearm	.58
Pelvis	.30
Hip	.10
Knee	−.04
Cranium*	.89
Lower teeth*	.58
Upper teeth†	.75
Molars	−.37
Premolars	.91
Anterior teeth	.39

*Juvenile-adult vector within *P. troglodytes* compared with *P. troglodytes*-*P. paniscus* vector. †Male-female vector within *P. troglodytes* compared with male *P. troglodytes*-male *P. paniscus* and female *P. troglodytes*-female *P. paniscus* vectors.

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References and Notes

1. D. C. Johanson, T. White, Y. Coppens, *Kirtlandia* **28**, 1 (1978); D. C. Johanson and T. D. White, *Science* **203**, 321 (1979).
2. A. Zihlman, *Am. J. Phys. Anthropol.* **44**, 216 (1976); ——, J. E. Cronin, D. L. Cramer, V. M. Sarich, *Nature (London)* **275**, 744 (1978).
3. A. L. Zihlman and D. L. Cramer, *Folia Primatol.* **29**, 68 (1979).
3a. Zoo weights from A. D. Horn [*Am. M. Phys. Anthropol.*, in press] and the museum catalog at the Musée Royal de l'Afrique Centrale, Tervuren, result in weights of eight male *P. paniscus* of 44, 44, 38, 38, 37, 39, 30, and 36 kg, and of five females of 35, 35, 27, 33, and 31 kg. This compares with mean weights for *P. troglodytes* of 48.9 kg (male) and 40.6 kg (female) according to J. R. Napier and P. Napier [*A Handbook of Living Primates* (Academic Press, London, 1967)] and 39.5 kg (male) and 30 kg (female) for Gombe chimpanzees given in Zihlman and Cramer (*3*). Weights of Gombe females are, therefore, actually less than those from the *P. paniscus* sample.
4. S. J. Gould, *Ontogeny and Phylogeny* (Belknap, Cambridge, 1977).
5. Only the considerably larger *P. troglodytes* samples are used for computation of within-species allometry. The within-species allometry vector is computed as the eigenvectors of the logarithmic covariance matrix following P. Jolicoeur [*Biometrics* **19**, 497 (1963)]. The between-species vector is computed as

$$k_i = \frac{(\log X_{i1} - \log X_{i2})\, p}{\left[\sum_{j=1}^{p} (\log X_{j1} - \log X_{j2})^2\right]^{1/2}}$$

where X is the array of means and p the number of measurements.
6. H. M. McHenry and R. S. Corruccini, *Folia Primatol.* **23**, 227 (1975).
7. C. L. Brace, *Am. Anthropol.* **80**, 982 (1978); S. Luvtrup, *Syst. Zool.* **27**, 125 (1978); R. H. Tuttle, *Am. J. Phys. Anthropol.* **49**, 287 (1978); D. Wake, *Paleobiology* **4**, 96 (1978).
8. M. Cartmill, *Science* **199**, 1194 (1978).
9. P. M. Butler, *Biol. Rev.* **31**, 30 (1956); B. S. Kraus and R. E. Jordon, *The Human Dentition Before Birth* (Lea & Febiger, Philadelphia, 1965).
10. Sample sizes ranged from 57 to 20 for *H. sapiens*, 49 to 20 for *P. troglodytes*, and 23 to 10 for *P. paniscus*.
11. Measurements include humeral head width (*12*), humeral distal articular surface width (*6*), humeral length (*12*), radial head diameter (*12*), radial length (*12*), femoral head diameter (*12*), femoral transverse shaft diameter (*12*), femoral bicondylar width (*12*), femoral length (*12*), tibial length (*12*), tibial transverse condylar width (*12*), ulnar width (*13*), and ulnar shaft transverse diameter (*13*).
12. H. M. McHenry, *Am. J. Phys. Anthropol.* **40**, 329 (1974).
13. ——, R. S. Corruccini, F. C. Howell, *ibid.* **44**, 295 (1976).
14. Significantly near *H. sapiens* on the first canonical variate at $P = .01$.

