interferometry (7) that showed that the trailingside point source contributed 30 to 40 percent of the total flux.

- 20. Performing an F test on the reduction of χ^2 obtained by the addition of the third hot spot and its additional three parameters shows that it is unlikely (P < 0.01) that a similar reduction of χ^2 would result from a fortuitous distribution of data.
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- 24. Our model assumes that the emitting surface is flat and smooth on the wavelength scale. This is justifiable if we are seeing the surface of a quiescent, molten lava lake but is less so if we are seeing cooling flows with textures resem-

bling Hawaiian aa. In general, a rough surface will exhibit less polarization than a similar smooth one because the average viewing angle is reduced for a rough surface by the preference for the observer to see slopes whose normal is in the direction of the line of sight and for the disappearance of slopes slanted in the opposite direction.

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- 26. The authors are visiting astronomers at the Infrared Telescope Facility (IRTF), operated by the University of Hawaii under contract from the National Aeronautics and Space Administration. Supported in part by NASA grant NGL 12-001-057 and NSF grant AST83-11105. We thank R. R. Howell for providing data in advance of publication; the IRTF staff, particularly R. Capps, R. Koehler, and C. Kaminski, for assistance; and R. R. Howell and H. M. Dyck for helpful discussions.

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Nodal Modulation of the Lunar Semidiurnal Tide in the Bay of Fundy and Gulf of Maine

Abstract. Observations, numerical modeling, and theoretical calculations show how the 18.6-year modulation of the main lunar semidiurnal tide in the Bay of Fundy and Gulf of Maine is reduced from its astronomical value of 3.7 percent to 2.4 percent by the effects of friction and resonance. The agreement of the three approaches increases confidence in model predictions of widespread changes in the tidal regime resulting from development of tidal power.

L.-F. Ku

Fisheries and Oceans Canada, Canadian Hydrographic Service, Ottawa, Ontario K1A 0E6 **D. A. Greenberg** Fisheries and Oceans Canada, Coastal Oceanography, Bedford Institute of Oceanography, Dartmouth, Nova Scotia B2Y 4A2 C. J. R. Garrett Department of Oceanography, Dalhousie University, Halifax, Nova Scotia B3H 4J1 F. W. Dobson Fisheries and Oceans Canada, Ocean Circulation, Bedford Institute of Oceanography

The world's highest tides occur in the Bay of Fundy and have been attributed (1) to a near-resonant response of the Bay of Fundy–Gulf of Maine system. Proposed development of tidal power would increase the tides throughout this region by bringing the system closer to resonance. Detailed predictions of this increase have been made by a numerical model (2) that has been calibrated against observed tides. We report that study of the long-period variations in the tide support the idea of resonance and the validity of the numerical model.

The astronomical forcing of the main lunar semidiurnal tide $(M_2 \text{ tide})$, which

has a period of 12.42 hours, varies by ± 3.73 percent during 18.61 years because of variations in the declination of the moon's orbit (3). It is generally assumed in tidal prediction that the observed M₂ tide will be modulated by the same amount. In the few instances in which long records of sea level have been analyzed to check this assumption (4, 5), they show noticeable differences from the astronomical values. Using more recent data, we examined the departure of the modulation of M_2 from the value predicted in the equilibrium tide and we compared our results with those from a numerical model and from theoretical calculations.

An unmodulated tidal constituent would take the form of $A \cos(\omega t - \theta)$, where A is amplitude, ω is the tidal frequency, t is time, and θ is a phase lag. The theoretically modulated tide may be written $A(1 + R\cos\Delta t)\cos(\omega t - \theta + u)$ where $\Delta = -2\pi(18.61 \text{ years})^{-1}$, u = $-R\sin\Delta t$. As mentioned, for the M₂ tide, R = 0.0373 and so | u | is 2.14°. Because the observed tide may be different from this, we consider it in the form A[1 + R'] $\cos(\Delta t - \delta') \cos(\omega t - \theta + u'),$ where $u' = -a\sin\Delta t + b\cos\Delta t$, with R', δ' , a, and b to be determined from the data. When the observed modulation equals the theoretical astronomical modulation, $R' = R, \delta' = 0, a = R, and b = 0.$

Data on sea level from Saint John (Fig. 1) for the years 1947 through 1971 were Fourier analyzed at the M_2 frequency for each year and the amplitude and phase fitted with our model. The results may be compared (Table 1) with those of a similar analysis by Doodson (4) for 1894 through 1916. Data from Boston and Bar Harbor for 1947 through 1966 were analyzed by complex demodulation. To ensure that we were looking at the response of the Fundy-Maine system and not the North Atlantic, data from Halifax for 1920 to 1980 were also analyzed.

