however, its action could readily be demonstrated, but only in the absence of hemolysin A. The action of hemolysin B became manifest as a colorless transparent spot of lysis on the blood plate. During cataphoresis, hemolysin B always migrated to the cathode, independently of the pH, in the studied range from 3 to 8.

Since hemolysin B could not be demonstrated by the blood plate method in the presence of hemolysin A, a different technique had to be employed for its determination. This consisted in the titration of the minimal hemolysin concentration which produced complete hemolysis of a suspension of rabbit blood cells under defined conditions, lecithin being used as activator. Lecithin only activated hemolysin B. The method will be described later.[1] By this means it could be confirmed that, during cataphoresis, hemolysin B migrated only to the cathode in the studied pH range. The anode liquid was practically always negative for hemolysin B when tested in this way.

It was thus possible to demonstrate that moccasin venom contained 2 different hemolytic components which could be separated by cataphoresis: At pH 6 and 7, hemolysin A migrated only to the anode, hemolysin B only to the cathode. It could also be shown that in the same pH range, the hemorrhagin and hemolysin B could be separated by cataphoresis. The hemorrhagin migrated only to the anode, the hemolysin B only to the cathode.

Our findings seem to indicate that the molecules responsible for the action of hemolysin B are different from those which give rise to the other toxic effects of moccasin venom so far studied. Hemolysin B has a much more basic character.

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Protection of Rabbits Against Naturally Acquired Infectious Myxomatosis by Previous Infection with Fibroma Virus.

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It is generally agreed that the viruses of rabbit fibroma and rabbit myxoma are closely related immunologically and that infection with the benign fibroma virus usually protects rabbits against fatal infec-

tion with the myxoma virus.[1-8] This suggested that the fibroma virus might make a satisfactory immunizing agent for practical use in the control of infectious myxomatosis. The possibility was tested by McKenney and Shillinger[9] with discouraging results, and they concluded that the infection of rabbits with fibroma virus did not constitute a satisfactory or practical means of immunizing against infectious myxomatosis. However, careful consideration of the experiments of McKenney and Shillinger suggests that their difficulties lay more in the maintenance of a potent infective fibroma virus for use in their experiments than in an actual failure of fibroma virus to enhance the resistance of rabbits to myxomatosis.

Partly because of McKenney and Shillinger's adverse report on the practicability of using fibroma virus prophylactically and partly to determine whether fibroma-recovered rabbits would prove as resistant when exposed to cases of infectious myxomatosis as they had when inoculated with myxoma virus, the experiment to be reported in this paper was conducted.

The testicles from a rabbit inoculated intratesticularly 7 days earlier with strain A fibroma virus were removed aseptically and stored for one month in 50% glycerol in the refrigerator. (The period of storage in glycerol is not important providing it does not exceed 2 months.) At the end of this time they were washed in 3 changes of physiological saline, ground with sterile sand and suspended in physiological saline to make a 5% suspension. After the suspension had stood for 10 minutes in a tall glass graduate, the supernatant fluid was decanted for use as fibroma virus.

Each of 7 rabbits received 0.5 cc. of the fibroma virus intradermally. Another 7 rabbits were inoculated by applying 0.5 cc. of the virus to a small area of shaven skin that had been freshly scarified by means of sandpaper. The virus was rubbed into the scarified area with the rounded closed end of a small sterile tube. Fibromas appeared at the sites of inoculation in all 14 rabbits, reached their maximal size in from 12 to 14 days and then regressed within a month.

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TABLE I.

Effect of Previous Infection with Fibroma Virus on Resistance to Infectious Myxomatosis.

Rabbit No.	Previous treatment	Result of pen exposure to infectious myxoma
1599	None—control	Died of myxomatosis—14th day after initial exposure
1601	" "	" " " 18th " " " "
1602	" "	" " " 19th " " " "
1603	" "	" " " 16th " " " "
1606	" "	" " " 17th " " " "
1607	" "	" " " 13th " " " "
1608	" "	" " " 15th " " " "
1612	" "	" " " 17th " " " "
1613	" "	" " " 15th " " " "
1629	Fibroma virus intradermally	No illness
1630	" " "	" "
1631	" " "	" "
1632	" " "	" "
1633	" " "	" "
1636	" " "	" "
1637	" " "	" "
1620	" " on scarified skin	Left-sided blepharitis—survived
1622	" " " " "	No illness
1624	" " " " "	" "
1625	" " " " "	" "
1626	" " " " "	" "
1627	" " " " "	" "
1628	" " " " "	" "

Thirty-three days after infection with fibroma virus the 14 rabbits, together with 9 normal rabbits, were placed in a large pen with a rabbit that had received myxoma virus subcutaneously 3 days previously. The inoculated rabbit subsequently developed clinically typical infectious myxomatosis and died on the 9th day following inoculation. It served as the initial source of myxoma virus in the pen. The later course of the disease among the exposed animals in the pen is depicted in Table I.

As shown in the table, all 9 of the control rabbits died of typical infectious myxomatosis between the 13th and 19th days following their initial exposure to a myxoma-infected rabbit. None of the 14 rabbits recovered from fibroma, on the other hand, died, and only one showed slight symptoms of infectious myxomatosis (left-sided blepharitis) in spite of prolonged exposure to the virus not only in the inoculated rabbit but in the 9 control animals as well. The experiment was terminated on the 33rd day, 2 weeks after the death of the last control rabbit.

It would appear from this experiment that rabbits recovered from infection with fibroma virus possess a high degree of resistance to infectious myxomatosis transmitted by contact.

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Quantitative Studies on the Replacement of Body Chlorides.*

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The dangers of excessive salt administration have been pointed out by several observers,[1-4] while others have called attention to

* This study was aided by the Horace H. Rackham Fund for Graduate Studies, and the James and Elizabeth Inglis Fund for Surgical Research.

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