codlelure showed a peak in the GC-MS plot of m/e–182 amplitude as a function of spectrum number at the same retention time as authentic codlelure.

Although mass spectral data do not establish positions of double bonds, the agreement between the retention times of codlelure and the natural pheromone on the Carbowax column (*7*) as well as the agreement in elution volumes on the silica gel–silver nitrate column provide strong confirmatory evidence for the assigned positions of the double bonds and the configurations, which were fixed by the data of Roelofs *et al.* (*1*).

The presence of codlelure in the moth extract raises the question of why it has not been isolated from the moth. We ascribe this difficulty to the exceptional instability of the pure compound when exposed to air and light during isolation. For example, virtually no codlelure was detectable as such when we allowed a thin film of it in a test tube to stand for a few days at room temperature exposed to air and light.

Sex attractants have been found by empirical screening of chemicals (*8*) and by the antennogram technique of Roelofs *et al.* The technique of computerized searching of GC-MS data can supplement these procedures by verifying or rejecting the presence of specific chemicals in the extract of the natural pheromone. The procedures are compatible because they can be used with minute amounts of chemical, rigorous purification of insect extracts is not necessary, and the number of insects needed is few compared with the number needed to elucidate chemical structure by conventional methods of isolation and identification.

The codling moth is a major and worldwide pest of apples. With the presence of the compound in the insect established, we now have a firm basis for proceeding with the use of codlelure in control efforts against this highly injurious insect.

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3. A total of 500 abdominal tips from 3-day-old female codling moths was ground in a mortar with acetone and then hexane, and the slurry was filtered. The filtrate was extracted with water (to remove the acetone) and with saturated aqueous NaCl, and then dried (over Na_2SO_4). The concentrated extract was then chromatographed on a 25-g column of silica gel (J. T. Baker No. 3404) used as received and eluted successively with 100-ml portions of 0, 2, and 5 percent ether in hexane, two 100-ml portions of 15 percent ether in hexane, one 100-ml and six 50-ml portions of 30 percent ether in hexane, and 100 ml of ether.
4. We used a Finnigan Corp. (Palo Alto, Calif.) 1015 quadrupole mass spectrometer interfaced with a glass Gohlke separator to a gas chromatograph equipped with a glass column 1.5 m by 2 mm (inside diameter) containing 3 percent OV-1 on Varaport at 135°C and operated with a helium flow rate of 15 ml/min. With these conditions, the retention time of codlelure was 9.8 minutes. A Systems Industries (Palo Alto, Calif.) 150 computer system was used to control the scan of the mass spectrometer, record the mass spectra on magnetic tape, and retrieve the data.
5. Background interference is removed by this procedure.
6. Supplied by T. P. McGovern.
7. The column was 3.6 m by 6 mm (outside diameter); the temperature was 165°C, and the helium flow rate was 70 ml/min. The retention time of codlelure was 11.4 minutes.
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Creutzfeldt-Jakob Disease: Focus among Libyan Jews in Israel

Abstract. *A countrywide search for Creutzfeldt-Jakob disease in Israel disclosed 29 cases with onset between 1963 and 1972. Incidence in various ethnic groups varied in the narrow range of 0.4 to 1.9 per million population except among Jewish immigrants from Libya, among whom the incidence was 31.3 per million. An extraordinary excess of Creutzfeldt-Jakob disease exists in this ethnic group.*

Creutzfeldt-Jakob disease (CJD) is a rapidly progressive, fatal "degenerative" disease of the central nervous system which is now classified, along with kuru, among the unconventional slow virus diseases of man (*1*). Passage of the disease from affected individuals to certain primates has been accomplished (*2*), but no conventional viral agent has been isolated. A genetic cause or at least a familial predisposition has been recognized in some instances (*3*), but even familial cases are transmissible (*4*). With both an unconventional virus and a genetic (familial) predisposition in mind as possible etiologic factors, we report a focus of CJD among Libyan Jewish immigrants to Israel.

Israel has a heterogeneous population composed of immigrants from many different countries, and analysis of the ethnic distribution of different diseases can easily be performed. All ethnic groups have equal access to Israel's excellent medical facilities. Behar *et al.* (*5*) reported a series of six cases of CJD in Israel, two of whom were Libyan immigrants. Goldhammer *et al.* (*6*) reported their experience with CJD in Israel, and five of their twelve cases were Jewish immigrants from Libya. Cases of CJD have been reported in widely scattered regions of the world (*4*), but no ethnic focus has previously been recognized.

We thought that the observations by Braham's group (*6*) and by Behar *et al.* (*5*) might indicate an unusual predilection to CJD among Libyans in

Table 1. Creutzfeldt-Jakob disease among ethnic groups in Israel. The numbers in parentheses exclude possible CJD cases.