The numerical model of Greenberg (2) was used with the imposed M_2 tide at the shelf edge increased by 3.73 percent (R = 0.0373) to see how well the model response corresponded to observation. To account for open boundaries (6), a trial was also made with boundary amplitudes scaled up by 4.0 percent.

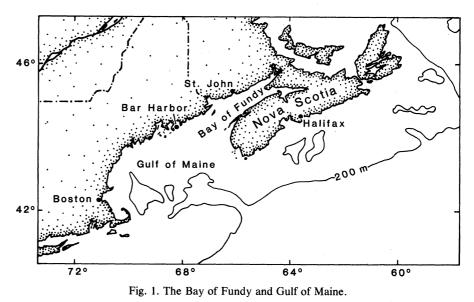


Table 1. Results from tidal analyses and model trials. For a discussion of error limits see (7).

Data	R' (%)			δ'	a (den)	b (deg)		
	Anal- ysis	Model 3.73	Model 4.0	(deg) anal- ysis	(deg) anal- ysis	Anal- ysis	Model 3.73	Model 4.0
Astronomical	3.73			0	2.14	0		
Halifax (1920–1980)	3.64			0.4	2.08	0.01		
Saint John (1947–1971)	2.30	2.15	2.30	-12.0	2.22	-0.18	-0.13	-0.14
Saint John (Doodson) (1894–1916)	2.40			28.6	2.71	-0.19		
Bar Harbor (1947–1966)	2.35	2.39	2.56	3.6	2.39	-0.11	0.02	0.02
Boston (1947–1966)	2.61	2.55	2.74	6.8	2.52	-0.34	0.04	0.04
Transport		2.36	2.53				0.03	0.03

The model results agree well with the observations (Table 1). The variation in the response, R', at different ports, appears to be real, with the lower values near the upper Bay of Fundy, where tidal friction is most important. We can think of no reason why the modulation of the response should not be in phase with the modulation of the forcing, and thus we consider δ' to be a measure of the confidence in the analysis (7). Similarly, a should be equal to the astronomical value; any difference is a measure of the error in a and b. The fact that b is small from data and models, less than 0.4° everywhere $(1^{\circ} \approx 2 \text{ minutes})$, indicates that the phase is not greatly modified from the nodal value. The analysis of the Halifax data indicates that the North Atlantic responds directly to the nodal modulation without any other complicating effects.

The reduced nodal modulation in the Bay of Fundy and Gulf of Maine can be interpreted in terms of the proximity of the system to resonance, at M_2 , and the effect of quadratic bottom friction. A simple model (1) for the response at location x to forcing at frequency ω is $Re[\zeta(\mathbf{x},\omega)\exp(i\omega t)]$ with

$$\zeta(\mathbf{x},\omega) = F(\omega)S(\mathbf{x})[1 - \omega/\omega_0 + i(2Q)^{-1}]^{-1}$$
(1)

where $F(\omega)$ represents the forcing amplitude, $S(\mathbf{x})$ the spatial response (a "normal mode" in which friction as well as other dynamics produce spatial variations of amplitude and phase), ω_0 the resonant frequency, and Q the "quality factor" that is inversely proportional to the damping effect of bottom friction.

For linear bottom friction (that is, friction proportional to the speed of the current), Q is a constant, independent of the amplitude ζ of the response, which thus increases by the same amount as the forcing F. However, for the more generally accepted quadratic friction (propor-

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tional to the square of the current), $Q \propto \zeta^{-1}$ and so the fractional increase in $|\zeta|$ is less than that in |F|. The extent to which the modulation is reduced depends on the parameter $r = 2Q_0 + 1 - 1$ ω/ω_0 | which relates the two terms in the denominator of Eq. 1; Q_0 is the Q of the average tide. If |F| and $|\zeta|$ increase by factors of 1 + n and 1 + m, respectively, then

$$1 + m = (1 + n) | r + i |$$

[| r + i(1 + m) |]⁻¹ (2)

and for small m and n

$$\frac{m}{n} = \left(\frac{1+r^2}{2+r^2}\right) \text{ or } r = \left(\frac{2m/n-1}{1-m/n}\right)^{\frac{1}{2}}$$
(3)

as plotted in Fig. 2. We note that for a system exactly at resonance r = 0 and hence m/n = 0.5.