15. R. S. Corruccini and R. L. Ciochon. *Am. J. Phys. Anthropol.* **45**, 19 (1976).
16. R. S. Corruccini, *J. Hum. Evol.* 7, 307 (1978).
17. H. M. McHenry, in preparation.
18. Significantly nearer *H. sapiens* on the second canonical variate at $P = .01$.
19. H. M. McHenry, *Am. J. Phys. Anthropol.* **43**, 245 (1975); ——— and R. S. Corruccini, *ibid.*, p. 263.
20. H. M. McHenry, *Nature (London)* **259**, 657 (1976); *Folia Primatol.* **26**, 139 (1976); *Am. J. Phys. Anthropol.* **49**, 473 (1978).
21. D. L. Cramer, *Contrib. Primatol.* **10**, 1 (1977); W. W. Howells, *Peabody Mus. Pap. Anthropol.* **67**, 1 (1973).
22. Expressed in units of *d* (simple Euclidean distance) rather than *D* (generalized distance) since sample means only are available for these following P. H. A. Sneath and R. R. Sokal [*Numerical Taxonomy* (Freeman, San Francisco, 1971)].
23. Crown diameters from E. H. Ashton and S. Zuckerman, *Philos. Trans. R. Soc. London Ser. B* **234**, 471 (1950); A. M. Henderson, thesis, University of Colorado, Boulder (1975); D. C. Johanson, *Am. J. Phys. Anthropol.* **41**, 39 (1974); D. R. Pilbeam, *Peabody Mus. Nat. Hist. Yale Univ. Bull.* **31**, 1 (1969); A. Remane, *Bibl. Primatol.* **1**, 229 (1962); E. L. Schuman and C. L. Brace, *Hum. Biol.* **26**, 239 (1954); M. H. Wolpoff, *Case West. Reserve Univ. Stud. Anthropol.* **2**, 1 (1971).
24. R. S. Corruccini, *Z. Morphol. Anthropol.* **68**, 14 (1977).
25. ———, *J. Dent. Res.* **56**, 1093 (1977).
26. Cervical lengths and breadths of incisors and canines.
27. We thank M. Poll, D. F. E. T. van den Audenaerde, G. Vandebroek, L. R. Barton, T. Molleson, B. Lawrence, C. Mack, and W. W. Howells for the use of comparative material in their charge; R. E. F. Leakey, C. K. Brain, E. Vrba, M. Day, and D. C. Johanson for use of fossil material in their charge; and L. J. McHenry for assistance. Supported in part by the Wenner Gren Foundation for Anthropological Research and the Committee on Research, University of California, Davis.

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ACTH and Vasopressin Treatments Immediately After a Defeat Increase Future Submissiveness in Male Mice

Abstract. *Male mice were given a single injection of either adrenocorticotropic hormone (ACTH) or lysine vasopressin immediately after a defeat in an encounter with an aggressive male mouse. The defeated mice were tested for submissiveness at either 24 hours, 48 hours, or 7 days after the initial encounter. Both hormone treatments increased future submissiveness, although the time courses of the effects were different: The effects of ACTH disappeared after 48 hours, whereas those of vasopressin persisted for 7 days. These results suggest that changes in peptide hormone levels following naturally stressful experiences can affect the memory of those experiences, as expressed in future adaptive responses.*

Recent evidence has suggested that the hormonal responses to initial experiences may be important in the mediation of the effects of those experiences on future behavioral reactions, perhaps through effects on memory processes (*1*-*4*). The majority of this evidence comes from studies of the effects of the pituitary peptides adrenocorticotropic hormone (ACTH) and vasopressin on avoidance responding in rats and mice, where posttraining treatments with these hormones lead to enhanced retention of the avoidance response (*1*, *3*, *5*, *6*). We report here the results of two experiments showing that these pituitary peptides may play a similar role in social situations as well; increasing ACTH and vasopressin levels immediately after an initial defeat increases an animal's submissiveness in future encounters.