Country of birth	Patients (No.)	Population at risk	Average annual incidence per million population (1963 to 1972), age-adjusted to population of	
			Israel, 1968	U.S., 1970*
Libya	13 (13)	30,792	31.3 (31.3)	33.0
Iraq	3 (2)	117,587	1.9 (1.3)	2.5
West and Central Europe	4 (3)	199,300	1.0 (0.6)	1.1
Israel	2 (1)	1,020,411	1.0 (0.5)	1.0
Morocco, Algeria, Tunisia	2 (1)	275,432	0.8 (0.3)	0.9
East Europe	5 (3)	509,317	0.4 (0.2)	0.5
Total	29 (23)			

* Based on total CJD cases.

Israel and decided to determine the frequency of this disease in various ethnic groups. A countrywide search for CJD was instituted. In this extensive search, stress was laid on clinical features only, and no special effort was directed toward finding cases among Libyans. We reviewed hospital and autopsy records for the period 1963 to 1972 for cases diagnosed as CJD. In addition, all neurological consultants in Israel were personally interviewed, and cases of CJD in their files were identified. All electroencephalographic laboratories were visited, and cases in which CJD had been considered were solicited. In the initial hospital record review, patients with illnesses diagnosed as presenile dementia, Pick's and Alzheimer's syndromes, subacute sclerosing panencephalitis, and amyotrophic lateral sclerosis were also included on the chance that some cases of CJD might have been so labeled. The clinical protocols of all of these cases were reviewed and, when available, the autopsy or brain biopsy material was analyzed.

Criteria for acceptance of a case as "definite" CJD included typical histopathologic appearance of the brain (subacute spongiform encephalopathy) in association with a rapidly progressive organic dementia. Most of the patients in this group had well-documented clinical evidence of involvement of pyramidal tracts, anterior horn cells, the extrapyramidal system, and the cerebellar pathways. Akinetic mutism and myoclonic jerks were often noted. Impaired vision was only occasionally documented. If dementia and at least two of the last-mentioned seven clinical signs were present together with an electroencephalogram characterized by periodic sharp waves, spikes, and suppression bursts (*7*), the patient was classified as "probable" CJD. A group of "possible" CJD cases included patients with a typical clinical history and at least three of the listed neurological signs but in whom neither electroencephalographic nor histopathological information was available. Demographic data on the population of Israel were derived from official abstracts published by the Central Bureau of Statistics (*8*).

Our search disclosed 29 acceptable patients, of whom 16 were histopathologically confirmed as definite CJD cases (13 autopsies and 3 brain biopsies), 7 were probable cases, and 6 were possible cases. Table 1 shows the frequency of CJD in various ethnic groups in Israel. The average annual age-adjusted incidence of CJD varied in the narrow range of 0.4 to 1.9 per million population for all ethnic groups except the Jewish immigrants from Libya; in the latter, it was 31.3 per million population or at least 16- to 78-fold higher. Even after age adjustment to another standard population (United States, 1970) and after exclusion of possible cases of CJD, an extraordinary Libyan focus was still apparent.

Table 2. Clinical comparisons between Libyan and other patients with Creutzfeldt-Jakob disease; *N*, number of cases.

Comparison	Libyans ($N = 13$)		Others ($N = 16$)	
	No.	Percent	No.	Percent
Clinical signs				
Dementia	13	100	16	100
Pyramidal tract	11	85	13	81
Lower motor neuron	1	8	3	19
Extrapyramidal	3	23	8	50
Myoclonic jerks	7	54	8*	53
Cerebellar	7	54	8	50
Akinetic mutism	4	31	10	62
Visual	0	0	1	6
Characteristic electroencephalogram	10*	83	8*	53
Characteristic histopathology	8	62	8†	57
Diagnosis				
Definite	8	62	8	50
Probable	5	38	2	13
Possible	0	0	6	37

* Data lacking in one patient. † Data lacking in two patients.

A comparison of characteristics of CJD between the Libyan ethnic group and the others revealed an average age at onset of 56 years for both groups and a sex ratio (male to female) of 1.2 : 1 and 3.0 : 1, respectively ($P > .05$). The average duration of illness was 4.4 months for Libyans and 11.0 months for others ($P < .05$). Other clinical manifestations were similar in both groups (Table 2). The percentage of histopathologically confirmed cases was almost identical in Libyan and other ethnic groups. All of the Libyan cases but only 63 percent of the others were classified as definite or probable CJD, which suggests that criteria for acceptance of Libyans were, if anything, more stringent. There were no familial cases of CJD in this Israeli series. Most of the Libyan immigrants were not recent arrivals in Israel; the wave of immigration from Libya commenced in 1948 with the founding of the state and was virtually completed by 1950.

In kuru (*9*), epidemiologic studies among the Fore natives of New Guinea revealed exposure to human nervous tissue which is believed to account for the transmission of that disease. An epidemiologic study comparing Libyans with other ethnic groups in Israel may identify differences that account for the dramatic excess of CJD in the Libyan immigrants. Parallel studies in Libya would be desirable.

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