In applying this formula we use n = 0.0373 (the astronomical, rather than the shelf edge, modulation is appropriate). The data and model results (Table 1) show that *m* varies a little spatially, so that our model in Eq. 1 is an oversimplification, but m is certainly less

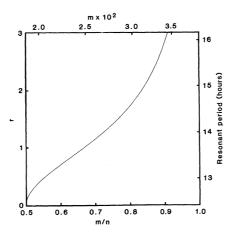


Fig. 2. The parameter $r = 2Q_0 + 1 - \omega/\omega_0 + as$ a function of the ratio m/n of the response modulation to forcing modulation. The value of m plotted assumes n = 0.0373 and the resonant period assumes $O_0 = 5$.

than n. We could use the average, 0.0242, for the three ports analyzed, but the most appropriate value is that for the modulation of the mass transport across the shelf edge. For models 3.73 and 4.0, this is 0.0236 or 0.0253 (Table 1), leading to r = 0.85 or 1.05, so that (8) for $Q_0 \approx 5$, the resonant period $2\pi/\omega_0$ is 13.5 or 13.7 hours. A comparison of models and data suggests that an average of the two models is appropriate, leading to 13.6 hours for the resonant period (9).

A general theory of tides in gulfs (10)suggests that the resonant period is decreased by a fraction of about $Q^{-2}/8$ from its frictionless value and ω_0 will change if Q changes. Allowing for this reduces the resonant period for M₂ from 13.6 to 13.5 hours and suggests that weaker tidal constituents [which have 50 percent more damping than M₂ for quadratic bottom friction (1)] should have a resonant period of 13.4 hours. This is close to the 13.3 hours estimated in a study (1) of the fortnightly and monthly changes in the tides that also allowed for quadratic bottom friction and the frequency-dependent response implied by Eq. 1.

Alternatively, if the resonant period of M_2 were 13.3 hours, Fig. 2 shows a predicted value of 2.2 percent for m, slightly less than, but still close to, the observed values.

In conclusion, we have shown that (i) the nodal modulation of the M₂ tide in the Bay of Fundy and Gulf of Maine is spatially variable but approximately ± 2.4 percent rather than the astronomical value of ± 3.7 percent; (ii) this is in agreement with the predictions of a numerical model; and (iii) the effect can be attributed to the quadratic nature of bottom friction by a simple model for nearresonant response. The results of this simple model are in reasonable agreement with another estimate and confirm that the tides in this region are roughly equally limited by friction and the frequency difference between M_2 and the resonant frequency. The internal consistency of these results confirms the accuracy of the models in their description of the present regime and hence increases confidence in their use for predictions (2) of changes due to tidal power development (11).

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 $V_0 - Z_0 I$, where V_0 is the value that would occur in the absence of the Bay of Fundy and Gulf of Maine, I is the tidal volume flux across the shelf edge, and Z_0 is the ocean impedance. If nodal modulation increases V_0 by a factor of 1 + n, with n = 0.0373, and I by 1 + m, then $V_0 - Z_0 J$ increases by a factor [1 + n + C(n - m)] with $C = Z_0 J(V_0 - Z_0 J)^{-1}$. C. Garrett and D. A. Greenberg [J. Phys. Oceanogr. 7, 171 (1977)] estimated C to have a magnitude of 2 with careful many three three theorem. 0.2 with a small phase that we ignore here. Hence, with $m \approx 0.025$, the shelf-edge tide in-0.2 with a small phase that we ignore here. Hence, with $m \approx 0.025$, the shelf-edge tide in-creases by 1.0398, which we round off to 1.040. The model 3.73 and model 4.0 predictions in Table 1 more or less straddle the results of data analysis, suggesting that C may even be a little less than 0.2. This would reduce Garrett and Greenberg's small corrections to Greenberg's (2) estimates, with $Z_0 = 0$, of the impact of tidal

The root-mean-square lag at Saint John, Bar Harbor, and Boston from our analysis is about 8° or 0.14 radians. This is small, but suggests that the value of R' may be wrong by about 14 percent, due to random and systematic errors. Thus R' is uncertain to ± 0.3 , although agreement between the data and the models actually seems better than this. A better estimate of the seems better than this. A octor counter in the power error limits could be obtained from the power encounter density of the M_2 amplitude in the spectral density of the M_2 amplitude in the neighborhood of the 18.6-year spectral line, but this cannot be estimated without longer records

- than are available. This value, $Q_0 \approx 5$, is probably accurate to about 10 percent (1, 2). A period of less than the 12.42-hour period of M₂
- could also be a solution; we adopt a longer period to be consistent with the results of Garrett (1) and Greenberg (2).
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Induction of AIDS-Like Disease in Macaque Monkeys with **T-Cell Tropic Retrovirus STLV-III**

Abstract. The T-cell tropic retrovirus of macaque monkeys STLV-III has morphologic, growth, and antigenic properties indicating that it is related to HTLV-III/LAV, the etiologic agent of the acquired immune deficiency syndrome (AIDS) in humans. Four of six rhesus monkeys died within 160 days of STLV-III inoculation with a wasting syndrome, opportunistic infections, a primary retroviral encephalitis, and immunologic abnormalities including a decrease in T4+ peripheral blood lymphocytes. These data show that an immunodeficiency syndrome can be produced experimentally in a nonhuman primate by an agent from the HTLV-III/LAV group of retroviruses. The STLV-III-macaque system will thus provide a useful model for the study of antiviral agents and vaccine development for human AIDS.