The experiments reported here examine the interaction between prior experiences of defeat and experimentally altered hormone levels. We conducted two initial studies: the first to show that prior experiences of defeat do increase future submissiveness, and the second to show that the hormone treatment schedule used here does not affect submissiveness independently of the prior experience of defeat (*7*). In this way, we could be certain that the design of the two experiments described here does permit evaluation of the interaction of prior experiences with modified hormone levels.

In the first experiment on the interaction of prior defeat and hormone treatment, we examined the effects of postdefeat treatments with ACTH on future submissiveness. Fifty-nine male CD-1 mice, 7 weeks old and kept isolated from other mice for 1 week, experienced an initial defeat in an encounter with an aggressive opponent. The opponents were untreated, adult male CD-1 mice that had been housed in isolation for 6 to 10 weeks to enhance aggressiveness (*8*). The encounter was in a wooden arena 30.3 by 33.0 by 15.0 cm. The subject and opponent were placed together in the arena and allowed to interact until the test animal submitted. An animal was considered to have submitted when it (i) displayed the characteristic upright submissive posture, and (ii) failed to fight back when subsequently attacked by the opponent (*9*).

The mice then were randomly assigned to one of three control groups or one of three experimental groups (8 to 11 per group). Immediately after the initial defeat, all mice received a single injection. The mice in the control groups received a subcutaneous injection of 0.05 ml of saline (placebo), whereas those in the experimental groups received a subcutaneous injection of 2 I.U. of ACTH (Cortrophin-Zn, Organon) in 0.05 ml of saline. This dosage of ACTH was selected because it is effective in modifying avoidance responding in mice (*10*).

The mice in one experimental and one control group were tested for submissiveness with a novel opponent at one of three times: 24, 48, or 168 hours after the initial defeat and hormone or placebo treatment. The test of submissiveness was conducted as was the encounter for the initial defeat: The test an-

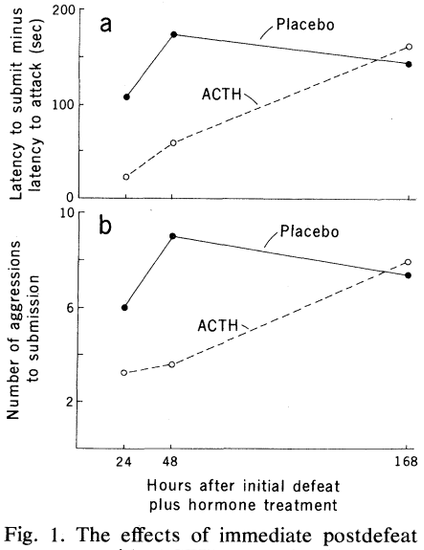

Fig. 1. The effects of immediate postdefeat treatment with ACTH or a placebo on (a) mean latency to submit minus latency to attack at each postdefeat interval, and (b) mean number of aggressions to submission at each postdefeat interval.

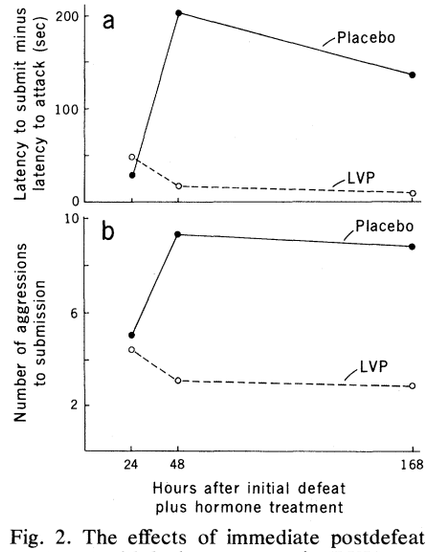

Fig. 2. The effects of immediate postdefeat treatment with lysine vasopressin (*LVP*) or a placebo on (a) mean latency to submit minus latency to attack at each postdefeat interval, and (b) mean number of aggressions to submission at each postdefeat interval.

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