ESSA PHARMA INC. REPORTS RESULTS OF ANNUAL GENERAL AND SPECIAL MEETING OF SHAREHOLDERS

Houston, Texas and Vancouver, BC, March 10, 2016 – ESSA Pharma Inc. ("ESSA" or the "Company") (TSX: EPI, NASDAQ: EPIX) is pleased to announce the results of the votes on matters considered at its Annual General and Special Meeting of Shareholders held on March 10, 2016 in Vancouver, BC (the "Meeting").

At the Meeting, the shareholders of the Company (the "Shareholders") set the number of directors at seven and re-elected board members David Parkinson, Richard M. Glickman, Marianne Sadar, Raymond Andersen, Gary Sollis, Franklin M. Berger, and Scott Requadt to serve in office until the next annual meeting or until their successors are duly elected or appointed. Detailed results of the voting in respect of the election of directors are as follows:

Nominee	Votes For	% Votes For	Votes Withheld	% Votes Withheld
David Parkinson	16,112,928	95.35%	786,300	4.65%
Richard M. Glickman	16,152,328	95.58%	746,900	4.42%
Marianne Sadar	16,116,928	95.37%	782,300	4.63%
Raymond Andersen	16,112,928	95.35%	786,300	4.65%
Gary Sollis	16,152,328	95.58%	746,900	4.42%
Franklin M. Berger	16,152,328	95.58%	746,900	4.42%
Scott Requadt	16,134,178	95.47%	765,050	4.53%

At the Meeting, the Shareholders also re-appointed Davidson & Company LLP, Chartered Professional Accountants, as auditors of the Company by show of hands, approved the amendment and restatement of the Company's existing stock option plan by show of hands, and ratified and confirmed the grant of 570,705 options to David Parkinson by show of hands.

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About ESSA Pharma Inc.

ESSA Pharma is a clinical-stage pharmaceutical company focused on developing novel and proprietary therapies for the treatment of castration-resistant prostate cancer ("CRPC") in patients whose disease is progressing despite treatment with current therapies. ESSA believes that its product candidate, EPI-506, can significantly expand the interval of time in which patients suffering from CRPC can benefit from hormone-based therapies. EPI-506 acts by disrupting the androgen receptor ("AR") signaling pathway, which is the primary pathway that drives prostate cancer growth. We have shown that EPI-002, the

primary metabolite of EPI-506, prevents AR activation by binding selectively to the N-terminal domain ("NTD") of the AR. A functional NTD is essential for activation of the AR. Blocking the NTD prevents activation of the AR by all of the three known mechanisms of activation. In pre-clinical studies, blocking the NTD has demonstrated the capability to overcome the known AR-dependent mechanisms of CRPC. ESSA was founded in 2009 and is located in Houston, Texas, and Vancouver, British Columbia.

About Prostate Cancer

Prostate cancer is the second-most commonly diagnosed cancer among men and the fifth most common cause of male cancer death worldwide (Globocan, 2012). Adenocarcinoma of the prostate is dependent on androgen for tumor progression and depleting or blocking androgen action has been a mainstay of hormonal treatment for over six decades. Although tumors are often initially sensitive to medical or surgical therapies that decrease levels of testosterone (for example, Androgen Deprivation Therapy), disease progression despite castrate levels of testosterone generally represents a transition to the lethal variant of the disease metastatic castration-resistant prostate cancer (mCRPC) and most patients ultimately succumb to the illness. The treatment of mCRPC patients has evolved rapidly over the past five years; despite these advances, additional treatment options are needed to improve clinical outcomes in patients, particularly those who fail existing treatments including abiraterone or enzalutamide, or those that have contraindications to receive those drugs. Over time, patients with mCRPC generally experience continued disease progression, worsening pain, leading to substantial morbidity and limited survival rates. In both in vitro and in vivo studies, ESSA's novel approach to blocking the androgen pathway has been shown to be effective in blocking tumor growth when current therapies are no longer effective.