N. L. LETVIN, M. D. DANIEL P. K. SEHGAL, R. C. DESROSIERS R. D. HUNT, L. M. WALDRON J. J. MACKEY, D. K. SCHMIDT L. V. CHALIFOUX, N. W. KING New England Regional Primate Research Center. Harvard Medical School, Southborough, Massachusetts 01772

A T-cell tropic retrovirus with striking similarities to HTLV-III/LAV, the etiological agent of the acquired immune deficiency syndrome (AIDS) in humans, was recently isolated from macaques (Macaca mulatta) (1). We have called this virus simian T-lymphotropic virus type III (STLV-III) of macaques. Like HTLV-III/LAV, STLV-III can be isolated from T lymphocytes and grows preferentially in T4⁺ rather than T8⁺ lymphocytes. By electron microscopy, it has a cylindrical nucleoid and buds in a fashion typical of type C retroviruses (1). Furthermore, radioimmunoprecipitation of proteins from STLV-III-infected cells, with the use of a monoclonal antibody as well as sera from humans seropositive for HTLV-III/LAV, has revealed virus-specific proteins of 160, 120, 55, and 24 kilodaltons, all similar in

size to the major gag and env proteins encoded by HTLV-III/LAV (2).

All the isolates of STLV-III to date have been obtained from animals with evidence of an immune deficiency syndrome or lymphoma. Macaques with this syndrome develop profound T-lymphocyte dysfunction and eventually die of lymphomas or opportunistic infections by a spectrum of agents similar to those seen in humans with AIDS (3). It is therefore important to determine the pathogenicity of this macaque retrovirus and to explore the possibility of developing an animal model for human AIDS. Such a model would facilitate both the testing of new treatment modalities for this disease and the development of a vaccine to protect against this syndrome. We now report the results of experimental infection of macaques with STLV-III.

The STLV-III used in these studies was isolated from a rhesus monkey designated Mm251-79, which had been inoculated with tissue from another rhesus monkey with a naturally occurring retroorbital lymphoma (4). Mm251-79 died 26 months after inoculation and at necropsy was found to have a poorly differentiated lymphocytic lymphoma. Viably frozen splenocytes from this animal were cocultivated with phytohemagglutinin (PHA)stimulated human peripheral blood lymphocytes (PBL) that were maintained in cultures to which crude supernatants containing interleukin-2 (IL-2) were added. The growth of STLV-III was detected by the measurement of reverse transcriptase (RT) in the culture supernatant and was confirmed by electron microscopy (1). The virus stock was expanded by seeding the day-14 supernatant from this culture onto normal human T cells growing in the presence of IL-2. Inoculation of this supernatant onto Raji cells and canine thymus cells yielded no virus growth as determined by RT assay of culture supernatants, indicating that no simian type D retrovirus was present. Inoculation of this material onto Vero cells and human embryonic lung cells yielded no virus growth as determined by the appearance of cytopathic effect. Thus there was no evidence that other viral agents were present in the inoculum.

Six monkeys were selected for inoculation with this agent. Four of them were 6 months old and had not been used in previous studies; the other two were 18 months old and had been inoculated 15 months previously with the macaque retrovirus D/New England. While it has been suggested that type D retroviruses may play some part in immune deficiency syndromes in macaques (5), the two previously inoculated animals used in the present study appeared to be free of disease when the study began (6). They did, however, have a few enlarged axillary and inguinal lymph nodes. Except for lymphadenopathy in the D/New England infected animals, the six monkeys were found to be normal when they underwent physical examinations at the beginning of the study. Cocultivation of PBL from the four 6-month-old animals with Raji cells and canine thymus cells yielded no type D retrovirus, and cocultivation of PBL from all six animals with HUT-78 cells yielded no type C retroviruses. That the animals were normal was also indicated by the results of routine hematologic and serum chemistry studies, by their PBL absolute T4 and T8 numbers (Table 1), and by the blastogenic responses of their PBL to concanavalin A (Con A), pokeweed mitogen (PWM), xenogeneic stimulating cells, and *Candida* antigen.

The virus used in the inoculations was grown on human T cells maintained in culture with IL-2. Each animal received an intravenous inoculation of 1.7 ml of pooled supernatants from day 14 and 18 cultures of the second passage of this STLV-III isolate. The RT activity of the